

WP5: Health Impact Assessments

Full Report on HIA of Outdoor Air Pollution

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HIA on Outdoor Air Pollution

Twenty two Apehis centres contribute to the HIA of OAP in WP5 of ENHIS-1 totalling 31 participating cities of 18 European countries (Figure A).

Figure A. APHEIS centres by country participating in ENHIS-1

Country	Centres	Cities
Austria	Vienna	Innsbruck Vienna
Belgium	Brussels	Brussels
Czech Republic	Prague	Prague
Denmark	Copenhagen	Copenhagen
France	France (PSAS-9 Programme)	Bordeaux Le Havre Lille Lyon Marseille Paris Rouen Toulouse
Germany	Hamburg	Hamburg
Greece	Athens	Athens
Hungary	Budapest	Budapest
Ireland	Dublin	Dublin
Italy	Rome	Rome
Netherlands	Rotterdam	Rotterdam
Poland	Cracow	Cracow
Portugal	Lisbon	Lisbon
Romania	Bucharest	Bucharest
Slovenia	Ljubljana	Ljubljana
Spain	Barcelona Bilbao Madrid Seville Valencia	Barcelona Bilbao Madrid Seville Valencia
Sweden	Sweden	Gothenburg Stockholm
United Kingdom	London	London

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Health Impact Assessment of Outdoor Air Pollution: Key HIA Findings and Recommendations

As part of ENHIS-1, this report sought to analyse the number of health events that could be prevented and are related to outdoor air pollution (PM₁₀ and ozone) in the 31 cities in 18 European countries of the Apheis network.

Because the ENHIS-1 project pays special attention to children, for the present health impact assessment (HIA), based on the available exposure-response functions (ERFs), we have analysed the effects of PM₁₀ on postneonatal mortality (total and respiratory mortality and sudden infant death syndrome), on hospital respiratory admissions (0-14 years), and on cough and lower respiratory symptoms (5-17 years); and the effects of ozone on emergency room visits for asthma (<18 years).

To complete the picture provided by the Apheis-3 HIA for the general and adult population (see www.apheis.net), we also estimated the impact of exposure to ozone on premature mortality (total, respiratory and cardiovascular mortality) in the general population, and the impact of exposure to ozone on hospital respiratory admissions for two age groups: 15-64 years and >64 years.

To select the most suitable ERFs for HIA, we used the following criteria:

- ?? Summary estimates from meta-analysis
- ?? Original studies involving large populations
- ?? Interrelated outcomes for which the overall evidence of a causal contribution of air pollution is high. Effect estimates were either based on statistically significant meta-analytic summary estimates or derived from single studies.

In our HIA, the European annual limit value of 40 µg/m³ for PM₁₀ is still exceeded in a few cities in southern and eastern Europe, although 26 of the 31 cities that measured PM₁₀ already meet the annual cut-off of 40 µg/m³. However, excepting the two Swedish cities, Hamburg and London, the 2010 annual limit value of 20 µg/m³ for PM₁₀ is exceeded in most of the cities.

Regarding ozone, all the cities are already below the long-term objective of the third Daughter Directive of February 2002 that regulates the target values of ozone concentration in ambient air for health protection: maximum daily 8-h mean value, 120 µg/m³. The cities are also below the information threshold: maximum 1-h value: 180 µg/m³. For acute effects of O₃, studies suggest effects to be particularly evident during the summer, i.e. the season of higher ranges of concentrations. However, a clear threshold of no effect has not been defined for O₃ (or for particles), and if one exists it must be in the low ranges of natural background levels of O₃. The current WHO air quality guideline for ozone of 120 µg/m³ as an eight-hour mean value does not represent a safe level of “no adverse effects”.

Regarding exposure to PM₁₀, as a reminder, in Apheis-3 a reduction of PM₁₀ levels by 5 µg/m³ would be associated with a decrease of 2 deaths per 100 000 on average for all-causes mortality (17 deaths per 100 000 for long-term exposure), 1 death per 100 000 for cardiovascular mortality and 0.5 death per 100 000 for respiratory mortality in the general population. In ENHIS-1, we completed this picture with the impact on postneonatal mortality (children between ages 1 month and 1 year).

All other things being equal, a reduction of the annual mean value of PM₁₀ levels by 5 µg/m³ would be associated with a decrease of 4.7 deaths per 100 000 children on average for total postneonatal mortality, 1.4 deaths per 100 000 children for respiratory postneonatal mortality and 1.8 deaths per 100 000 children for sudden infant death syndrome. In absolute numbers, in the cities that could provide PM₁₀, totalling almost 45 million inhabitants, the number of total postneonatal deaths would decrease by 23, for respiratory postneonatal deaths the reduction would be of 5 deaths and for sudden infant death syndrome it would be of 7. Regarding morbidity, a reduction of short-term exposure to PM₁₀ by 5 µg/m³ would be associated with a decrease of 2% for cough and lower respiratory symptoms in children 5 to 17 years of age and of 0.5% for hospital respiratory admissions in children <15 years.

Regarding ozone, all other things being equal, a reduction of 10 µg/m³ in daily maximum 8-hour mean levels in summer would be associated with a decrease in total mortality of 1.28 deaths per 100 000, 0.75 death per 100 000 for cardiovascular mortality and 0.39 death per 100 000 for respiratory mortality in the general population. This reduction would also be associated with a decrease of 0.10% in hospital respiratory admissions 15-64 years and 0.5% in hospital respiratory admissions >64 years.

A reduction of daily 1-hour maximum levels of ozone (all year) by 10 µg/m³ would be associated with a decrease of 1.14% in emergency room visits for asthma <18 years.

In absolute numbers, in the 30 cities that could provide ozone measurements, totalling more than 45 million inhabitants, reducing the daily 8-h maximum levels of ozone to 120 µg/m³ would prevent respectively 80, 48 and 21 premature deaths for total, cardiovascular and respiratory mortality in the general population, while an absolute reduction of 10 µg/m³ would increase considerably these numbers, respectively 567, 333 and 174 deaths. Regarding hospital respiratory admissions, the attributable fractions when reducing the daily 8-h maximum levels of ozone to 120 µg/m³ would be 0.02% for patients 15-64 years of age and 0.08% for patients over 64 years.

In conclusion, in this HIA we followed the Apheis-3 guidelines to establish a good basis for comparing methods and findings between 31 cities in Europe in ENHIS-1.

Our HIA in ENHIS-1 with special emphasis on children, added more evidence to the findings from Apheis-2 and 3 and other HIAs performed in Europe that air pollution continues to pose a significant threat to public health in urban areas in Europe.

The main obstacle to creating a more complete picture of the health impacts of outdoor air pollution in Europe remains the availability of morbidity data sources. Our study stresses that local, national and European public health authorities should advocate:

- Reducing the time needed to obtain validated total and cause-specific mortality data in some countries
- Producing more-uniform hospital-admissions statistics in Europe
- Accessibility, preferably on a routine basis, to other important morbidity indicators, such as asthma attacks and respiratory symptoms, using standardised methodology.

Our HIA findings continue to demonstrate that incentives to reduce PM₁₀ levels in the short and medium terms are needed to help reduce air-pollution levels further. A coordinated initiative by European legislators and national and local policy-makers could help achieve this goal.

Abbreviations

ACS study	American Cancer Society Study
AF	Attributable fraction
AirQ	Air Quality Health Impact Assessment WHO software
APHEA	Air Pollution and Health: A European approach
APHEIS	Air Pollution and Health: A European Information System
CEHAPE	Children's Environment and Health Action Plan for Europe
CI	Confidence intervals
DWP	Drinking Water Pollution
ENHIS-1	First phase of the ENvironment and Health Information System
ERFs	Exposure-Response functions
HIA	Health Impact Assessment
ICD	International Classification of Diseases
InVS	French Institute of Public Health Surveillance
LCA	Lung cancer mortality
P5	5 th percentile of the distribution of the pollutant
P95	95 th percentile of the distribution of the pollutant
PM₁₀	particulate matter less than 10 micrometers of diameter
PM_{2.5}	particulate matter less than 2.5 micrometers of diameter
PSAS-9	French national programme on the surveillance of the effects of air pollution on health in nine French cities
OAP	Outdoor Air Pollution
O₃	Ozone
RPG	Regional Priority Goal
RR	Relative risk
SD	Standard deviation

SIDS	Sudden Infant Death Syndrome
TEOM	Tapered oscillating microbalance method
TSP	Total suspended particulates
USEPA	Environmental Protection Agency of the United States of America
WHO-ECEH	World Health Organization European Centre for Environment and Health
WP5	Work Package 5

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Introduction

The final aim of our HIA is to enable the evaluation of different policy scenarios for reducing air-pollution levels in Europe. More concretely, our HIA in ENHIS-1 aims to provide the number of health events that could be prevented from outdoor air pollution (PM₁₀ and ozone) exposure with a special focus on children.

Methods

HIA methodology

We follow the recommendations of the WHO Guidelines on the Assessment and Use of Epidemiological Evidence for Environmental Health Risk Assessment (WHO 2000, 2001):

- “Specify exposure. If exposure represents a mixture, the selection of the most reasonable indicator(s) of the mixture has to be discussed. Attention should be paid to the time dimension of exposure (averaging times and duration). The distribution of exposure in the target population and in the epidemiological studies used to derive the exposure-response functions should be coherent. The magnitude of the impact depends on the level and range of exposure for which HIA is required to estimate the number of cases. The choice of a reference level may consider epidemiological and other data with regard to issues such as the existence of thresholds and natural background levels. If exposures in the target population of the HIA exceed or are below those studied, it will be necessary to determine whether exposure-response functions should be extrapolated or not.”
- “Define the appropriate health outcomes. The purpose of the HIA, the definition of exposure and the availability of the necessary data will guide the selection of outcomes. In some cases, the HIA should be assessed separately for each health outcome for which there is evidence of an effect. In other cases, in particular when estimating the monetary costs, we should avoid overlapping of various health outcomes.”
- “Specify the exposure-response relationship. The exposure-response function is the key contribution of epidemiology to HIA. The function may be reported as a slope of a regression line or as a relative risk for a given change in exposure. Exposure-response functions may be derived from pooled analysis or published meta-analyses.”
- “Derive population baseline frequency measures for the health outcomes under consideration. This is to quantify the prevalence or incidence of the selected outcomes. This information should preferably be obtained from the target population for which HIA is being made.”
- “Calculate the number of cases, under the assumption that exposure causes the health outcome, based on the distribution of the exposure in the target population, the estimates of the epidemiology exposure-response function and the observed baseline frequency of the health outcome in the population.“

Air pollution indicators: Particulate matter and ozone

Air pollution indicators were selected on the basis of the epidemiological studies that provided the exposure-response functions (ERFs) necessary for HIA. The working team of WP5 of ENHIS-1 in Bilbao prepared a report on the selection of the ERFs based on the most recent available evidence (Anderson 2004, WHO 2004, CARB 2004) (Appendix 1).

Exposure measurements

In order to harmonise and compare the information relevant to exposure assessment provided by the 31 Apehis cities, the Apehis guidelines were updated and completed by the guidelines for site selection and selection of monitoring stations developed by the French surveillance system on air pollution and health, the PSAS-9 programme, coordinated by InVS, the French Institute of Public Health Surveillance (<http://www.invs.sante.fr/psas9>) (Appendix 2). The WP5 team of ENHIS-1 in Barcelona prepared a questionnaire to assess the cities' fulfilment of the Apehis guidelines on exposure assessment. A description of the exposure assessment in each city appears in Appendix 3. The description includes: the total number and type of monitoring stations and the number used for HIA purposes; the indicators measured (PM₁₀ and ozone as basic indicators); the measurement methods and the use of a correction and/or conversion factors; the quality assurance and control and data quality; and finally the last year for data availability for each centre.

PM₁₀ measurements

PM₁₀ measurements were available in all the cities except Bucharest, Budapest and Valencia. The daily exposure indicator of PM₁₀ was calculated as the arithmetic mean of the daily concentrations of the selected stations. For the purpose of HIA of short-term exposure to PM₁₀, direct automatic PM₁₀ measurements were used. For HIA on postneonatal mortality, because the exposure-response functions used were taken from publications that used gravimetric methods (Lacasana et al. 2005 and Woodruff et al 1997), to be consistent, we decided to correct the automatic PM₁₀ measurements (β -attenuation and TEOM) used by most of the cities by a specific correction factor in order to compensate losses of volatile particulate matter. When available, a local correction was used factor, chosen with the advice of the local air-pollution network; otherwise, the cities used the 1.3 European default correction factor recommended by the EC Working Group on Particulate Matter <http://europa.eu.int/comm/environment/air/pdf/finalwgreporten.pdf>

Ozone measurements

Ozone (O₃) was measured using ultraviolet absorption methods. All the cities, except Bucharest, could provide O₃ data. Based on the relevant ERFs selected for HIA, two ozone indicators were used: the maximum daily 8-h mean in summer and the daily 1-h maximum all year. For the maximum daily 8-h mean, the Apehis exposure guidelines for ozone indicate to use the maximum daily 8-h moving average, which is directly in line with the 3rd Daughter Directive (2002/3/EC). The daily maximum 1-hour indicator was calculated as the arithmetic mean of the daily 1-hour maximum of the selected stations. The maximum daily 8-hour moving average of each day have been calculated as the arithmetic mean of the maximum 8-hour moving averages of the selected stations for the summer period (1st April to 30th September).

Total suspended particulates (TSP) conversion factor

Only two cities, Bucharest and Budapest, evaluated TSP monitoring stations as appropriate for HIA. They converted TSP to PM₁₀, using respectively 0.6 and 0.58 as local conversion factors.

Table 1. Measurement methods and correction factors used in ENHIS-1

City	Measurement method			PM ₁₀ correction factor
	PM ₁₀	Ozone	TSP ¹	
Athens	β-attenuation	Ultraviolet (UV) absorption		1.3*
Barcelona ²	gravimetric	UV absorption		1
Bilbao	β-radiation absorption	UV absorption		1.2 [#]
Bordeaux	TEOM (50°C)	UV absorption		(a)
Brussels	TEOM	UV absorption		1.47
Bucharest	not available	not available	gravimetric	x
Budapest	not available	UV absorption	β-ray-operation	xx
Copenhagen	gravimetric	UV absorption		1
Cracow	β-gauge-monitor	UV absorption		1 [#]
Dublin	gravimetric	UV absorption		1
Gothenburg	TEOM (50°C)	UV absorption		1.2 [#]
Hamburg	TEOM, β-Absorption	UV absorption		1.3*
Innsbruck	β-radiation absorption	UV absorption		1.3*
Le Havre	TEOM (50°C)	UV absorption		(a)
Lille	TEOM (50°C)	UV absorption		(a)
Lisbon	β-attenuation	UV absorption		1,11
Ljubljana	TEOM (50°C)	UV absorption		1.3*
London	TEOM	UV absorption		1.3*
Lyons	TEOM	UV absorption		(a)
Madrid	β-attenuation	UV absorption		1 [#]
Marseille	TEOM (50°C)	UV absorption		(a)
Paris	TEOM	UV absorption		(a)
Prague	β-radiation absorption	UV spectroscopy		1.3*
Rome	β-gauge monitor	UV absorption		1.3*
Rotterdam	β-gauge monitor	UV absorption		1.3*
Rouen	TEOM (50°C)	UV absorption		(a)
Seville	β-radiation-attenuation	UV absorption		1.13 [#]
Stockholm	TEOM (50°C)	UV absorption		1.2 [#]
Toulouse	TEOM (50°C)	UV absorption		(a)
Valencia	not available	UV absorption		not applicable
Vienna	gravimetric	UV absorption		1

¹ TSP: total suspended particulates

² PM10 data from Barcelona begin in April 2002 and correspond to 3 workable days per week. The annual completeness of the series of the monitoring stations ranges from 16% to 38%

* For HIA of postneonatal mortality PM₁₀ TEOM has been corrected by European default factor of 1.3 or a local one

Derived from parallel PM₁₀ measurements within the city

x PM10=TSP*0.6

xx PM10=TSP*0.58

(a) French cities: as part of the national pilot program for PM surveillance, specific polynomial regression has been used for each city PM₁₀ correction. The coefficients of these regressions were derived from parallel PM₁₀ measurements within each city

Health outcomes and E-R functions

To select the most suitable ERFs for HIA we observed the following criteria:

- ?? It was considered preferable to use summary estimates from meta-analysis
- ?? Only original studies involving great populations were deemed suitable for HIA
- ?? We used interrelated outcomes for which the overall evidence of a causal contribution of air pollution is high. Effect estimates were either based on statistically significant meta-analytic summary estimates or derived from single studies.

The full report on the selection of ERFs is in Appendix 1. Appendix 3 gives a full description of the health indicators used (mortality and morbidity data concerning children and general population separately, according to the selected ERFs). Were included the type of sources, the coverage, the delay to obtain the data, the last year available, the existence of information about the validity of data as well as quality control procedures in place, the type of coding used, the completeness of the data, and conclusions about the comparability of the data.

Because the ENHIS-1 project pays a special attention to children, for the present HIA, based on the available ERFs, we have analysed the effects of PM₁₀ on postneonatal mortality (total and respiratory mortality and Sudden Infant Death Syndrome), on hospital respiratory admissions (0-14 years), on cough and lower respiratory symptoms (5-17 years), and the effects of ozone on emergency room visits for asthma (<18 years).

In order to complete the picture of the Apehis-3 HIA for the general and adult population (www.apheis.net), we also estimated the impact of exposure to ozone on premature mortality (total, respiratory and cardiovascular mortality) in the general population, and the impact of exposure to ozone on hospital respiratory admissions for two age-groups: 15-64 years and >64 years.

HIA tools: Excel spreadsheets

Number of cases

Calculations of the number of cases were made using an Excel spreadsheet developed by the PSAS-9 centre in Marseille. Guidelines for this excel tool were developed by the WP5 team of ENHIS-1 in Bilbao (Appendix 4).

An estimate of the impact can be based on the calculation of the attributable proportion (AP), indicating the fraction of the health outcome that can be attributed to the exposure in a given population (provided there is a causal association between the exposure and the health outcome). With the population distribution of exposure determined in the exposure assessment stage, and the identified E-R function, the attributable proportion can be calculated using the formula:

$$AP = \frac{[RR(c) - 1] * p(c)}{[RR(c) * p(c)]} \quad [1]$$

where: RR(c) is the relative risk for the health outcome in category c of exposure

p(c) is the proportion of the target population in category c of exposure

Knowing (or, often, assuming) a certain underlying frequency of the outcome in the population, I, the rate (or number of cases per unit population) attributed to the exposure in the population can be calculated as:

$$IE = I * AP$$

Consequently, the frequency of the outcome in the population free from the exposure can be estimated as:

$$INE = I - IE = I * (1 - AP) \quad [2]$$

For a population of a given size N, this can be converted to the estimated number of cases attributed to the exposure, $NE = IE * N$.

Knowing the (estimated) incidence among the non-exposed population and the relative risk at a certain pollution level, it is also possible to estimate an excess incidence ($I+(c)$) and excess number of cases ($N+(c)$), at a certain category of exposure:

$$I+(c) = (RR(c) - 1) * p(c) * INE \quad [3]$$

$$N+(c) = I+(c) * N \quad [4]$$

Attributable fractions

For the outcomes for which a population baseline frequency measure was not available (cough, lower respiratory symptoms) or was not comparable between cities (respiratory hospital admissions and emergency room visits for asthma), an attributable number of cases could not be calculated. Instead, an attributable fraction (AF) was calculated in a complementary excel file developed by the WP5 ENHIS-1 working team in Bilbao and at the InVS in Saint Maurice:

$$AF = (RR - 1) / RR$$

RR is the relative risk (or ER function)

For a disease for which the numbers (incidence or prevalence) are not known, the AF is the part, expressed in percentage, that can be attributed to the exposure factor, here air pollution.

Health Impact Assessment scenarios

1 - HIA scenarios for PM₁₀

The first two scenarios for PM₁₀ were chosen according to the European Council Directive 1999/30/EC of 22 April 1999 relating to limit values for sulphur dioxide, nitrogen dioxide and all nitrogen oxides, particulate matter and lead in ambient air (Official Journal L 163, 29/06/1999 P. 0041 – 0060): a PM₁₀ 24-hour limit value of 50 µg/m³ should not be exceeded more than 35 times per year by 1 January 2005 and no more than seven times per year by 1 January 2010 in the Member States. Also, a PM₁₀ annual limit value should not exceed 40 µg/m³ by 1 January 2005 and 20 µg/m³ by 1 January 2010. The third scenario for PM₁₀ is for an absolute reduction by 5 µg/m³.

1.1. PM₁₀ and postneonatal mortality (total, respiratory and sudden infant death syndrome-SIDS)

1.1.1 Reduction of the annual mean value of PM₁₀ to a level of 40 µg/m³ (Limit of 1999/30/EC Directive for 2005)

1.1.2 Reduction of the annual mean value of PM₁₀ to a level of 20 µg/m³ (Limit of 1999/30/EC Directive for 2010)

1.1.3 Reduction by 5 µg/m³ of the annual mean value of PM₁₀

1.2. PM₁₀ and cough and lower respiratory symptoms (5-17 years), and hospital respiratory admissions in people under 15 years (<15 years)

1.2.1 Reduction of PM₁₀ levels to a 24-hour value of 50 µg/m³ in all days exceeding this value (Limit of 1999/30/EC Directive)

1.2.2 Reduction of PM₁₀ levels to a 24-hour value of 20 µg/m³ in all days exceeding this value

1.2.3 Reduction by 5 µg/m³ of all the 24-hour values

2.- HIA scenarios for ozone

For ozone' scenarios, the third Daughter Directive of February 2002 regulates the target values of ozone concentration in ambient air (http://europa.eu.int/eur-lex/pri/en/oj/dat/2002/l_067/l_06720020309en00140030.pdf): Health protection: maximum 8-hours 120 µg/m³; Information threshold: maximum 1-hour 180 µg/m³. The third scenario for ozone is for an absolute reduction by 10 µg/m³.

2.1 Daily maximum 8-hour moving average concentration and mortality in general population

2.1.1 Reduction of O₃ daily maximum 8-hour moving average concentrations to 120 µg/m³ in all days exceeding this value (Limit for health protection of 2002/3/EC Directive)

2.1.2 Reduction by 10 µg/m³ in the daily maximum 8-hour moving average concentrations.

2.2 Daily maximum 8-hour moving average concentration and hospital respiratory admissions in people 15-64 years and >64 years

2.2.1 Reduction of O₃ daily maximum 8-hour moving average concentrations to 120 µg/m³ in all days exceeding this value (Limit for health protection of 2002/3/EC Directive)

2.2.2 Reduction by 10 µg/m³ in the daily maximum 8-hour moving average concentrations.

2.3 Daily maximum 1-hour concentration and emergency room visits for asthma in people under 18 year (<18 years)

2.3.1 Reduction of O₃ daily maximum 1-hour concentrations to a level of 180 µg/m³ in all days exceeding this value (Information threshold of 2002/3/EC Directive)

2.3.2 Reduction by 10 µg/m³ of the daily maximum 1-hour concentrations

The following table summarises the HIAs on outdoor air pollution (OAP) conducted in ENHIS-1 specifying: the health outcomes and their ICD codes, the age groups, the air pollution indicators, the period and mean type, the relative risks (or E-R functions) selected, the scenarios chosen and the references of the ERFs selected.

Summary of data components for HIA outdoor air pollution in ENHIS-1							
Health outcome	Population	Pollutant	Period	Mean type	RR (for 10 µg.m ³ increase)	Scenarios	References
Mortality							
Total mortality excluding external causes (ICD9 < 800 - ICD10 A00-R99)	All ages	O ₃ 8h max	Summer ¹	Daily	1.0031 (1.0017-1.0052)	Reduction to 120 µg.m ³	Gryparis et al 2004
Cardiovascular mortality (ICD9 390-459 - ICD10 I00-I99)					1.0046 (1.0022-1.0073)	Reduction by 10 µg.m ³	
Respiratory mortality (ICD9 460-519 - ICD10 J00-J99)					1.0113 (1.0074-1.0151)		
Total postneonatal mortality	1 month-1 year	Corrected PM ₁₀ ²	Year	Annual	1.048 (1.022-1.075)	Reduction to 20 µg.m ³	Lacasaña et al 2005
Postneonatal respiratory mortality (ICD9 460-519 - ICD10 J00-J99)					1.216 (1.102-1.342)	Reduction to 40 µg.m ³	
Postneonatal Sudden Infant Death Syndrom Mortality (ICD9 798.0 - ICD10 R95)					1.12 (1.07-1.17)	Reduction by 5 µg.m ³	Woodruff 1997
Morbidity							
Emergency room visits for asthma (ICD-9 codes 493, ICD-10 codes J45, J46)	< 18 years	O ₃ 1h max	Year	Daily	1.0115 (1.0067-1.0163)	Reduction to 180 µg.m ³ Reduction by 10 µg.m ³	CARB 2004
Cough	5-17 years	PM ₁₀ daily mean			1.0407 (1.0202-1.0511)	Reduction to 20 µg.m ³ Reduction to 50 µg.m ³ Reduction by 5 µg.m ³	Ward & Ayres 2004
Lower respiratory symptoms LRS		PM ₁₀ daily mean			1.0407 (1.0202 -1.617)	Reduction to 20 µg.m ³ Reduction to 50 µg.m ³ Reduction by 5 µg.m ³	Ward and Ayres 2004
Hospital respiratory admissions (ICD9 460-519 - ICD10 J00-J99)	< 15 years	PM ₁₀ daily mean			1.010 (0.998-1.021)	Reduction to 20 µg.m ³ Reduction to 50 µg.m ³ Reduction by 5 µg.m ³	Anderson et al 2004
Hospital respiratory admissions (ICD9 460-519 - ICD10 J00-J99)	15 - 64 years	O ₃ 8h max			Summer	1.001 (0.991 -1.012)	
Hospital respiratory admissions (ICD9 460-519 - ICD10 J00-J99)	> 64 years		1.005 (0.998-1.012)	Reduction by 10 µg.m ³			

¹ Definition of summer period: 01 April – 30 September

² PM₁₀ reference papers for HIA on postneonatal mortality use gravimetric methods to measure PM₁₀. If the local air quality network uses automatic methods (TEOM or other) a correction factor is required to compensate for loss of volatile compounds: if available, a local correction factor recommended by the air quality network or, by default, the European factor 1.3.

City reports

Besides this general comparative report, we provided a city report template (Appendix 5) to allow each centre elaborate the city-specific reports. We produced 29 city-specific reports, which are posted in the Apheis web site (www.apheis.net).

Compilation of findings

Descriptive findings

The Apehis network including eight new cities (Brussels, Copenhagen, Hamburg, Innsbruck, Lisbon, Prague, Rotterdam and Vienna) provided the information required for the ENHIS-1 HIA on outdoor air pollution. Thirty-one cities of 18 European countries contributed to the HIA of OAP. The most recent common year for air pollution and health data for HIA for all the cities was 2001 or 2002 (Table 2). This was mainly due to the long delay required to get validated mortality data in some countries.

Table 2. Years for air pollution and health data

City	Air pollution data		Health data				
	PM10	Ozone	Mortality*	Hospital respiratory admissions	Emergency room visits for asthma	Cough ¹	Lower Respiratory Symptoms ²
Athens	2001	2001	2001	na.	na.	na.	na.
Barcelona ³	2002	2002	2002	2002	na.	na.	na.
Bilbao	2002	2002	2002	2002	na.	na.	na.
Bordeaux	2001	2001	2001	2001	na.	na.	na.
Brussels	2001	2001	2001	2001	2001	na.	na.
Bucharest	2001	not available	2001	na.	na.	na.	na.
Budapest	2001	2001	2001	na.	na.	na.	na.
Copenhagen	2001	2001	2001	2001	2001	na.	na.
Cracow	2001	2001	2001	na.	na.	na.	na.
Dublin	2002	2002	2002	2002	na.	na.	na.
Gothenburg	2002	2002	2002	2002	2002	na.	na.
Hamburg	2001	2001	2001	2001	na.	na.	na.
Innsbruck	2002	2002	2002	2002	na.	na.	na.
Le Havre	2001	2001	2001	2001	na.	na.	na.
Lille	2001	2001	2001	2001	na.	na.	na.
Lisbon	2002	2002	2002	2002	na.	na.	na.
Liubliana	2001	2001	2001	2001	na.	na.	na.
London	2001	2001	2001	2001	na.	available	available
Lyon	2001	2001	2001	2001	na.	na.	na.
Madrid	2002	2002	2002	2002	2002	na.	na.
Marseille	2001	2001	2001	2001	na.	na.	na.
Paris	2001	2001	2001	2001	na.	na.	na.
Prague	2001	2001	2001	2001	na.	na.	na.
Rome	2001	2001	2001	2001	na.	na.	na.
Rotterdam	2001	2001	2001	2001	na.	na.	na.
Rouen	2001	2001	2001	2001	na.	na.	na.
Seville	2001	2001	2001	2001	na.	na.	na.
Stockholm	2002	2002	2002	2002	2002	na.	na.
Toulouse	2001	2001	2001	2001	na.	na.	na.
Valencia	na.	2002	2002	2002	na.	na.	na.
Vienna	2002	2002	2002	2002	na.	na.	na.

* including postneonatal mortality

^{1 2} Data on cough and lower respiratory symptoms were not available from a routine source except in London but payable

² PM10 data from Barcelona begin in April 2002 and correspond to 3 workable days per week. The annual completeness of the series of the monitoring stations ranges from 16% to 38%

na.: data not available for the health impact assessment

Demographic characteristics

The total population covered in this HIA is of almost 46 million inhabitants. In those cities that could provide the information, population between 1 month and 1 year of age was around 1%. In Athens, Barcelona, Innsbruck, London, Madrid and Vienna, only population data below 1 year was available or could be estimated. The proportion of children younger than 15 years is the highest in Lille (21.7%) and the lowest in Bilbao (11.1%). The proportion of young adults (below 18 years) is also the highest in Lille (24.8%) but the lowest in Barcelona (14.1%) where the proportion of people over 64 years of age is the highest (21.7%) (Table 3).

Table 3. Demographic characteristics

City	Year	Population	Population between 1 month and 1 year	Population 0-14 years	Population below 18 years	Population 15-64 years	Population > 64 years
		Number	Percent	Percent	Percent	Percent	Percent
Athens	2001	3 188 305	0.96	13.98	20.25	70.15	15.87
Barcelona	2001	1 503 884	0.84	11.51	14.12	66.82	21.67
Bilbao	2001	708 395	0.71	11.07	15.35	69.60	19.35
Bordeaux	1999	604 238	1.07	15.52	18.92	68.82	15.66
Brussels	2001	961 861	1.59	17.91	21.22	65.43	16.66
Bucharest	2001	1 972 170	0.72	17.80	25.00	68.60	13.60
Budapest	2001	1 737 747	0.79	12.90	16.00	69.60	17.50
Copenhagen	2001	590 224	1.40	14.02	15.76	72.18	13.80
Cracow	2001	759 046	0.80	14.30	18.60	72.10	13.64
Dublin	2002	495 781	1.10	16.17	19.58	71.02	12.81
Gothenburg	2002	474 921	1.20	16.30	20.30	68.30	15.40
Hamburg	2001	1 720 964	0.83	13.48	17.01	69.53	16.98
Innsbruck	2002	113 095	0.9	13.90	18.91	69.80	16.30
Le Havre	1999	254 653	1.22	19.40	23.97	65.55	15.06
Lille	2001	1 090 151	1.34	21.73	24.80	66.11	12.16
Lisbon	2001	1 892 903	1.07	14.70	17.95	69.52	15.80
Ljubljana	2001	270 032	0.80	13.96	17.48	70.80	15.23
London	2001	7 172 091	1.33	19.04	22.58	68.53	12.44
Lyon	1999	782 828	1.20	16.50	22.70	67.80	15.70
Madrid	2001	2 957 058	0.94	12.30	16.20	68.60	19.10
Marseille	1999	856 507	1.10	18.00	22.70	64.10	17.90
Paris	1999	6 174 000	1.30	18.20	22.60	68.70	13.20
Prague	2001	1 169 773	0.80	13.20	19.00	70.70	16.10
Rome	2001	2 546 804	0.85	12.84	16.34	68.12	19.04
Rotterdam	2001	595 255	1.27	17.51	20.83	67.49	15.00
Rouen	1999	447 721	1.20	18.04	22.11	66.88	15.09
Seville	2001	702 522	1.00	15.05	na.	69.82	15.12
Stockholm	2002	1 185 841	1.30	17.00	21.00	68.10	14.90
Toulouse	1999	670 713	1.09	15.90	19.10	70.40	13.70
Valencia	2002	764 010	0.72	12.90	15.85	69.60	17.50
Vienna	2002	1 550 874	1.01	14.69	19.57	69.53	15.78

Athens, Barcelona, Innsbruck, London, Madrid and Vienna: population below 1 year

Athens, Innsbruck and Prague: population < 20 years

na.: data not available for the health impact assessment

Air pollution levels

All the cities provided PM₁₀ measurements except Valencia. Bucharest and Budapest converted TSP into PM₁₀. Ozone was provided by all the cities except Bucharest.

Table 4 gives in the first four columns, a detailed picture of directly measured (not adjusted for HIA) and in the four following columns, corrected (for HIA on postneonatal mortality) levels of PM₁₀ in the participating cities, as well as the daily 1-h maximum and the maximum daily 8-h mean of ozone (mean levels, standard deviation [SD], 5th and 95th percentiles of the distribution of the pollutants in each city).

Figures 1, 2 and 3 represent PM₁₀ and ozone levels in the 31 participating cities.

When reading these tables and figures, keep in mind the possible different sources of variability in the exposure measurements, other than the actual air pollutants concentrations (i.e. different sampling or analytical techniques; different sampling days during the week, different criteria for location of the sampling points, etc).

Table 4. PM₁₀ and ozone levels (µg/m³)

City	Measured PM ₁₀				Corrected PM ₁₀ [*]				Ozone				Ozone			
	Mean	SD ¹	P5 ²	P95 ³	Mean	SD	P5	P95	Daily 1-h max all year	SD	P5	P95	Daily 8-h max summer ⁶	SD	P5	P95
Athens	52,1	19,2	24,8	86,7	67,8	25,0	42,0	112,7	101,0	37,4	49,1	164,4	109,0	21,6	74,4	146,0
Barcelona ⁴	39,7	14,3	19,5	65,1	39,7	14,3	19,5	65,1	57,6	24,0	16,0	93,8	40,7	12,5	18,0	60,0
Bilbao	36,2	17,0	16,1	69,5	43,4	20,3	19,3	83,4	58,7	18,2	27,6	88,4	59,8	14,4	34,0	82,0
Bordeaux	21,0	10,0	10,1	38,0	25,3	14,5	11,1	48,7	70,8	31,5	25,3	130,6	83,9	24,3	49,8	130,0
Brussels	24,9	12,3	12,2	44,2	36,6	18,1	18,0	65,0	60,0	36,1	9,0	142,0	73,6	30,2	31,0	136,0
Bucharest ⁵	62,0	20,0	40,0	88,0	62,0	20,0	40,0	88,0	na.	na.	na.	na.	na.	na.	na.	na.
Budapest ⁵	22,2	10,9	9,9	42,7	28,9	14,2	12,9	55,5	58,4	28,7	17,1	107,0	74,0	20,9	42,0	113,0
Copenhagen	21,3	10,5	7,5	41,3	21,3	10,5	7,5	41,3	67,5	19,3	35,9	96,3	68,1	14,6	44,9	92,0
Cracow	42,2	24,0	15,5	82,0	42,2	24,0	15,5	82,0	65,5	27,4	23,0	114,0	62,1	23,5	27,0	102,0
Dublin	24,0	12,5	11,8	49,5	24,0	12,5	11,8	49,5	65,0	16,0	38,0	87,0	58,0	16,0	29,0	81,0
Göthenburg	17,8	8,3	7,5	32,4	21,4	10,0	9,1	38,9	75,0	23,1	35,6	115,8	78,7	18,3	50,6	111,0
Hamburg	19,1	10,2	8,8	34,6	24,8	13,2	11,4	45,0	59,0	27,0	15,5	104,3	69,0	24,8	11,3	92,0
Innsbruck	23,1	19,2	7,7	68,5	30,0	25,0	10,0	89,0	73,0	38,0	11,0	129,0	90,0	26,0	37,0	128,0
Le Havre	21,4	9,1	12,0	40,0	24,0	11,2	12,9	46,8	72,0	28,2	26,6	120,9	79,7	23,1	52,7	134,0
Lille	21,4	11,7	10,1	40,1	27,0	19,3	11,4	61,6	64,1	31,6	12,3	125,9	73,4	26,0	38,8	126,0
Lisbon	28,8	14,4	10,8	57,7	32,0	16,0	12,0	64,0	76,0	23,0	44,0	118,0	79,0	22,0	44,0	114,0
Ljubljana	29,5	16,9	6,9	65,3	38,4	22,0	9,0	84,9	77,0	46,6	8,6	158,0	78,0	35,8	27,2	129,0
London	13,1	5,6	6,9	24,0	17,0	7,0	11,3	31,2	47,1	24,3	11,0	88,0	48,0	20,8	17,9	83,0
Lyon	22,2	9,7	10,5	39,5	25,9	12,2	11,7	47,8	69,5	41,2	7,5	149,0	61,4	37,9	4,9	135,0
Madrid	33,3	15,5	13,6	59,1	33,3	15,5	13,6	59,1	61,0	28,0	18,0	106,0	70,0	16,0	46,0	97,0
Marseille	29,0	10,0	15,0	49,0	30,9	11,0	16,0	53,0	90,7	39,5	34,0	166,0	102,5	27,0	66,0	154,0
Paris	22,4	9,3	11,1	41,5	27,0	13,0	13,0	55,0	66,0	37,0	14,0	140,0	78,0	31,0	35,0	142,0
Prague	26,2	12,3	13,1	46,9	34,0	16,0	17,0	61,0	74,0	32,0	31,0	133,0	87,0	26,0	49,0	134,0
Rome	47,3	16,7	24,8	76,7	61,0	22,0	32,0	100,0	90,8	44,0	24,3	170,3	105,4	28,2	57,8	155,0
Rotterdam	28,5	5,9	19,3	44,3	37,1	7,7	25,0	57,6	64,6	25,1	9,0	121,4	73,2	19,1	37,5	115,0
Rouen	21,4	8,9	11,5	38,0	22,2	10,2	11,5	41,2	71,4	32,4	26,9	141,3	83,2	28,6	46,2	147,0
Seville	40,5	9,0	25,9	55,7	45,8	10,1	29,3	62,9	71,0	24,0	39,7	117,7	73,1	18,5	39,8	105,0
Stockholm	15,2	10,0	6,0	34,8	18,2	12,0	7,2	41,8	76,0	22,4	38,8	114,1	86,0	17,0	55,0	114,0
Toulouse	22,0	10,0	11,0	36,0	25,0	12,0	11,0	41,0	79,0	31,0	33,0	139,0	91,0	23,0	63,0	132,0
Valencia	na.	na.	na.	na.	na.	na.	na.	na.	67,8	25,3	24,6	108,5	69,8	17,3	45,3	100,0
Vienna	30,0	17,0	9,0	65,0	30,0	17,0	9,0	65,0	72,0	35,0	16,0	124,0	90,0	22,0	43,0	121,0

¹ SD: Standard deviation

² P5: 5th percentile of the distribution of the pollutant

³ P95: 95th percentile of the distribution of the pollutant

⁴ PM₁₀ data from Barcelona begin in April 2002 and correspond to 3 workable days per week. The annual completeness of the series of the monitoring stations ranges from 16% to 38%

⁵ PM₁₀ converted from TSP

^{*} PM₁₀ measurements corrected by European (1.3) or by a local correction factor

⁶ Definition of summer: 01 April to 30 September

na.: not available

These are the PM and ozone levels for 2001 or 2002, the years for which we could get the most recent mortality data.

Comparing PM and ozone levels for 2001-2002 and 2003-2004

From the figures reported by 25 of the 31 participating cities, PM levels for 2003 or 2004 showed a not negligible decrease in 15 cities. Six cities showed an increase in PM levels, Ljubljana showed the highest increase (table 5).

Table 5. Mean levels, standard deviation and 5th and 95th percentiles of the distribution of PM₁₀ in 2001-2002 and 2003-2004

City	Measured PM ₁₀								Corrected PM ₁₀ *							
	Mean		SD ¹		P5 ²		P95 ³		Mean		SD		P5		P95	
	2003 2004	2001 2002	2003 2004	2001 2002	2003 2004	2001 2002	2003 2004	2001 2002	2003 2004	2001 2002	2003 2004	2001 2002	2003 2004	2001 2002	2003 2004	2001 2002
Athens 2004	41.6	52.1	20.5	19.2	19.7	24.8	70.8	86.7	54.1	67.8	26.7	25.0	25.6	42.0	92.0	112.7
Barcelona 2004	42.0	39.7	17.0	14.3	17.7	19.5	73.4	65.1	42.0	39.7	17.0	14.3	17.7	19.5	73.4	65.1
Bilbao 2004	29.6	36.2	13.6	17.0	11.9	16.1	60.1	69.5	35.5	43.4	16.4	20.3	14.3	19.3	72.1	83.4
Bordeaux 2003	21.8	21.0	9.1	10.0	11.3	10.1	38.5	38.0	26.3	25.3	12.7	14.5	12.5	11.1	49.5	48.7
Brussels	na	24.9	na	12.3	na	12.2	na	44.2	na	36.6	na	18.1	na	18.0	na	65.0
Bucharest ⁴	na	62.0	na	20.0	na	40.0	na	88.0	na	62.0	na	20.0	na	40.0	na	88.0
Budapest ⁴	na	22.2	na	10.9	na	9.9	na	42.7	na	28.9	na	14.2	na	12.9	na	55.5
Copenhagen ⁵ 2004	19.5	21.3	8.8	10.5	8.0	7.5	36.7	41.3	19.5	21.3	8.8	10.5	8.0	7.5	36.7	41.3
Cracow 2004	56.0	42.2	38.0	24.0	16.0	15.5	133.0	82.0	56.0	42.2	38.0	24.0	16.0	15.5	133.0	82.0
Dublin	17.0	24.0	9.3	12.5	7.4	11.8	35.0	49.5	17.0	24.0	9.3	12.5	7.4	11.8	35.0	49.5
Gothenburg 2004	17.4	17.8	6.9	8.3	9.0	7.5	30.4	32.4	20.9	21.4	8.3	10.0	10.8	9.1	36.5	38.9
Hamburg	na	19.1	na	10.2	na	8.8	na	34.6	na	24.8	na	13.2	na	11.4	na	45.0
Innsbruck 2004	22.0	23.1	15.5	19.2	6.6	7.7	55.3	68.5	28.6	30.0	20.2	25.0	8.6	10.0	71.9	89.0
Le Havre 2004	20.6	21.4	8.1	9.1	11.7	12.0	35.9	40.0	22.9	24.0	9.9	11.2	12.5	12.9	41.5	46.8
Lille 2003	26.3	21.43	12.2	11.7	12.9	10.1	50.9	40.1	33.9	27.0	19.3	19.3	14.8	11.4	72.8	54.2
Lisbon 2004	27.7	28.8	16.2	14.4	11.6	10.8	59.1	57.7	30.7	32.0	17.9	16.0	12.9	12.0	65.6	64.0
Ljubljana 2004	40.2	29.5	1.0	16.9	16.8	6.9	80.9	65.3	51.6	38.4	1.3	22.0	21.6	9.0	101.1	84.9
London	na	13.1	na	5.6	na	6.9	na	24.0	na	17.0	na	7.0	na	11.3	na	31.2
Lyon 2004	24.9	22.2	11.6	9.7	12.6	10.5	44.6	39.5	29.5	25.9	15.2	12.2	14.2	11.7	54.7	47.8
Madrid 2004	33.4	33.3	17.7	15.5	12.8	13.6	68.8	59.1	33.4	33.3	17.7	15.5	12.8	13.6	68.8	59.1
Marseille 2004	28.1	29.0	11.8	10.0	13.3	15.0	47.3	49.0	29.9	30.9	13.1	11.0	13.9	16.0	51.0	53.0
Paris 2004	21.0	22.4	8.0	9.3	11.1	11.1	34.3	41.5	25.4	27.0	11.1	13.0	12.5	13.0	43.9	55.0
Prague	na	26.2	na	12.3	na	13.1	na	46.9	na	34.0	na	16.0	na	17.0	na	61.0
Rome 2002	48.0	47.3	9.3	16.7	22.0	24.8	88.2	76.7	62.4	61.0	27.9	22.0	28.6	32.0	114.6	100.0
Rotterdam 2004	27.3	28.5	6.5	5.9	17.5	19.3	43.3	44.3	36.3	37.1	8.0	7.7	24.2	25.0	56.6	57.6
Rouen 2004	19.4	21.4	7.6	8.9	11.1	11.5	32.8	38.0	20.1	22.2	8.6	10.2	11.0	11.5	35.0	41.2
Seville 2003	34.2	40.5	8.9	9.0	21.9	25.9	50.7	55.7	38.6	45.8	10.1	10.1	24.8	29.3	57.2	62.9
Stockholm 2004	14.3	15.2	7.9	10.0	6.3	6.0	32.3	34.8	17.2	18.2	9.5	12.0	4.6	7.2	38.8	41.8
Toulouse 2004	20.0	22.0	8.9	10.0	8.0	11.0	36.5	36.0	22.0	25.0	10.2	12.0	8.7	11.0	41.2	41.0
Valencia 2004	34.6	na.	14.0	na.	15.0	na.	63.7	na.	34.6	na.	14.0	na.	15.0	na.	63.7	na.
Vienna 2004	25.4	30.0	15.1	17.0	8.6	9.0	58.1	65.0	25.4	30.0	15.1	17.0	8.6	9.0	58.1	65.0

1. SD: Standard deviation

2. P5: 5th percentile of the distribution of the pollutant

3. P95: 95th percentile of the distribution of the pollutant

4. PM₁₀ converted from TSP

* PM₁₀ measurements corrected by European (1.3) or by a local correction factor

5. PM₁₀ daily gravimetric results

na.: not available

Contrary to PM levels, when comparing O₃ levels for 2001 or 2002 with years 2003 or 2004, the figures reported by 22 of the 31 participating cities, showed that daily 1-h maximum levels increased in 50% of the cities and that daily 8-h maximum levels reported by 21 cities increased in 12 cities (table 6).

Table 6. Daily 1-h maximum levels, daily 8-h maximum levels, standard deviation and 5th and 95th percentiles of the distribution of ozone in 2001-2002 and 2003-2004

City	Ozone															
	Daily 1-h max all year								Daily 8-h max summer ⁴							
	SD ¹		P5 ²		P95 ³				SD		P5		P95			
	2003	2001	2003	2001	2003	2001	2003	2001	2003	2001	2003	2001	2003	2001	2003	2001
	2004	2002	2004	2002	2004	2002	2004	2002	2004	2002	2004	2002	2004	2002	2004	2002
Athens 2004	88.4	101.0	27.3	37.4	47.2	49.1	139.6	164.4	93.4	109.0	16.1	21.6	66.7	74.4	120.7	146.7
Barcelona 2004	65.1	57.6	28.8	24.0	20.7	16.0	111.9	93.8	53.4	40.7	14.8	12.5	25.4	18.0	74.7	60.5
Bilbao 2004	62.5	58.7	21.1	18.2	26.0	27.6	96.2	88.4	68.2	59.8	14.8	14.4	43.3	34.0	91.6	82.1
Bordeaux 2002	69.5	70.8	29.0	31.5	24.7	25.3	115.4	130.6	99.6	83.9	24.8	24.3	63.0	49.8	144.3	130.2
Brussels	na	60.0	na	36.1	na	9.0	na	142.0	na	73.6	na	30.2	na	31.0	na	136.0
Bucharest	na	na.	na	na.	na	na.	na	na.	na	na.	na	na.	na	na.	na	na.
Budapest	na	58.4	na	28.7	na	17.1	na	107.0	na	74.0	na	20.9	na	42.0	na	113.1
Copenhagen 2004	127.0	67.5	19.0	19.3	38.0	35.9	100.0	96.3	119.0	68.1	16.0	14.6	47.0	44.9	103.0	92.4
Cracow 2004	33.0	65.5	19.0	27.4	5.0	23.0	65.0	114.0	34.0	62.1	18.0	23.5	11.0	27.0	65.0	102.0
Dublin	77.2	65.0	12.0	16.0	60.4	38.0	102.0	87.0	57.5	58.0	13.8	16.0	40.2	29.0	79.7	81.0
Gothenburg 2004	83.4	75.0	22.4	23.1	49.3	35.6	127.2	115.8	89.0	78.7	19.9	18.3	62.9	50.6	126.1	111.8
Hamburg	na	59.0	na	27.0	na	15.5	na	104.3	na	69.0	na	24.8	na	11.3	na	92.6
Innsbruck 2004	71.6	73.0	35.3	38.0	10.6	11.0	125.9	129.0	86.6	90.0	25.2	26.0	44.8	37.0	130.0	128.0
Le Havre 2004	71.8	72.0	23.4	28.2	28.8	26.6	108.8	120.9	76.3	79.7	17.6	23.1	51.4	52.7	106.6	134.2
Lille 2003	72.9	64.1	40.7	31.6	15.7	12.3	84.5	125.9	75.2	73.4	38.0	26.0	43.5	38.8	161.8	126.7
Lisbon 2004	82.4	76.0	26.7	23.0	43.6	44.0	131.3	118.0	87.7	79.0	23.6	22.0	48.2	44.0	130.2	114.0
Ljubljana 2004	77.3	77.0	2.1	46.6	10.3	8.6	139.9	158.0	68.4	78.0	2.0	35.8	8.3	27.2	127.3	129.3
London	na	47.1	na	24.3	na	11.0	na	88.0	na	48.0	na	20.8	na	17.9	na	83.1
Lyon 2004	72.9	69.5	39.8	41.2	6.0	7.5	144.0	149.0	88.6	61.4	28.6	37.9	42.3	4.9	135.6	135.0
Madrid 2004	61.5	61.0	29.5	28.0	13.9	18.0	113.9	106.0	71.2	70.0	17.2	16.0	45.5	46.0	103.8	97.0
Marseille 2004	85.7	90.7	33.8	39.5	33.7	34.0	142.5	166.0	97.9	102.5	21.2	27.0	65.9	66.0	130.9	154.0
Paris 2004	68.0	66.0	33.6	37.0	12.9	14.0	126.9	140.0	80.3	78.0	25.0	31.0	43.5	35.0	128.5	142.0
Prague	na	74.0	na	32.0	na	31.0	na	133.0	na	87.0	na	26.0	na	49.0	na	134.0
Rome 2002	82.1	90.8	33.8	44.0	26.4	24.3	138.0	170.3	92.5	105.4	19.8	28.2	60.6	57.8	122.3	155.6
Rotterdam	na	64.6	na	25.1	na	9.0	na	121.4	58.4	73.2	23.3	19.1	7.2	37.5	100.0	115.3
Rouen 2004	67.5	71.4	27.4	32.4	22.2	26.9	113.3	141.3	75.3	83.2	20.8	28.6	47.4	46.2	116.5	147.7
Seville 2003	77.4	71.0	27.0	24.0	39.0	39.7	128.1	117.7	84.3	73.1	16.7	18.5	58.6	39.8	116.0	105.3
Stockholm 2004	70.0	76.0	17.7	22.4	45.5	38.8	104.6	114.1	73.4	86.0	17.7	17.0	48.2	55.0	108.2	114.0
Toulouse 2004	na	79.0	na	31.0	na	33.0	na	139.0	na	91.0	na	23.0	na	63.0	na	132.0
Valencia 2004	66.0	67.8	22.9	25.3	29.0	24.6	102.8	108.5	70.6	69.8	15.8	17.3	44.7	45.3	96.3	100.0
Vienna 2004	na	72.0	na	35.0	na	16.0	na	124.0	79.1	90.0	16.4	22.0	52.1	43.0	106.7	121.0

1. SD: Standard deviation

2. P5: 5th percentile of the distribution of the pollutant

4. Definition of summer: 01 April to 30 September (for Cracow data available only until July 2004)

na.: not available

Figure 1 shows the annual mean levels and 5th and 95th percentiles of the distribution of directly measured PM₁₀ for the year selected for HIA, 2001 or 2002 depending on the city (see Table 2).

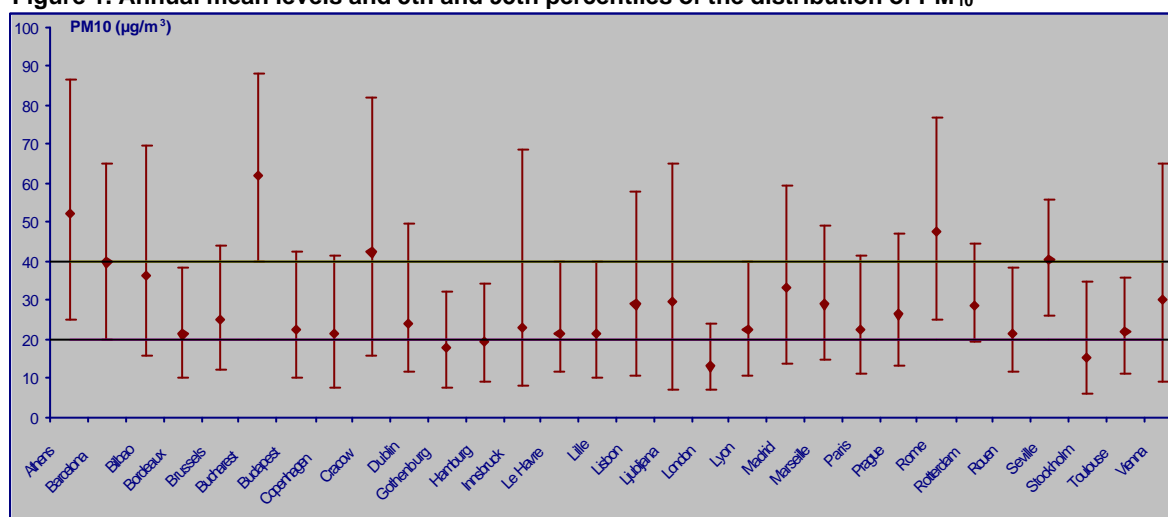
Bucharest shows the highest PM₁₀ levels (62 µg/m³) but in this city measurements are available for 4 weekdays (Monday to Thursday); this may explain the high levels observed.

Athens, Cracow, Rome and to a lesser extend Seville show PM₁₀ levels higher than the PM₁₀ annual limit value (40 µg/m³) that should not have been exceeded by 1 January 2005. Barcelona almost reaches this limit value.

Most of the cities are in the range between 40 and 20 µg/m³. Only Gothenburg, Hamburg, London and Stockholm show levels below 20 µg/m³.

Please note that the bars are slightly shifted to the right.

Figure 1. Annual mean levels and 5th and 95th percentiles of the distribution of PM₁₀



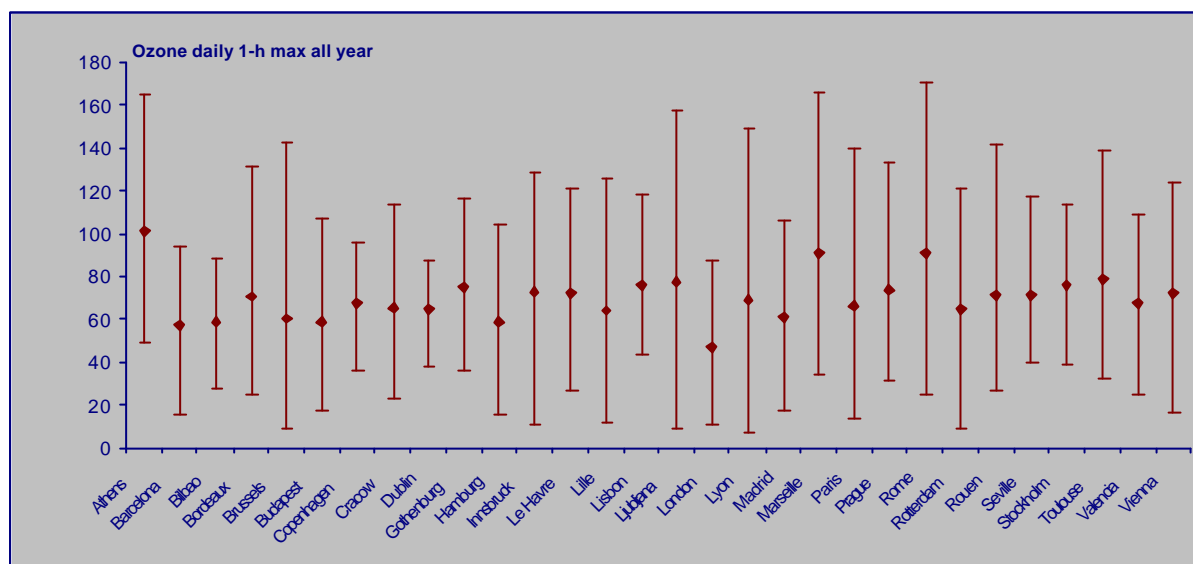
Horizontal lines indicate the European Commission (EC) PM₁₀ annual mean cut-offs of 40 µg/m³ and 20 µg/m³ respectively for 2005 and 2010.

NOTE: It is important to take into account that following the “Margin of tolerance” established in the Council Directive 1999/30/EC the accepted limit values for years 2001 and 2002 are 44.6 µg/m³ and 44.8 µg/m³, respectively.

Regarding ozone, all the cities are already below the long-term objective of the third Daughter Directive of February 2002 that regulates the target values of ozone concentration in ambient air for health protection: maximum daily 8-h mean value: $120 \mu\text{g}/\text{m}^3$. They are also below the information threshold: maximum 1-h value: $180 \mu\text{g}/\text{m}^3$.

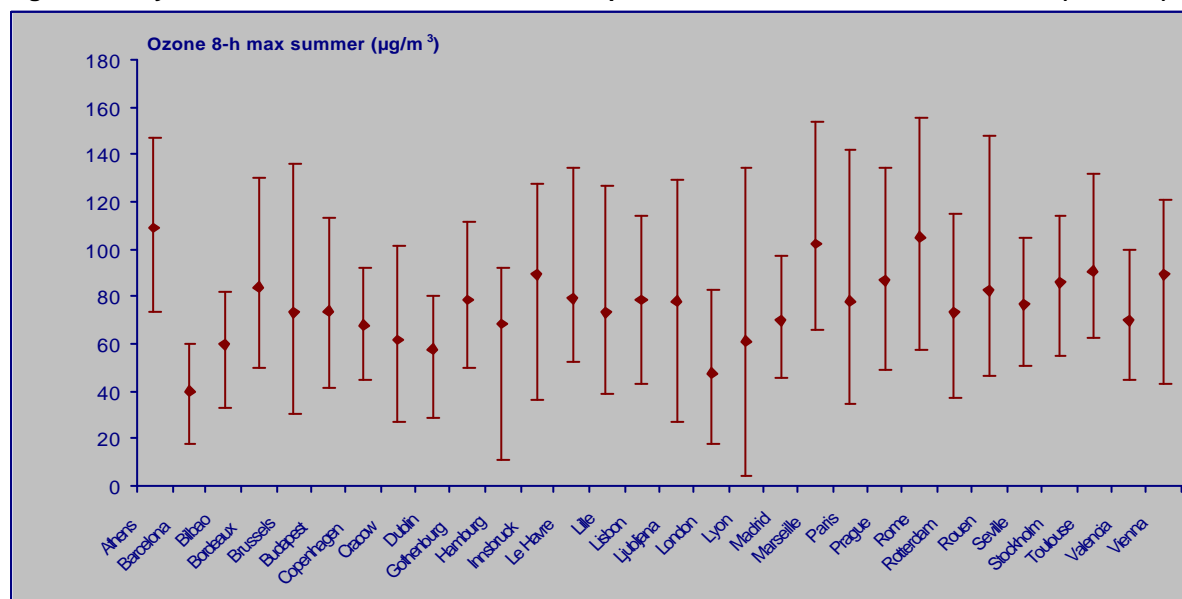
Figure 2 shows the highest daily 1-hour max levels of ozone (all year) for Athens ($101 \mu\text{g}/\text{m}^3$). Marseille and Rome follow very closely ($91 \mu\text{g}/\text{m}^3$). Most of the cities show levels higher than $60 \mu\text{g}/\text{m}^3$. The lowest levels are observed in London ($47 \mu\text{g}/\text{m}^3$).

Figure 2. Daily 1-h maximum levels and 5th and 95th percentiles of the distribution of ozone (all year)



The same patterns are observed for the maximum daily 8-hour mean levels in summer (Figure 3). Athens reaches $109 \mu\text{g}/\text{m}^3$, Rome and Marseille: 105 and $102 \mu\text{g}/\text{m}^3$ respectively and most of the cities show levels above $60 \mu\text{g}/\text{m}^3$. The lowest levels are observed in Barcelona ($40.7 \mu\text{g}/\text{m}^3$).

Figure 3. Daily 8-h maximum levels and 5th and 95th percentiles of the distribution of ozone (summer)

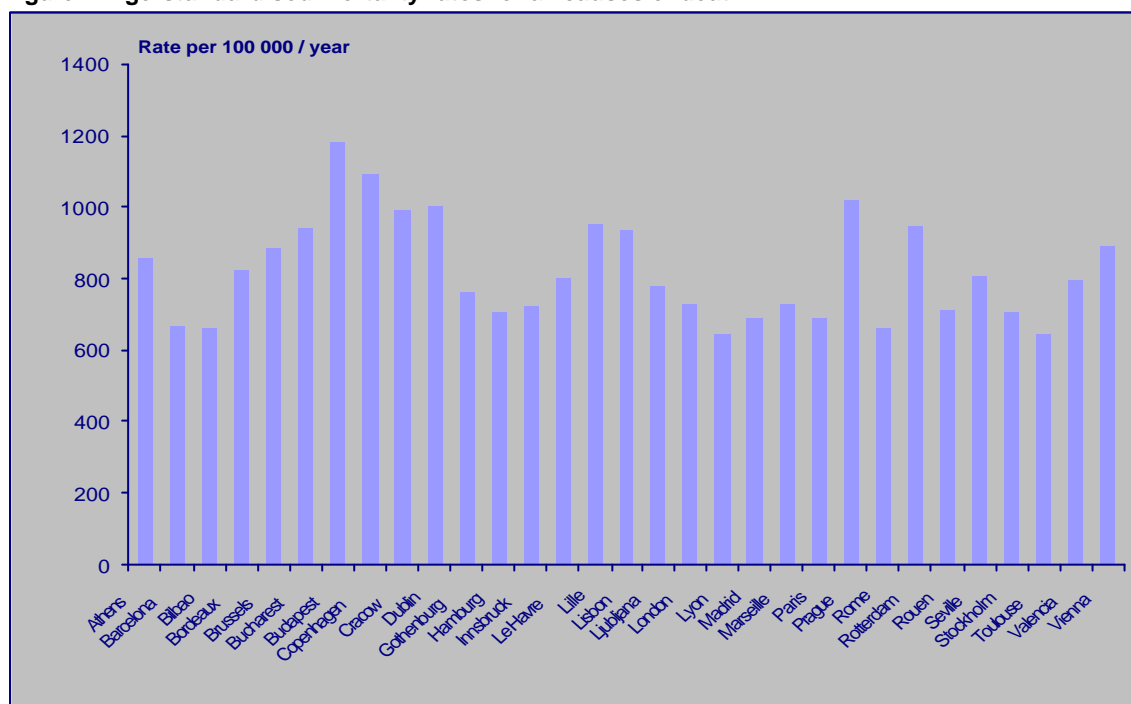


Health indicators

Mortality

Figure 4 shows the standardised mortality rates for all causes of death, including external causes, in the 31 cities. The highest rates are for Budapest, Copenhagen, Dublin and Prague (over 1000 per 100 000).

Figure 4. Age-standardised mortality rates for all causes of death



Age-standardised mortality rate per 100 000 including violent deaths using the European population for 2000 year (United Nations, 2001)²

² United Nations. Population Division Department of Economic and Social Affairs. World Population Prospects: The 2000 Revision.

Table 7 presents the daily mean and standard deviation for total, cardiovascular and respiratory mortality in the 31 cities. In terms of daily means, because London and Paris are the biggest cities, they show the biggest numbers for total mortality while London show the biggest daily mean for cardiovascular and, particularly, respiratory mortality.

Table 7. Daily mean and standard deviation for total, cardiovascular and respiratory mortality

City	All causes mortality ¹			Cardiovascular mortality ²			Respiratory mortality ³		
	Daily mean	Standard deviation	Daily rate (per 100 000)	Daily mean	Standard deviation	Daily rate (per 100 000)	Daily mean	Standard deviation	Daily rate (per 100 000)
Athens	76.0	11.0	2.4	38.3	7.6	1.2	6.0	2.8	0.2
Barcelona	39.3	8.2	2.6	13.2	4.4	0.9	4.4	2.6	0.3
Bilbao	15.9	4.2	2.3	5.1	2.4	0.7	1.7	1.4	0.3
Bordeaux	12.7	3.8	2.1	4.3	2.1	0.7	0.7	0.9	0.1
Brussels	25.0	5.2	2.6	9.6	3.1	1.0	3.1	1.7	0.3
Bucharest	59.0	na.	3.0	28.5	na.	1.4	2.2	na.	1.1
Budapest	63.1	9.0	3.6	32.9	6.4	1.9	1.9	1.4	0.1
Copenhagen	18.9	4.5	3.2	7.4	2.7	1.3	2.0	1.4	0.3
Cracow	17.7	5.0	2.3	9.5	3.8	1.3	0.6	0.9	0.1
Dublin	11.3	3.6	2.3	4.5	2.2	0.9	1.8	1.4	0.4
Gothenburg	12.6	3.7	2.7	5.6	2.4	1.2	1.0	1.0	0.2
Hamburg	44.2	7.5	2.6	17.7	4.4	1.0	3.1	1.8	0.2
Innsbruck	2.8	na.	2.5	1.3	na.	1.2	0.2	na.	0.1
Le Havre	5.7	2.4	2.2	1.6	1.2	0.6	0.4	0.7	0.2
Lille	20.7	4.6	1.9	6.1	2.5	0.6	1.6	1.3	0.1
Lisbon	48.3	10.8	2.6	21.3	6.3	1.7	4.1	2.4	0.3
Liubliana	7.3	2.8	2.7	3.0	1.6	1.1	1.3	0.6	0.5
London	144.1	18.4	2.0	57.9	9.6	0.8	22.1	6.4	0.3
Lyon	15.1	4.1	1.9	4.9	2.2	0.6	0.9	0.9	0.1
Madrid	71.0	12.6	2.4	23.0	5.8	0.8	9.8	4.4	0.3
Marseille	20.3	4.9	2.4	6.4	2.7	0.8	1.4	1.2	0.2
Paris	112.5	14.1	1.8	31.2	6.4	0.5	6.9	3.1	0.1
Prague	34.0	6.0	2.9	19.6	4.7	1.7	1.6	1.3	0.1
Rome	56.7	9.5	2.2	23.3	5.7	0.9	3.1	1.9	0.1
Rotterdam	16.9	na.	2.8	6.0	na.	1.0	1.8	na.	0.3
Rouen	9.7	3.3	2.2	3.1	1.8	0.7	0.6	0.7	0.1
Seville	15.0	4.1	2.2	5.7	2.5	0.8	1.0	0.8	0.1
Stockholm	29.4	6.4	2.5	13.0	4.0	1.1	2.5	2.0	0.2
Toulouse	12.0	3.6	1.8	4.0	2.0	0.6	1.0	0.8	0.1
Valencia	14.8	4.3	1.9	4.9	2.0	0.6	1.7	1.4	0.2
Vienna	44.1	8.1	2.8	23.6	5.8	1.5	2.2	1.5	0.1

na.: not available

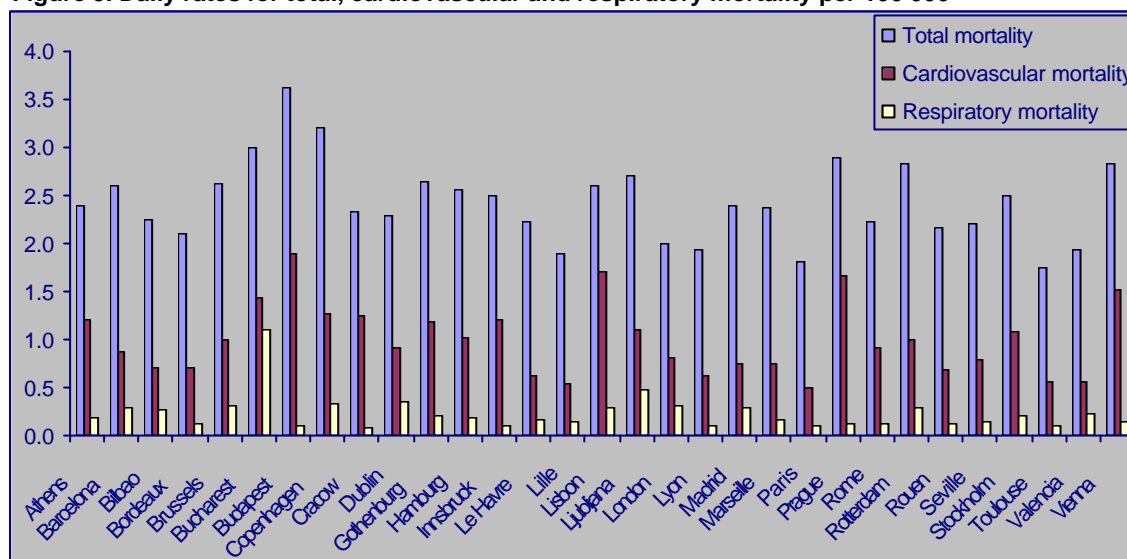
¹ Total mortality excluding external causes (ICD9 < 800 - ICD10 A00-R99)

² Cardiovascular mortality (ICD9 390-459 - ICD10 I00-I99)

³ Respiratory mortality (ICD9 460-519 - ICD10 J00-J99)

Figure 5 shows the daily death rates per 100 000 in each city. Bucharest, Budapest, Copenhagen, Prague, Rotterdam and Vienna show the highest daily rates for total mortality. The highest daily rates for cardiovascular mortality are for Budapest, Lisbon, Prague and Vienna. Bucharest shows the highest respiratory daily rates.

Figure 5. Daily rates for total, cardiovascular and respiratory mortality per 100 000



Post-neonatal mortality

Because the present HIA focus mainly on children, three post-neonatal mortality indicators were studied.

As shown in table 8 and figure 6, the highest annual rates (>500 per 100 000) for total post-neonatal mortality (children between 1 month and 1 year) are for Bucharest and Budapest. Innsbruck and Vienna also shows rates close to or higher than 500 per 100 000 but for children below 1 year. Although Athens shows the highest post-neonatal respiratory mortality rates (49 per 100 000) but note that they are for children below 1 year. Prague shows the highest post-neonatal respiratory mortality rates for children 1 month to 1 year (31 per 100 000). Dublin shows the highest post-neonatal sudden infant death syndrome rates (89.9 per 100 000).

Table 8. Annual deaths and annual death rates per 100 000 for total and respiratory post-neonatal mortality and sudden infant death syndrome

City	Total postneonatal mortality ¹		Postneonatal respiratory mortality ²		Postneonatal Sudden Infant Death Syndrome ³	
	Annual deaths	Annual rate (per 100 000)	Annual deaths	Annual rate (per 100 000)	Annual deaths	Annual rate (per 100 000)
Athens	47.0	153.6	15.0	49.0	2.0	6.5
Barcelona	10.0	78.8	2.0	15.8	1.0	7.9
Bilbao	5.0	99.1	0.0	0.0	0.0	0.0
Bordeaux	10.0	154.1	0.0	0.0	1.0	15.4
Brussels	24.0	157.0	1.0	6.5	8.0	52.3
Bucharest	75.0	500.0	na.	na.	na.	na.
Budapest	92.0	663.6	2.0	14.4	1.0	7.2
Copenhagen	11.0	133.1	1.0	12.1	0.0	0.0
Cracow	11.0	183.4	1.0	16.7	3.0	50.1
Dublin	15.0	269.6	0.0	0.0	5.0	89.9
Gothenburg	3.0	51.3	0.0	0.0	1.0	17.1
Hamburg	25.0	175.0	0.0	0.0	8.0	56.0
Innsbruck	4.9	484.0	0.0	0.0	0.0	0.0
Le Havre	8.0	256.9	0.0	0.0	2.0	64.2
Lille	22.0	151.1	0.0	0.0	10.0	0.9
Lisbon	42.0	206.5	2.0	9.8	3.0	14.7
Liubliana	5.0	221.9	0.0	0.0	0.0	0.0
London	189.8	181.0	14.6	13.9	29.2	27.9
Lyons	15.0	160.8	0.0	0.0	2.0	21.4
Madrid	54.0	190.2	na.	na.	na.	na.
Marseille	14.0	145.2	0.0	0.0	2.0	20.7
Paris	150.0	183.9	7.0	8.6	30.0	36.8
Prague	11.0	113.6	3.0	31.0	na.	na.
Rome	20.0	92.4	1.0	4.6	0.0	0.0
Rotterdam	15.0	198.3	0.0	0.0	0.0	0.0
Rouen	8.0	148.3	0.0	0.0	2.0	37.1
Seville	11.0	161.0	0.0	0.0	2.0	29.0
Stockholm	12.0	77.0	1.0	6.4	0.0	0.0
Toulouse	9.0	123.1	1.0	13.6	2.0	27.2
Valencia	7.0	127.3	1.0	18.2	1.0	18.2
Vienna	94.0	600.3	0.0	0.0	8.0	51.1

na.: not available

52.6

123.2

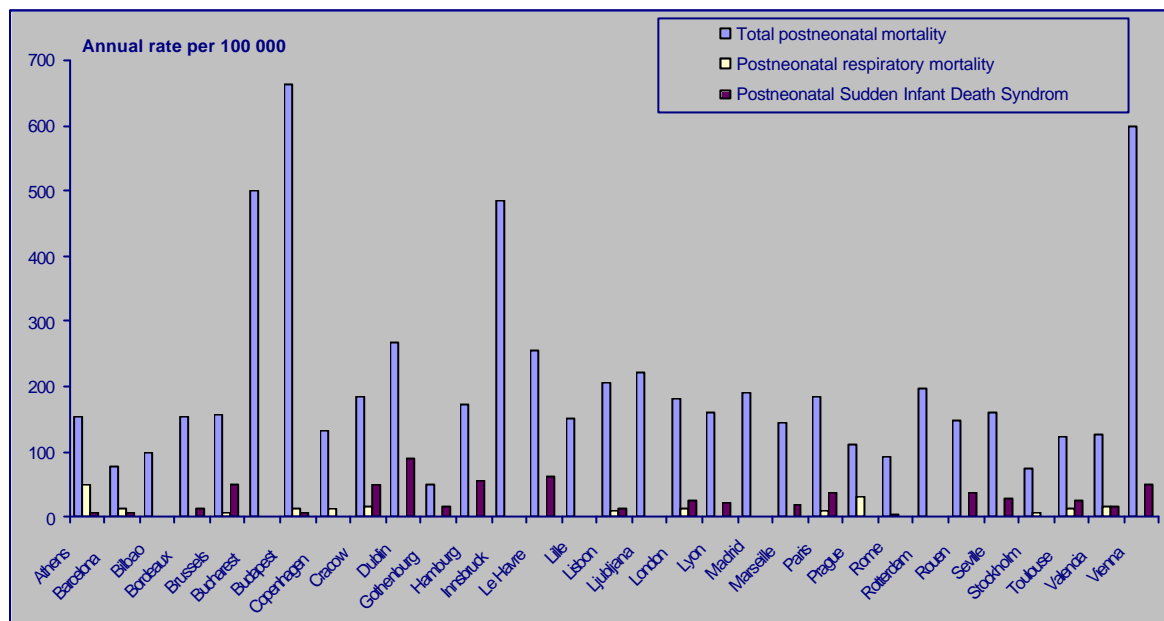
¹Total postneonatal mortality include all causes

²Postneonatal respiratory mortality (ICD9 460-519 - ICD10 J00-J99)

³Postneonatal Sudden Infant Death Syndrome Mortality (ICD9 798.0 - ICD10 R95)

For Athens, Barcelona, Innsbruck, Madrid and Vienna: population data and death rates for 0-1 year

Figure 6. Annual deaths rates per 100 000 for total and respiratory post-neonatal mortality and sudden infant death syndrome



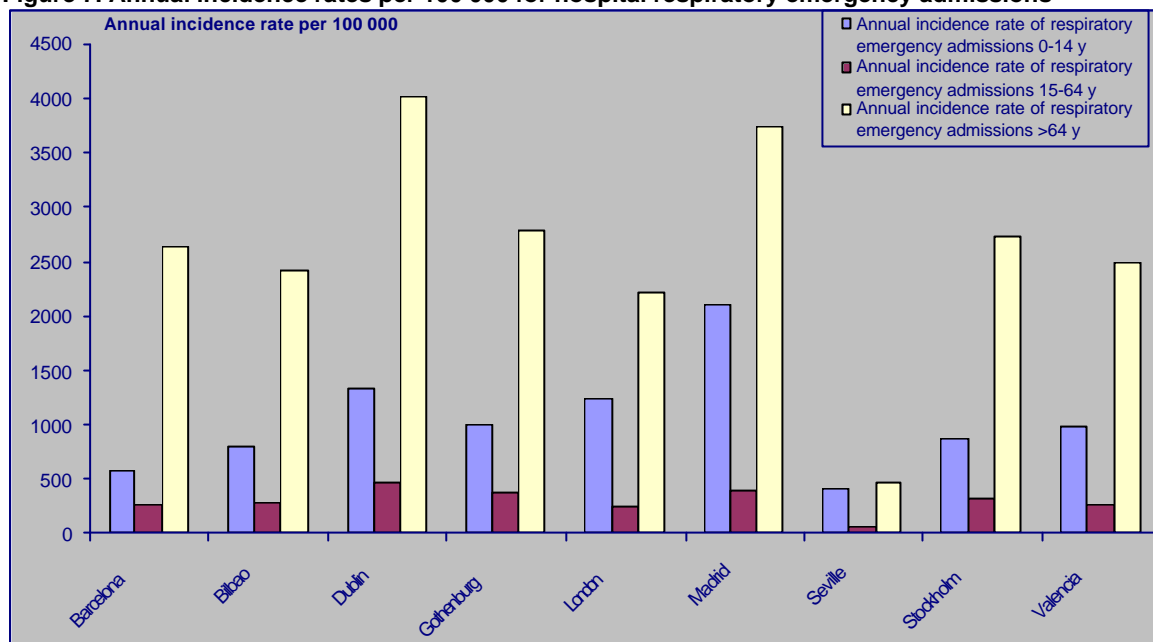
Hospital admissions

Twenty-seven cities provided data on hospital respiratory admissions. The main problem for comparability remains the differences in the availability of information in the registries. The information sources used in Barcelona, Bilbao, Dublin, Gothenburg, London, Madrid, Seville, Stockholm and Valencia allowed selecting emergency admissions. Yet, for Bordeaux, Brussels, Copenhagen, Hamburg, Innsbruck, Le Havre, Lille, Lisbon, Ljubljana, Lyon, Marseille, Paris, Prague, Rome, Rotterdam, Rouen, Toulouse and Vienna, it was not possible to distinguish between emergency and total admissions.

Athens, Bucharest, Budapest and Cracow have not estimated the impact on hospital admissions.

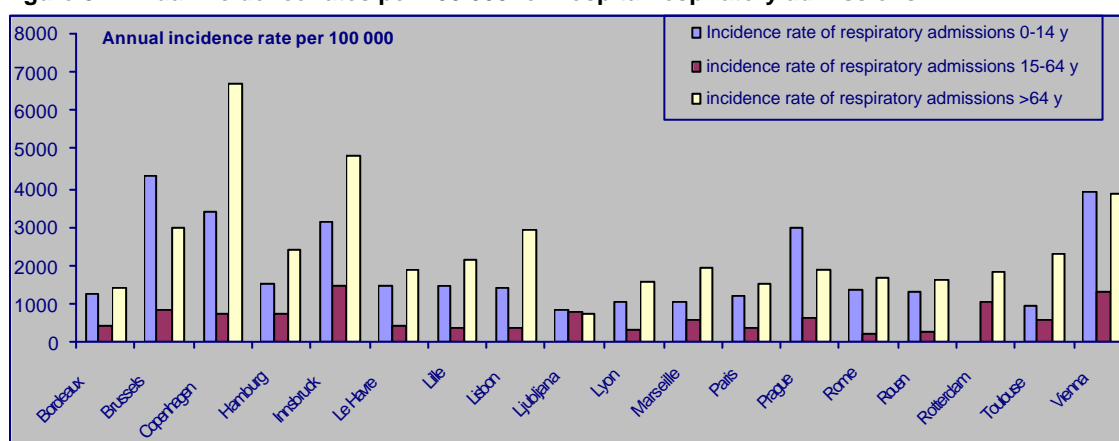
For the nine cities that could provide emergency respiratory admissions (Figure 7), the highest rates for children 0-14 years was observed in Madrid (2109 per 100 000), while the rates for people over 64 years were the highest for Dublin (4015 per 100 000).

Figure 7. Annual incidence rates per 100 000 for hospital respiratory emergency admissions



For the 18 cities that provided general respiratory admissions, the highest rates for children 0-14 years were observed in Brussels (4310 per 100 000), Copenhagen (3385 per 100 000), Innsbruck (3126 per 100 000) and Vienna (3916 per 100 000). Copenhagen shows the highest rates (6735 per 100 000) for respiratory admissions in people over 64 years followed by the Austrian cities.

Figure 8. Annual incidence rates per 100 000 for hospital respiratory admissions



Other morbidity outcomes

Emergency room visits for asthma in <18 years

This indicator was available only in four of the 31 cities. The daily rate was the highest for Gothenburg (0.7 per 100 000), it was 0.3 per 100 000 in Brussels, 0.1 per 100 000 in Copenhagen, and 0.4 per 100 000 in Stockholm.

Cough and lower respiratory symptoms in children

Information on these outcomes was not available on a routine basis in any city except London.

Health Impact Assessment

Summary findings of HIAs in terms of potential reductions in the health impacts of outdoor air pollution

Thirty-one cities in 18 countries participated in this HIA. The following tables summarise the HIA findings in terms of number of anticipated health events and rates per 100 000 that, all other things being equal, could be potentially reduced for different scenarios of PM₁₀ and ozone reductions. For the outcomes for which a population baseline frequency measure was not available (cough, lower respiratory symptoms) or was not comparable between cities (hospital respiratory admissions and emergency-room visits for asthma), an attributable number of cases could not be calculated. Instead, an attributable fraction (AF), expressed in % was calculated.

Regarding exposure to PM₁₀, as a reminder, in Apheis-3, a reduction of PM₁₀ levels by 5 µg/m³ would be associated with a decrease of 2 deaths per 100 000 on average for all causes-mortality (17 deaths for long-term exposure), 1 death per 100 000 for cardiovascular mortality and 0.5 death per 100 000 for respiratory mortality in the general population. In ENHIS-1, we completed this picture with the impact on postneonatal mortality (children 1 month-1 year). A reduction of PM₁₀ levels by 5 µg/m³ would be associated with a decrease of 4.7 deaths per 100 000 children on average for total postneonatal mortality, 1.4 death per 100 000 children for respiratory postneonatal mortality and 1.8 deaths per 100 000 children for sudden infant death syndrome (table 9).

Table 9. Potential benefits of reducing corrected¹ PM₁₀ levels. Absolute numbers and deaths rates (per 100 000 children).

POSTNEONATAL MORTALITY	PM ₁₀ reduction ¹	Number of attributable cases per year	95% CI		Annual rates per 100 000 ²	95% CI	
	Annual mean levels						
Total	by 5 µg/m ³	23.2	10.7	36.0	4.73	2.18	7.34
	to 20 µg/m ³	55.6	24.9	88.9	14.64	6.57	23.40
	to 40 µg/m ³	15.3	6.9	24.3	18.07	8.14	28.75
Respiratory	by 5 µg/m ³	4.7	2.3	7.2	1.40	0.68	2.15
	to 20 µg/m ³	13.1	5.3	24.8	5.83	2.36	10.99
	to 40 µg/m ³	6.7	2.9	11.6	11.42	4.92	19.95
Sudden Infant Deaths Syndrome	by 5 µg/m ³	6.7	3.9	9.4	1.77	1.04	2.48
	to 20 µg/m ³	9.3	5.4	13.3	3.29	1.90	4.72
	to 40 µg/m ³	0.7	0.4	1.1	1.68	0.95	2.45

¹ PM₁₀ reference papers for HIA on postneonatal mortality use gravimetric methods to measure PM₁₀. If the local air quality network uses automatic methods (TEOM or other) a correction factor is required to compensate for loss of volatile compounds: if available, a local correction factor recommended by the air quality network or, by default, the European factor 1.3.

² Annual rates per 100.000 have been calculated for the specific population of each city in which each scenario is applicable.

Regarding morbidity, a reduction of short-term exposure to PM₁₀ by 5 µg/m³ would be associated with a decrease of 2% for cough and lower respiratory symptoms and 0.5% for hospital respiratory admissions <15 years (table 10).

Table 10. Potential benefits of reducing measured PM₁₀ levels. Attributable fractions and 95%CI.

MORBIDITY	PM ₁₀ reduction	Attributable fraction (%)	95% CI	
	Daily levels			
Cough 5-17 y	by 5 µg/m ³	2.0%	1.0%	2.5%
	to 20 µg/m ³	7.0%	3.6%	8.6%
	to 50 µg/m ³	3.7%	1.9%	4.5%
LRS 5-17 y	by 5 µg/m ³	2.0%	1.0%	2.9%
	to 20 µg/m ³	7.0%	3.6%	10.1%
	to 50 µg/m ³	3.7%	1.9%	5.3%
Hospital respiratory admissions <15 y	by 5 µg/m ³	0.5%	0.0%	1.0%
	to 20 µg/m ³	1.8%	0.0%	3.8%
	to 50 µg/m ³	1.0%	0.0%	2.0%

Regarding ozone, a reduction by 10 µg/m³ of daily maximum 8-hour mean levels in summer would be associated with a decrease in total mortality of 1.28 deaths per 100 000, 0.75 death per 100 000 for cardiovascular mortality and 0.39 death per 100 000 for respiratory mortality in the general population (table 11).

Table 11. Potential benefits of reducing ozone daily levels. Absolute numbers and deaths rates (per 100 000 inhabitants).

MORTALITY	OZONE reduction	Number of attributable cases per year	95% CI		Annual rates per 100 000	95% CI	
	Daily 8-h max						
Total	by 10 µg/m ³	566.7	310.8	950.6	1.28	0.70	2.15
	to 120 µg/m ³	79.9	43.8	134.3	0.21	0.12	0.36
Cardiovascular	by 10 µg/m ³	333.2	159.3	528.7	0.75	0.36	1.20
	to 120 µg/m ³	47.6	22.7	75.8	0.13	0.06	0.20
Respiratory	by 10 µg/m ³	173.9	113.9	232.4	0.39	0.26	0.53
	to 120 µg/m ³	21.1	13.7	28.2	0.06	0.04	0.08

A reduction of daily 1-hour maximum levels of ozone (all year) by 10 µg/m³ would be associated with a decrease of 1.14% in emergency room visits for asthma <18 years. A reduction by 10 µg/m³ of daily maximum 8-hour mean levels in summer would be associated with a decrease of 0.10% in hospital respiratory admissions 15-64 years and 0.5% in hospital respiratory admissions >64 years (table 12).

Table 12. Potential benefits of reducing ozone daily levels. Attributable fractions and 95%CI.

MORBIDITY	OZONE reduction	Attributable fraction (%)	95% CI	
	Daily 1-h max			
Emergency room visits for asthma <18 y	by 10 µg/m ³	1.14%	0.67%	1.60%
	to 180 µg/m ³	0.04%	0.02%	0.06%
	Daily 8-h max			
Hospital respiratory admissions 15-64 y	by 10 µg/m ³	0.10%	0.00%	1.19%
	to 120 µg/m ³	0.02%	0.00%	0.20%
Hospital respiratory admissions > 64 y	by 10 µg/m ³	0.50%	0.00%	1.19%
	to 120 µg/m ³	0.08%	0.00%	0.20%

These findings show that for comparable health outcomes, the greatest benefits are for children.

All the findings are detailed in the following pages.

Note: it is of crucial importance to note that the HIA findings shown in the tables above are for different scenarios and for different air-pollution indicators. They must not be added together because the impacts provided by one air-pollution indicator are already included in another indicator and some of the impacts provided in one scenario are already included in another scenario. Besides this caution statement, it is also interesting to point out that the core of the evidence suggests that the short-term effects of ozone and PM₁₀ are fairly independent from each other.

1. Health Impact Assessment Findings for PM₁₀

1.1. PM₁₀ and postneonatal mortality (total, respiratory and sudden infant death syndrome-SIDS)

1.1.1 Reduction of the annual mean value of PM₁₀ to a level of 40 µg/m³ (Limit of 1999/30/EC Directive for 2005)

Figures 9 to 11 show that, in terms of total postneonatal mortality, four of the 31 cities (Athens, Bucharest, Rome and Seville) would get the highest benefit of a reduction of the annual mean value of PM₁₀ to a level of 40 µg/m³. Athens and Rome would show the greatest benefit in terms of respiratory postneonatal mortality and Athens, Cracow and Seville would benefit from this scenario for sudden infant death syndrome. The health benefits of this scenario for the other cities are extremely low.

Figure 9. Reduction of annual mean value of PM₁₀ to a level of 40 µg/m³ and impact on total postneonatal mortality. Number of “premature” deaths per 100 000 per year.

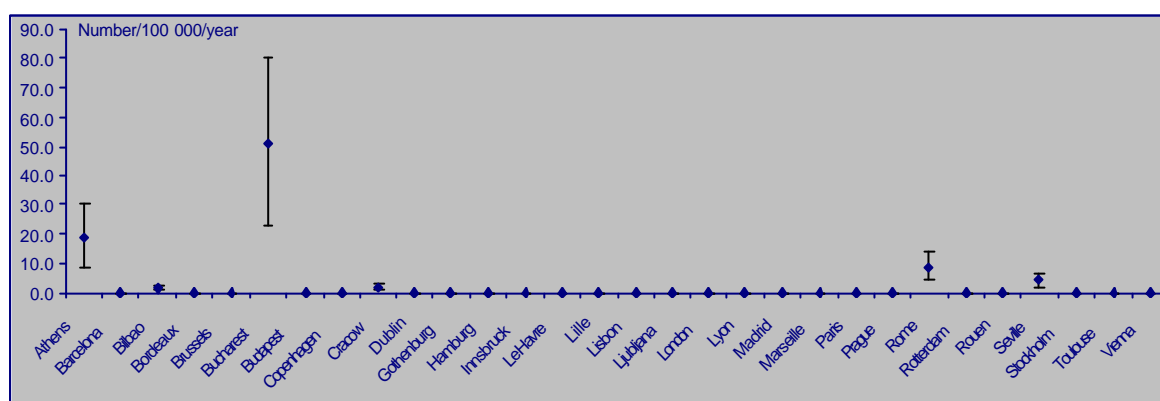


Figure 10. Reduction of annual mean value of PM₁₀ to a level of 40 µg/m³ and impact on respiratory postneonatal mortality. Number of “premature” deaths per 100 000 per year.

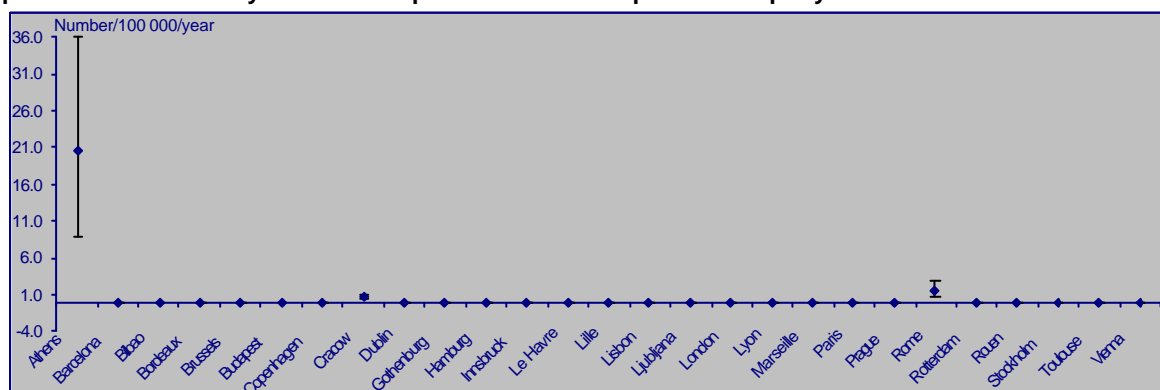
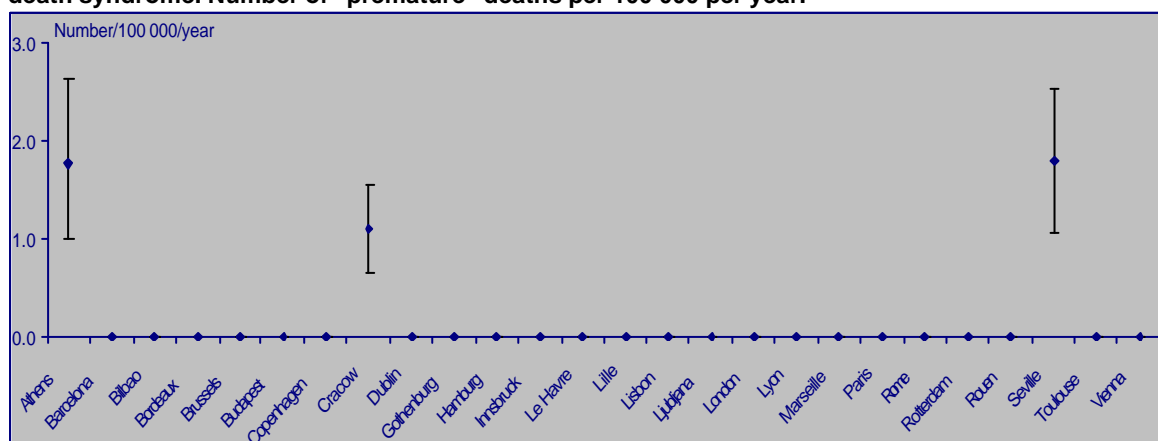


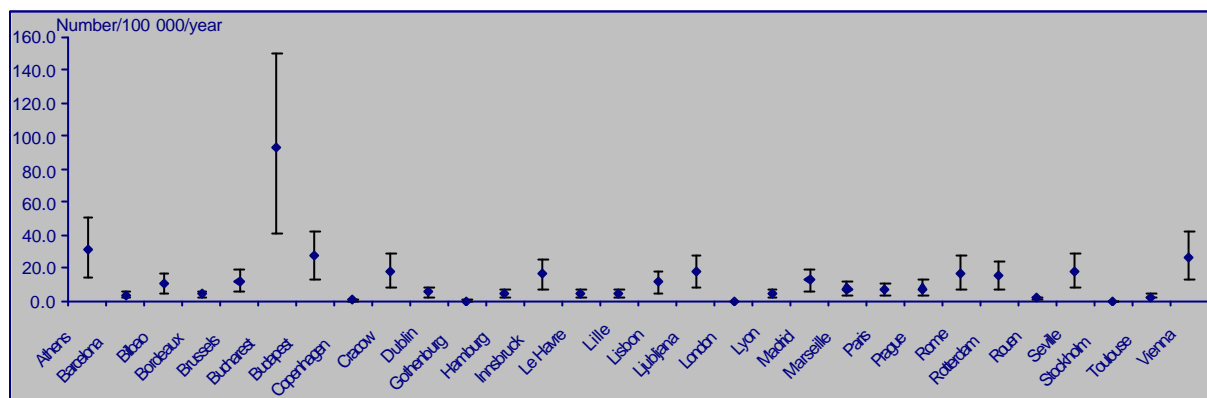
Figure 11. Reduction of annual mean value of PM₁₀ to a level of 40 µg/m³ and impact on Sudden infant death syndrome. Number of “premature” deaths per 100 000 per year.



1.1.2. Reduction of the annual mean value of PM₁₀ to a level of 20 µg/m³ (Limit of 1999/30/EC Directive for 2010)

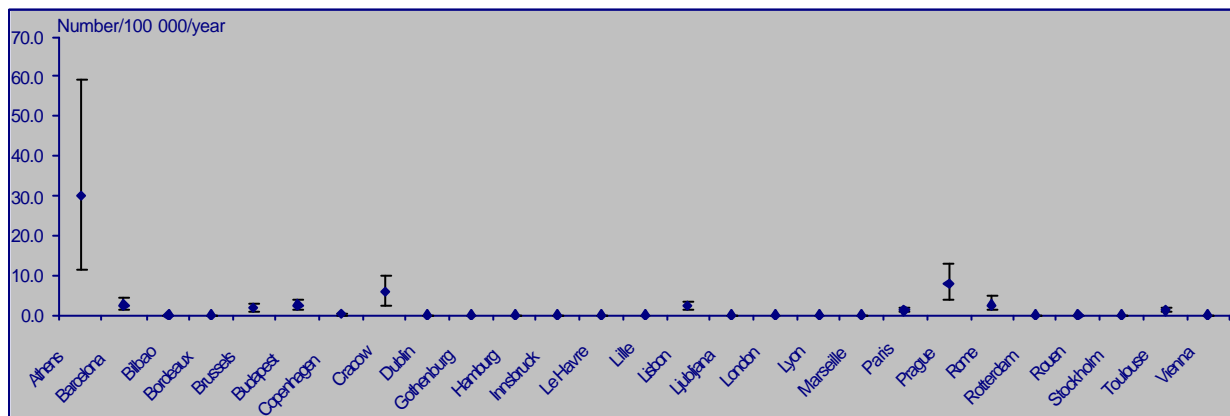
If the annual mean value of PM₁₀ would decrease to a level of 20 µg/m³, Bucharest would show the highest decrease in the number of total postneonatal deaths per 100 000 children. The health benefits for the other cities are higher than in the previous scenario (figure 12).

Figure 12. Reduction of annual mean value of PM₁₀ to a level of 20 µg/m³ and impact on total postneonatal mortality. Number of “premature” deaths per 100 000 per year.



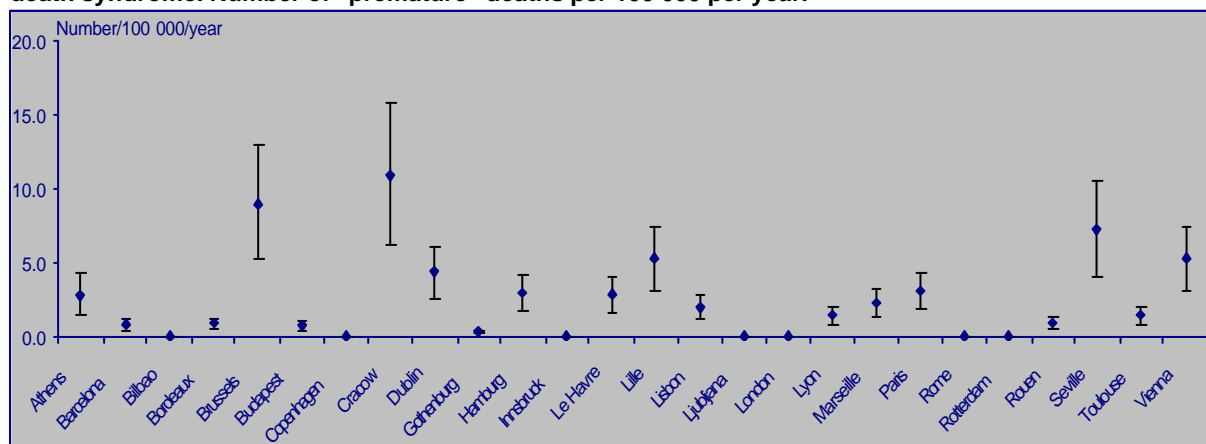
Regarding respiratory postneonatal mortality, Athens would show the greatest benefit. The health benefits of this scenario for the other cities are lower (figure 13).

Figure 13. Reduction of annual mean value of PM₁₀ to a level of 20 µg/m³ and impact on respiratory postneonatal mortality. Number of “premature” deaths per 100 000 per year.



All other things being equal, Cracow and Brussels would show the greatest decrease in the number of sudden infant death syndromes. Other cities would get a smaller benefit for this scenario (figure 14).

Figure 14. Reduction of annual mean value of PM₁₀ to a level of 20 µg/m³ and impact on Sudden infant death syndrome. Number of “premature” deaths per 100 000 per year.



1.1.3. Reduction by 5 $\mu\text{g}/\text{m}^3$ of the annual mean value of PM_{10}

If, all other things being equal, the annual mean value of PM_{10} was reduced by 5 $\mu\text{g}/\text{m}^3$, the consequent reduction in the number of total postneonatal deaths would be the highest for Bucharest and Budapest and Vienna (but for children <1 year) (figure 15). Athens and Prague would show the greatest benefits for respiratory postneonatal mortality (figure 16) and Dublin would show the biggest benefits for sudden infant death syndrome (figure 17).

Figure 15. Reduction of annual mean value of PM_{10} by 5 $\mu\text{g}/\text{m}^3$ and impact on total postneonatal mortality. Number of “premature” deaths per 100 000 per year.

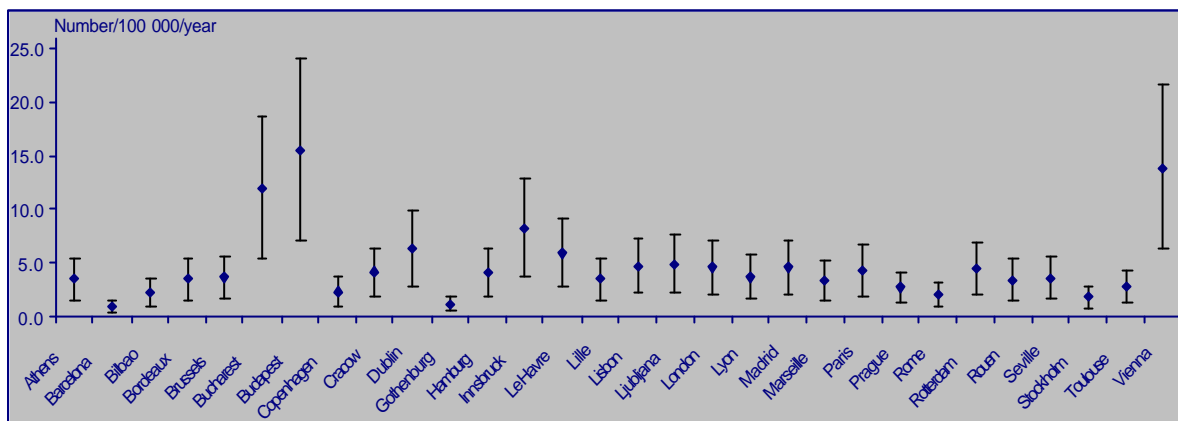


Figure 16. Reduction of annual mean value of PM_{10} by 5 $\mu\text{g}/\text{m}^3$ and impact on respiratory postneonatal mortality. Number of “premature” deaths per 100 000 per year.

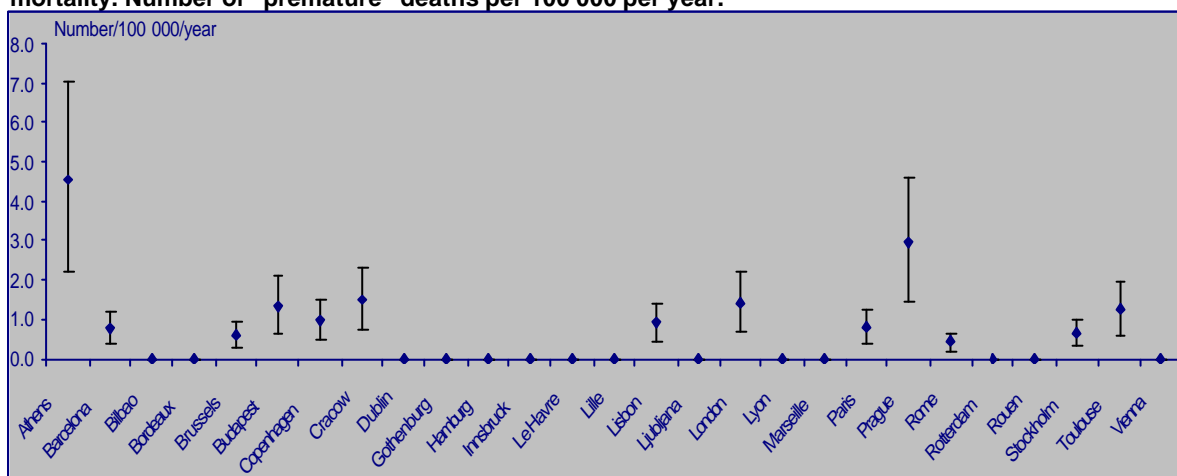
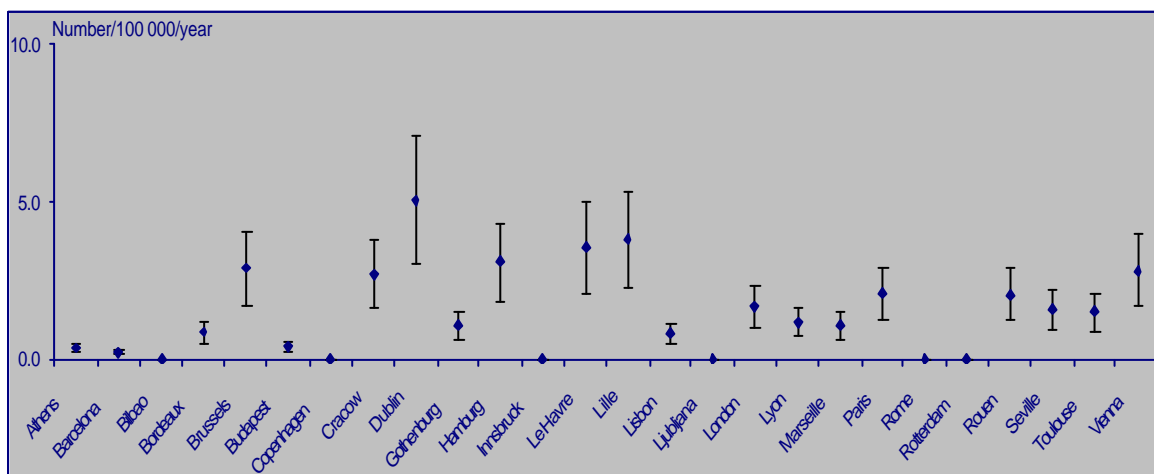


Figure 17. Reduction of annual mean value of PM_{10} by 5 $\mu\text{g}/\text{m}^3$ and impact on Sudden infant death syndrome. Number of “premature” deaths per 100 000 per year.



1.2. Short-term effects of PM₁₀ and cough and lower respiratory symptoms (5-17 years), and hospital respiratory admissions in people under 15 years (<15 years).

1.2.1 Reduction of PM₁₀ levels to a 24-hour value of 50 µg/m³ in all days exceeding this value (Limit of 1999/30/EC Directive)

For morbidity outcomes, the attributable fractions are reported here. The benefits of reducing PM₁₀ levels to a 24-hour value of 50 µg/m³ would reach more than 8% in Athens, Bucharest and Innsbruck for cough and lower respiratory symptoms. For respiratory hospital admissions < 15 years, for the same cities, the numbers would be above 2% (figures 18 to 20).

Figure 18. Reduction of PM₁₀ levels to a 24-hour value of 50 µg/m³ in all days exceeding this value (Limit of 1999/30/EC Directive). Attributable fractions and 95%CI on cough (5-17 years).

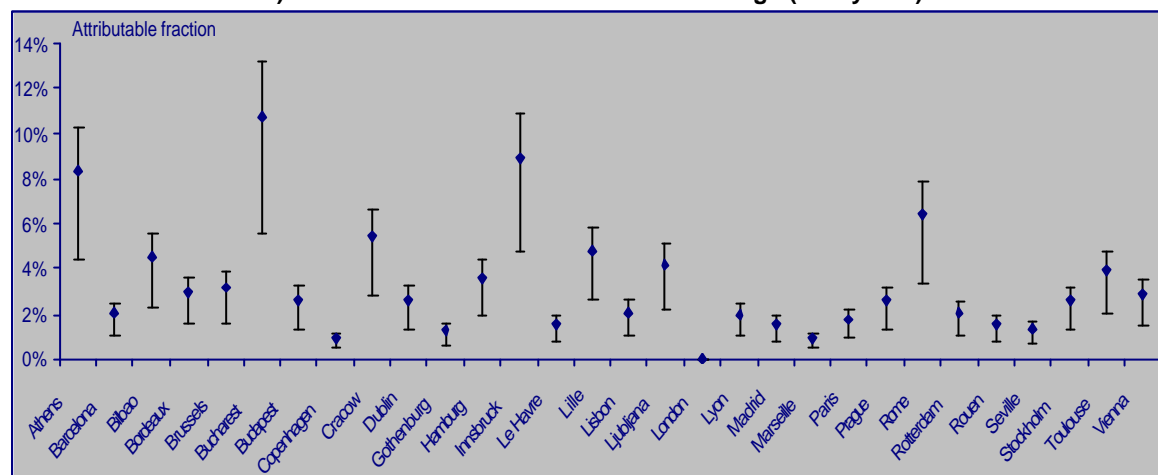


Figure 19. Reduction of PM₁₀ levels to a 24-hour value of 50 µg/m³ in all days exceeding this value (Limit of 1999/30/EC Directive). Attributable fractions and 95%CI on lower respiratory symptoms (5-17 years).

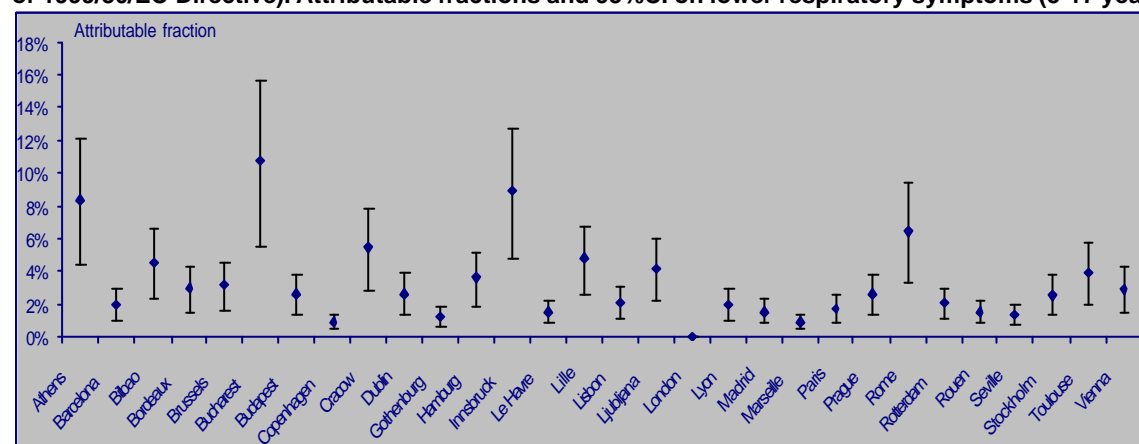
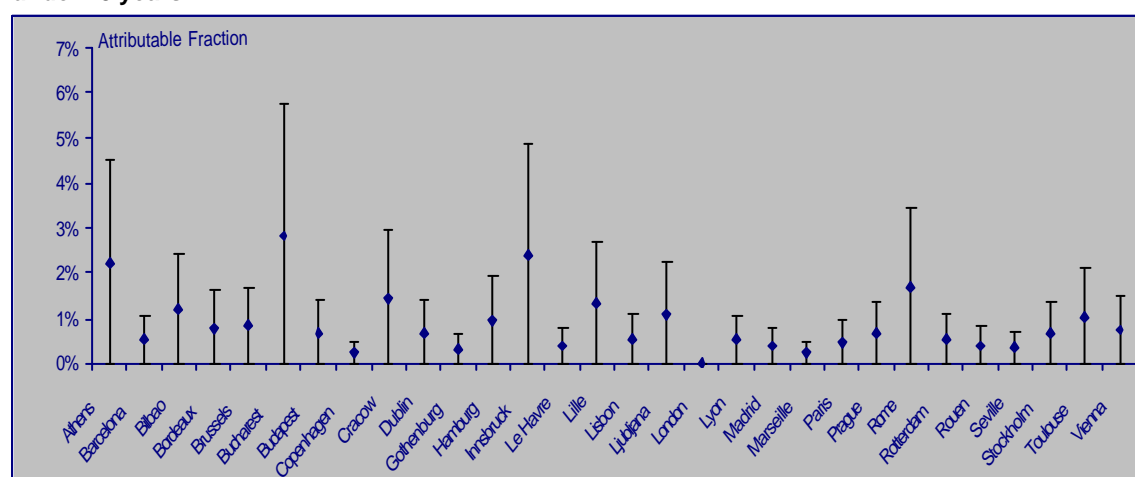


Figure 20. Reduction of PM₁₀ levels to a 24-hour value of 50 µg/m³ in all days exceeding this value (Limit of 1999/30/EC Directive). Attributable fractions and 95%CI on hospital respiratory admissions in people under 15 years.



1.2.2 Reduction of PM₁₀ levels to a 24-hour value of 20 µg/m³ in all days exceeding this value

The benefits of reducing PM₁₀ levels to a 24-hour value of 20 µg/m³ for cough and lower respiratory symptoms would be higher than 15% in Athens, Bucharest and Rome. They would exceed 4% for hospital respiratory admissions <15 years in the same cities (figures 21 to 23).

Figure 21. Reduction of PM₁₀ levels to a 24-hour value of 20 µg/m³ in all days exceeding this value. Attributable fractions and 95%CI on cough (5-17 years).

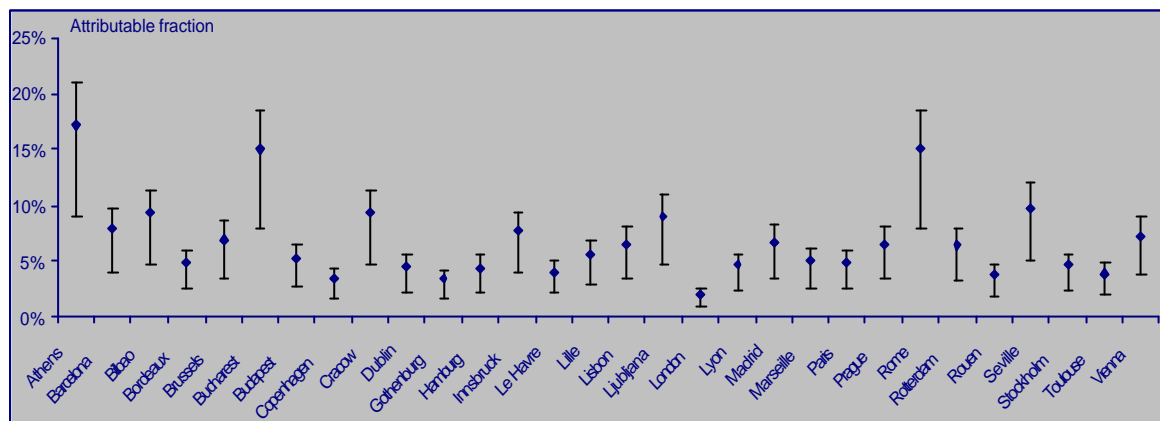


Figure 22. Reduction of PM₁₀ levels to a 24-hour value of 20 µg/m³ in all days exceeding this value. Attributable fractions and 95%CI on lower respiratory symptoms (5-17 years).

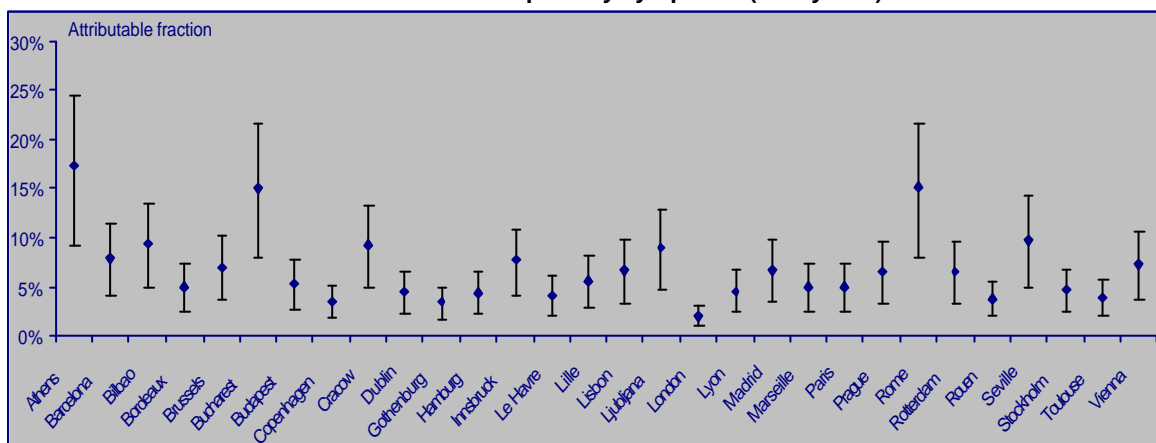
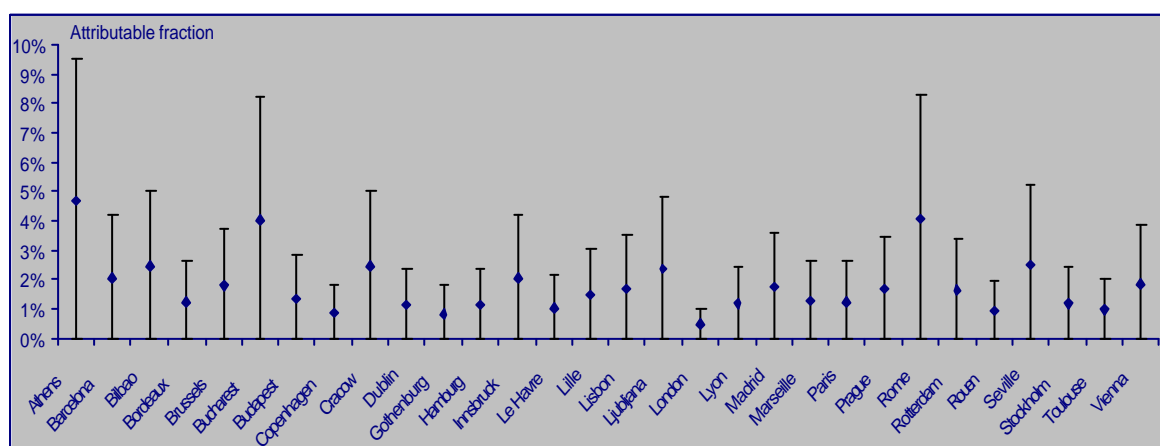


Figure 23. Reduction of PM₁₀ levels to a 24-hour value of 20 µg/m³ in all days exceeding this value. Attributable fractions and 95%CI on hospital respiratory admissions in people under 15 years.



1.2.3 Reduction by 5 $\mu\text{g}/\text{m}^3$ of all the 24-hour values of PM_{10}

The benefit of reducing PM_{10} levels all the 24-hour values by $5\mu\text{g}/\text{m}^3$ would be 2% on average for cough and lower respiratory symptoms. It would be of 0.5% on average for hospital respiratory admissions <15 years.

2. Health Impact Assessment Findings for Ozone

2.1 Daily maximum 8-hour moving average concentration and mortality in general population

2.1.1 Reduction of O_3 daily maximum 8-hour moving average concentrations to $120\mu\text{g}/\text{m}^3$ in all days exceeding this value (Limit for health protection of 2002/3/EC Directive) and impact on mortality in general population

All other things being equal, if ozone levels for all days when they exceeded this value were reduced to $120\mu\text{g}/\text{m}^3$, the greatest benefits on total mortality in the general population would be for Athens, Ljubljana, Marseille and Rome, although the numbers would be quite low for this scenario: 0.2 per 100 000 on average for all the cities. The figures would be quite similar for cardiac and respiratory mortality (figures 24 to 26).

Figure 24. Reduction of O_3 daily maximum 8-hour moving average concentrations to $120\mu\text{g}/\text{m}^3$ and impact on total mortality. Number of "premature" deaths per 100 000 per year.

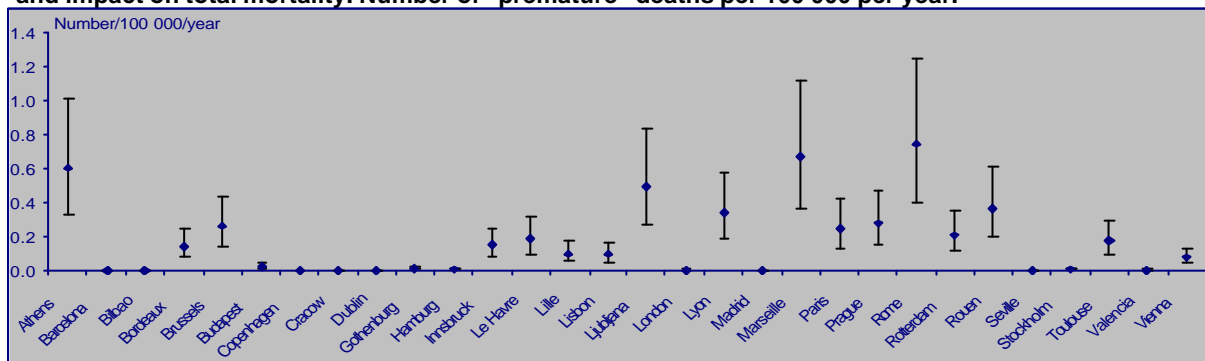


Figure 25. Reduction of O_3 daily maximum 8-hour moving average concentrations to $120\mu\text{g}/\text{m}^3$ and impact on cardiac mortality. Number of "premature" deaths per 100 000 per year.

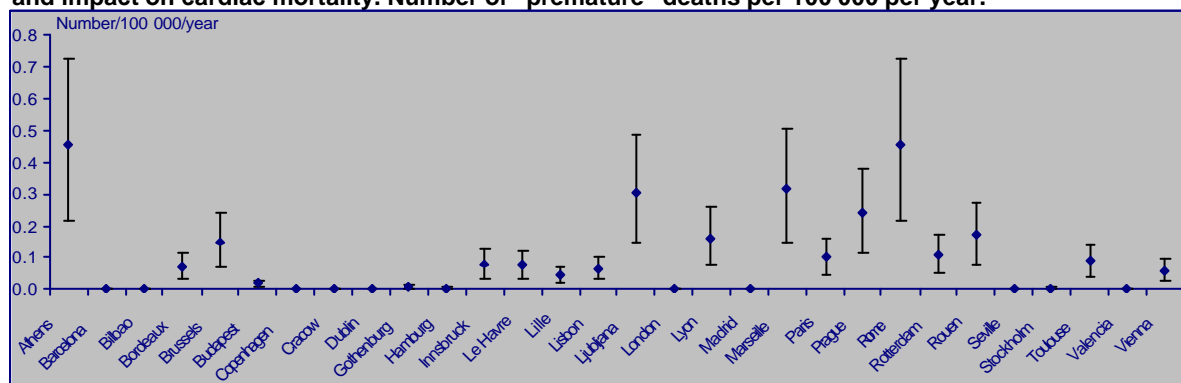
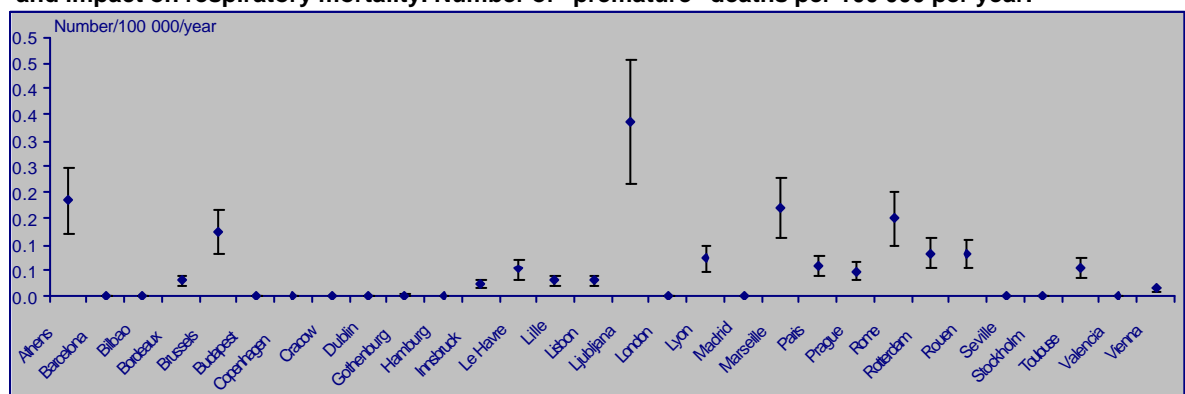


Figure 26. Reduction of O_3 daily maximum 8-hour moving average concentrations to $120\mu\text{g}/\text{m}^3$ and impact on respiratory mortality. Number of "premature" deaths per 100 000 per year.



2.1.2 Reduction by 10 µg/m³ in the daily maximum 8-hour moving average concentrations and impact on mortality in general population

A reduction by 10 µg/m³ in the daily maximum 8-hour moving average concentrations of ozone would lead to small decreases in the number of deaths in the general population (Figures 26 to 28): on average, 1.28 per 100 000 in total mortality, 0.75 per 100 000 in cardiac mortality and 0.39 per 100 000 in respiratory mortality. Budapest would show the highest benefits for cardiac mortality (1.6 per 100 000) while Ljubljana would show the highest benefits for respiratory mortality (0.9 per 100 000) (figures 27 to 29).

Figure 27. Reduction of O₃ daily maximum 8-hour moving average concentrations by 10 µg/m³ and impact on total mortality. Number of “premature” deaths per 100 000 per year.

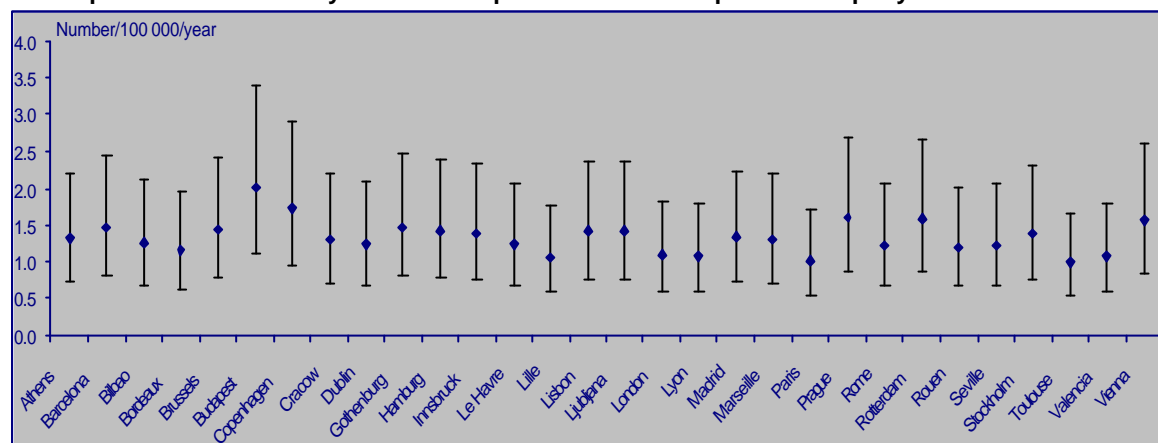


Figure 28. Reduction of O₃ daily maximum 8-hour moving average concentrations by 10 µg/m³ and impact on cardiac mortality. Number of “premature” deaths per 100 000 per year.

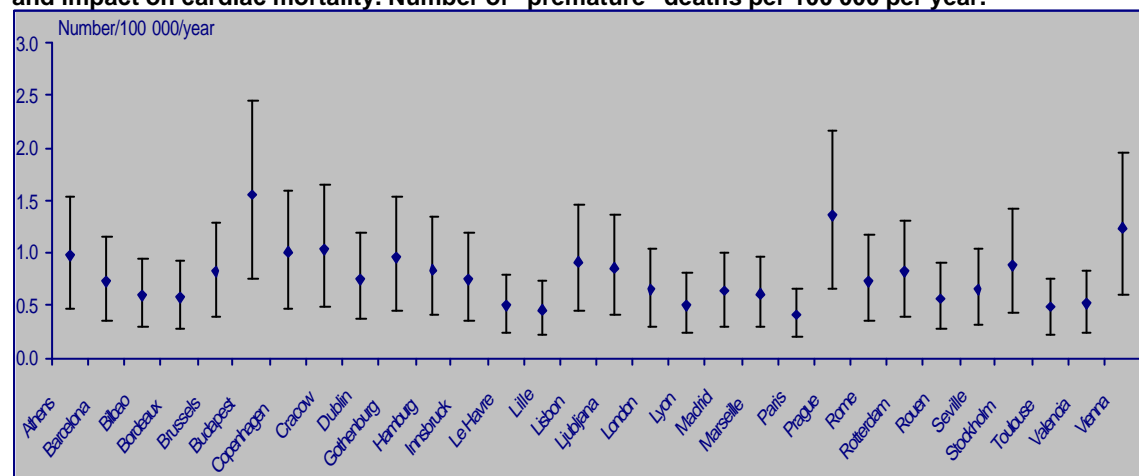
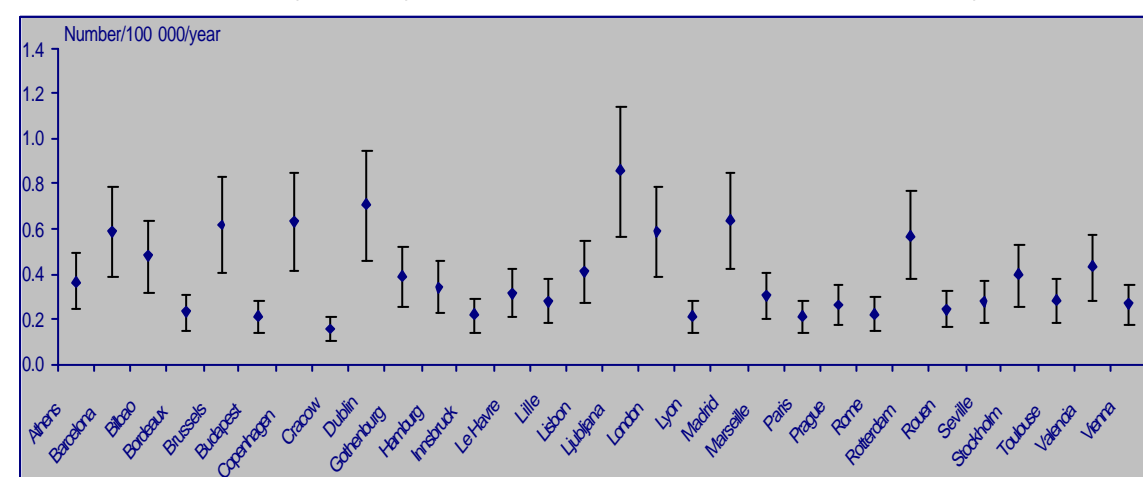


Figure 29. Reduction of O₃ daily maximum 8-hour moving average concentrations by 10 µg/m³ and impact on respiratory mortality. Number of “premature” deaths per 100 000 per year.



2.2. Daily maximum 8-hour moving average concentration and hospital respiratory admissions in people 15-64 and > 64 years.

2.2.1 Reduction of O₃ daily maximum 8-hour moving average concentrations to 120 µg/m³ in all days exceeding this value (Limit for health protection of 2002/3/EC Directive)

The benefit, in terms of attributable fractions, of reducing the daily maximum 8-hour moving average concentrations to 120 µg/m³ would be 0.02% on average for hospital respiratory admissions 15-64 years (figure 30). It would be 0.08% on average for hospital respiratory admissions >64 years (figure 31).

Figure 30. Reduction of O₃ daily maximum 8-hour moving average concentrations to 120 µg/m³ and impact on hospital respiratory admissions 15-64 years. Attributable fractions (%)

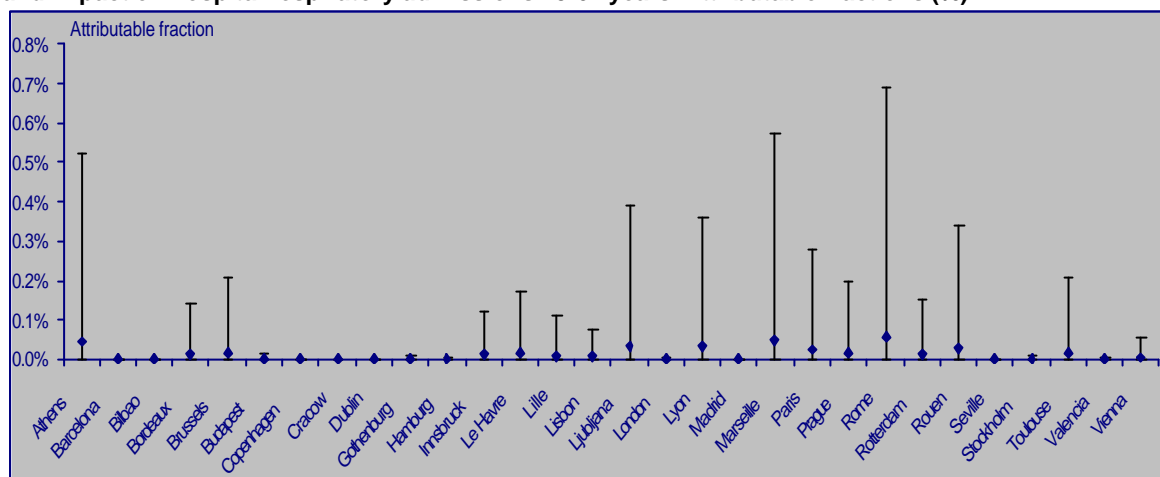
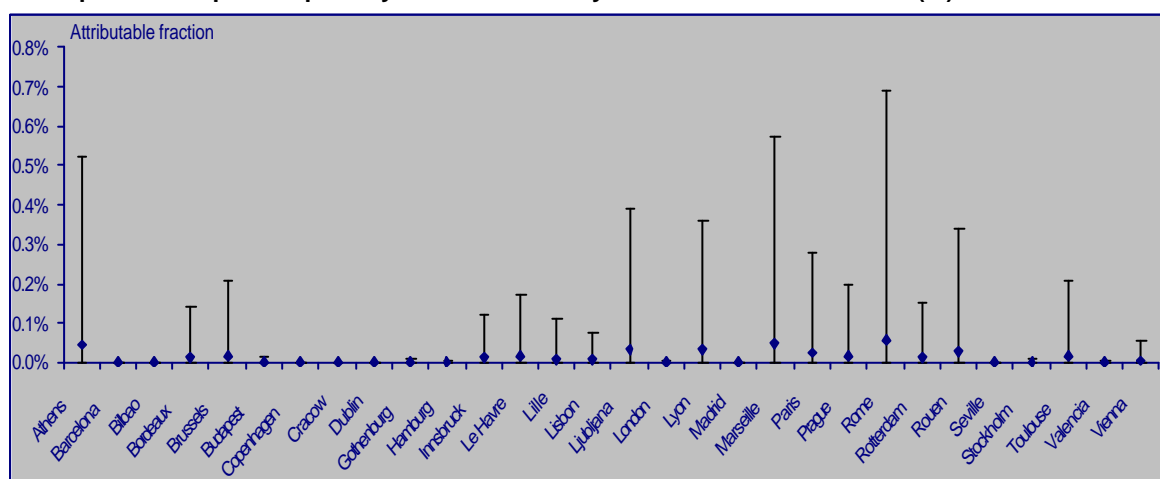


Figure 31. Reduction of O₃ daily maximum 8-hour moving average concentrations to 120 µg/m³ and impact on hospital respiratory admissions > 64 years. Attributable fractions (%)



2.2.2. Reduction by 10 µg/m³ in the daily maximum 8-hour moving average concentrations.

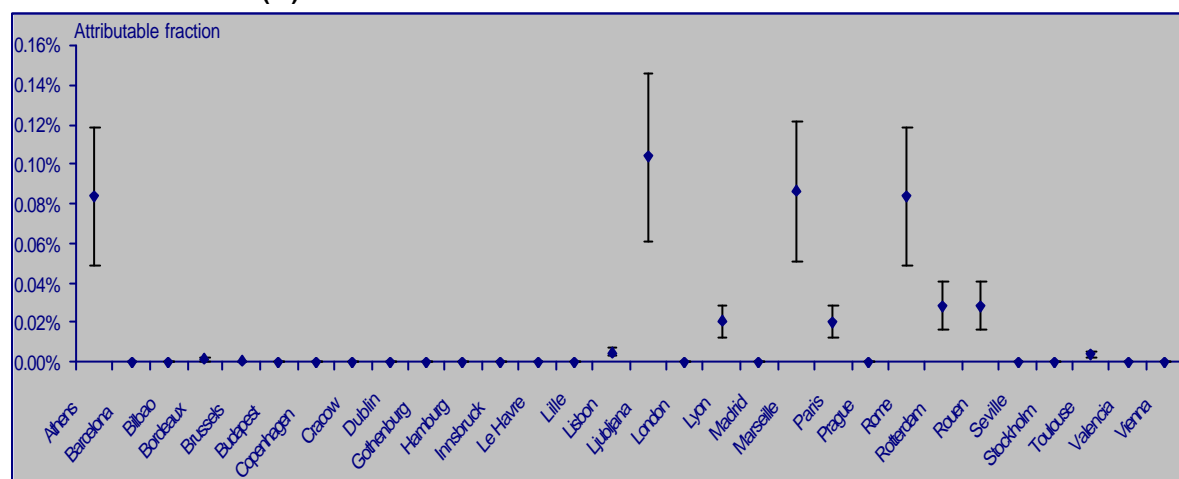
The benefit of reducing the daily maximum 8-hour moving average concentrations by 10µg/m³ would be 0.1% on average for hospital respiratory admissions 15-64 years. It would be 0.5% on average for hospital respiratory admissions >64 years.

2.3 Daily maximum 1-hour concentration (all year) and emergency room visits for asthma in people under 18 year.

2.3.1 Reduction of O₃ daily maximum 1-hour concentrations to a level of 180 µg/m³ in all days exceeding this value (information threshold of 2002/3/EC Directive)

Athens, Ljubljana, Marseille and Rome would show a small benefit (above 0.08%) if O₃ daily maximum 1-hour concentrations were reduced to a level of 180 µg/m³ in all days exceeding this value.

Figure 32. Reduction of O₃ daily maximum 1-hour all year to a level of 180 µg/m³ in all days exceeding this value³ and impact on emergency room visits for asthma in people < 18 years. Attributable fractions (%).



2.3.2 Reduction by 10 µg/m³ of the daily maximum 1-hour concentrations

The benefit of reducing the daily maximum 1-hour levels of ozone all year by 10 µg/m³ would be 1.14% in all the cities for emergency room visits for asthma in people under 18 year.

Interpretation of findings

Our HIA on outdoor air pollution in ENHIS-1 follows the approach used in the Apheis project (www.apheis.net). Hereafter, we report the general philosophy of the Apheis approach, outlining the specificities of the new HIA for ENHIS-1.

1. Objectives

Our HIA on outdoor air pollution has two main objectives:

1. Present a coherent methodology for local HIAs that the individual city-specific reports can use and refer to.
2. Establish a standard basis for comparing findings across cities; and report similarities and differences regarding both the application of methodologies and the HIA findings.

2. Causality assumption

Our HIA provides the number of health events attributable to air pollution in the target population assuming that air pollution actually *causes* the observed health effects. The scientific basis for this hypothesis has been widely discussed in the literature.

3. A conservative approach

First, we only used exposure-response functions (E-R functions) or risk estimates that are well established (see Annex 1).

Second, regarding the health outcomes described as associated with air pollution, the attributable numbers were only calculated for total and cause-specific mortality in the general population and for postneonatal mortality in children. We used postneonatal mortality even if we expected the baseline frequency rates, and consequently the HIA related findings, to be low in most of the countries participating in this HIA, compared to other regions in the world.

For the outcomes for which a population baseline frequency measure was not available or was not comparable between cities ((cough, lower respiratory symptoms, hospital respiratory admissions and emergency room visits for asthma), only attributable fractions (%) were calculated.

Regarding the air pollutants that could be considered, in ENHIS-1 it was decided to evaluate the effects of particulate pollution in children and the independent effects of ozone in children and in the general population. The HIA of particulate pollution in the general population was recently performed in Apheis-3.

We used different pollution indicators in order to provide a range of possible impacts of air pollution on health using different exposure-response functions, different cities and different age groups. But it is of crucial importance that HIA findings shown for different scenarios and different pollution indicators not be added together. This is because the pollutants are highly correlated, some of the impacts provided by one indicator may already be included in another indicator, and some of the impacts provided in one scenario are already included in another scenario.

Ozone measurements were taken in the cities not in suburban areas. In the vicinity of strong NO_x emission sources where there is abundance of NO, O₃ is “scavenged” and as a result its concentrations are often low in busy urban centres and higher in suburban and adjacent rural

areas (WHO 2003). Consequently our HIA for ozone scenarios may underestimate the health impacts.

Finally, in this HIA, an estimation of the long-term impact of outdoor air pollution on both mortality and morbidity has not been performed. It has been proven, especially for particles, that long term effects of air pollution account four times the short term effects, i.e. an increase of $10\text{ }\mu\text{g}/\text{m}^3$ on chronic exposure to ambient $\text{PM}_{2.5}$ has been associated with a 0.6%, 0.9% and 1.14% increase in total, cardiovascular and lung cancer mortality, respectively (Pope et al, 2002)

4. Threshold considerations

Recently WHO states that *“In the past, the concept of no-effect thresholds played an important role in deriving air quality guidelines. The existence of such thresholds implies no effects of increasing air pollution until a “threshold” concentration is surpassed, at which stage risk rises. Thresholds are in principle an appealing concept that has also been used in defining air quality policies, such as in justifying the numerical value of air quality limit values. Nevertheless, recent epidemiological studies investigating large populations have been unable consistently to establish such threshold levels, in particular for PM”* (WHO, 2004).

For acute effects of O_3 , studies suggest effects to be particularly evident during summer, i.e. the season of higher ranges of concentrations. However, a clear threshold of no effect has not been defined for O_3 either and if one exists it must be in the low ranges of natural background levels of O_3 . The current WHO air quality guideline for ozone of $120\text{ }\mu\text{g}/\text{m}^3$ as an eight-hour mean value does not represent a safe level of “no adverse effects”. This means that while individuals may have different thresholds regarding their sensitivity to air pollution, at the general population level there is no threshold below which air pollution has no impact on health (Schwartz et al 2000, Daniels et al 2000), at least not within the scenarios considered in our HIA.

Because the E-R functions used for PM_{10} in our HIA were linear and because there is little evidence from epidemiological studies on short-term effects of ozone to suggest a threshold at the population level, we did not assume any threshold in our calculations in ENHIS-1. And instead of choosing a single reference level, our HIA proposes a range of reference levels of air pollution used in different scenarios.

5. Attributable numbers vs attributable fractions

When possible, our HIA estimated the number of events that could be attributed to exposure to air-pollution in a specific city. We have expressed these numbers both in absolute terms directly related to the size of the population studied, and as rates per 100 000 inhabitants to allow comparisons between cities. When attributable numbers could not be calculated, a more general measure, the attributable fraction was used. The attributable fractions are only function of the exposure-response functions and of the air pollution levels in the cities.

6. Exposure assessment

Regarding exposure data, our HIA findings depend directly on the levels of particulate pollution measured. These levels vary widely as a function of the number and location of the monitoring sites, the analytical methods used, and the sites selected for our HIA. This explains the importance of using the Apehis guidelines to ensure comparability of the data.

As described in Appendix 2 on exposure assessment, the exposure measurements used in ENHIS-1 were compared to and interpreted using the Apheis Guidelines on Exposure Assessment and the PSAS-9 guidelines for site selection and selection of monitoring stations.

PM₁₀ measurements and correction factors

The PM₁₀ measurement methods were reported completely. Automatic PM₁₀ measurement methods (the β -ray absorption method and the tapered oscillating microbalance method (TEOM)) were generally used; TSP was measured by the β -ray absorption method in one city and by gravimetric method in another one.

Only four cities (Barcelona, Copenhagen, Dublin and Vienna) used the European PM₁₀ reference method (gravimetric method) for their PM measurements. Because the E-R functions used for postneonatal mortality were taken from studies that used gravimetric methods, to be consistent, we had to correct the automatic PM₁₀ measurements by a specific correction factor (local or, by default, European) in order to compensate for losses of volatile particulate matter. Cities where the information was available could use local correction factors. The final decision was taken with the advice of the local or national air pollution experts.

Ozone measurements

Ultraviolet absorption was used for ozone measurements. The O₃ levels reported were quite low. This is an important consideration because ozone in the troposphere is not emitted directly into the air, it is formed by photochemical reactions from NO_x and volatile organic compounds emission sources in the presence of heat and sunlight (EPA, 1997). As a result, O₃ is “scavenged” and its concentrations are often low in busy urban centres and higher in suburban and adjacent rural areas. On the other hand, O₃ is also subject to long-range atmospheric transport and is therefore considered as a trans-boundary problem (WHO 2003).

7. Health outcomes and baseline rates

Mortality data

The information sources for mortality data were the national, regional or local mortality registries for all the cities.

All-causes mortality remains our first choice for HIA because it is more robust, not subject to misclassification and easier to obtain. Cause-specific mortality was included to provide complementary information to enrich the mortality picture. Nevertheless, the delay to obtain validated total and cause-specific mortality data in some countries is very long and we were obliged to consider 2001 or 2002 as the most recent common year available in all the cities for our HIA.

Because ENHIS-1 focus mainly on children, our HIA looked for ERFs on mortality in children and two references fulfilled the ERFs selection criteria: Lacasana 2005 (total and respiratory postneonatal mortality) and Woddruff 1997 (postneonatal Sudden Infant Death Syndrome). Postneonatal mortality includes the period from 1 month to 1 year of life and was not available in all the cities, some provided infant mortality instead (period below 1 year) considering it as a good proxy of postneonatal mortality, although in these cities the result may lead to an overestimation of postneonatal mortality (in Madrid for eg., postneonatal

mortality is around 37 % of infant mortality and more than 60% of infant mortality occurs in the first 28 days of life).

We expected the baseline frequency rates for postneonatal mortality indicators to be low in most of the European countries participating in this HIA, compared to other regions in the world. We could not find precise and comparable statistics on postneonatal mortality (1 month-1 year) for Europe and other regions in the world but we found infant mortality rates (<1 year) and considered them useful to give an idea of the ranges between different regions worldwide (United Nations, 2003).

The infant mortality rates (deaths per 1000 live births) in Europe for 2000-2005 and 2005-2010 projections were the lowest compared to Africa, Asia, Latin America and Oceania, but higher compared to North America (Table 13).

Within Europe, Eastern Europe presents the highest mortality rates compared to Western, Northern and Southern Europe. In the 18 countries participating in this HIA, Romania presented by far the highest rates and Sweden the lowest ones (Table 14).

Table 13. Infant mortality rates in major areas of the world.

World region	Infant mortality rates (deaths per 1000 live births)	
	2000-2005	2005-2010
Africa	88.5	81.7
East Africa	96.6	89.0
Middle Africa	116	109.9
Northern Africa	48.7	41.5
Southern Africa	51.9	47.1
Western Africa	90	82.4
Asia	53.2	47.4
Eastern Asia	34.0	30.5
South Central Asia	68.2	60.9
South Eastern Asia	41.1	35.5
Western Asia	43.9	37.8
Europe	8.9	8.4
Eastern Europe	14.1	12.9
Northern Europe	5.4	5
Southern Europe	7.5	7
Western Europe	4.7	4.6
Latin America and the Caribbean	31.9	28.2
Caribbean	35.4	32.6
Central America	29.8	26.9
South America	32.5	28.4
North America	6.6	6.3
Oceania	25.9	23.2

UNITED NATIONS Population Division, Department of Economic and Social Affairs
World Population Prospects: The 2002 Revision
File 6: Infant Mortality by Major Area, Region and Country, 1950-2050 (deaths per 1,000 live births)
POP/DB/WPP/Rev.2002/1/F6
February 2003

Table 14. Infant mortality rates in the Apehis countries involved in ENHIS-1.

Apehis countries involved in ENHIS-1	Infant mortality rates (deaths per 1000 live births)	
	2000-2005	2005-2010
Romania	20.0	17.0
Poland	9.1	8.2
Hungary	8.8	8.1
Greece	6.4	6.1
Portugal	6.1	5.7
Ireland	5.8	5.4
Italy	5.4	5.2
Czech Republic	5.6	5.1
Slovenia	5.5	5.1
United Kingdom	5.4	5.0
Spain	5.1	4.9
France	5.0	4.8
Denmark	5.0	4.8
Austria	4.7	4.5
Austria	4.7	4.5
Germany	4.5	4.4
Netherlands	4.5	4.4
Germany	4.5	4.4
Belgium	4.2	4.1
Sweden	3.4	3.3

UNITED NATIONS Population Division, Department of Economic and Social Affairs
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Hospital admissions data

For a question of coherence with mortality findings, it was decided, with the experts' advice, to include RRs for hospital admissions in the health impact assessment calculations, even if they were not statistically significant. More concretely, it was decided that if there was not any new RR published by the time of making the calculations, the RRs for respiratory hospital admissions from Anderson's meta-analysis (Anderson et al., 2004) could be used although not statistically significant. One explanation for the not statistically significant findings for respiratory hospital admissions could be an insufficient statistical power of the studies.

We have selected hospital admissions for residents of each city with discharge diagnoses of respiratory diseases (ICD9: 460-519; ICD10: J00-J99) for <15 years, 15-64 years and > 64 years. Whenever possible we only used emergency admissions as being more specifically related to air pollution.

The cities obtained data from registries. Completeness in hospital admissions registries was of 95% or more in 8 cities (Bilbao, Budapest, Dublin, Gothenburg, Madrid, Stockholm, Innsbruck and Vienna); 90% or greater in 9 cities (Bordeaux, Le Havre, Lille, Lyon, Marseille, Paris, Rouen, Toulouse and Valencia). In 12 cities (Athens, Cracow, Ljubljana, London, Rome, Seville, Brussels, Copenhagen, Hamburg, Lisbon, Prague and Rotterdam) this information was not provided. Twenty eight cities run a Quality Control Programme, Athens and Cracow did not provide this information.

Only 9 cities differentiated emergency hospital admissions (Barcelona, Bilbao, Dublin, Gothenburg, London, Madrid, Seville, Stockholm and Valencia). Yet, for 18 cities, it was not

possible to distinguish between emergency and total admissions and four cities could not estimate the impact on hospital admissions.

Methodologically speaking, statistical analyses of the APHEA-2 cities showed no significant heterogeneity in the estimated RR of hospital admissions between cities that reported general hospital admissions and those that reported emergency hospital admissions only (Atkinson 2001, Le Tertre 2002). This might seem surprising initially but in fact general admissions include both planned and emergency admissions, and when controlling for season, we also control for general trends for both, leaving emergency admissions and some background noise. Nevertheless, for HIA purposes it can modify the number of attributable cases because this number depends directly on the number of observed hospital admissions.

The main problems for hospital admissions comparability remain the differences in population coverage by the registries in the cities and the difference in the availability of information in the registries (emergency vs general admissions).

Because the sources of hospital admissions data and the coverage of hospital registries differ between cities, it was decided to present only attributable fractions in this general report. It should be noted that the AFs were calculated for all the cities even if hospital admissions data was not available (this information was not required for AFs' calculations). In the city-specific reports of those cities that could gather data on hospital admissions, the attributable numbers for hospital admissions have been calculated.

Other morbidity outcomes

Emergency room visits for asthma < 18 years.

This indicator was available only in four of the 31 cities involved in this HIA.

Cough 5-17 years

All the cities except London could not accede to this information from a routine source.

Lower respiratory symptoms 5-17 years

The figures are the same for LRS although some surveys were conducted in Budapest, Cracow, Gothenburg, Rome, Stockholm, Lisbon and Prague but they were not comparable because they did not always use the same methodology.

8. Choosing the exposure-response functions

As a reminder, the criteria to select the ERFs were the following:

- ?? It was considered preferable to use summary estimates from meta-analysis
- ?? Only original studies involving great populations were deemed suitable for HIA
- ?? Only statistically significant estimates were selected for HIA (In meta-analysis this applies to the summary estimates), with the exception of hospital admissions (see above).

According to the assessment about causality made by the experts panel of 'The Review Of Health Impact of Air Pollution on Children' (WHO, 2004), the children-outcomes for which there is sufficient evidence to infer causal relationship with air pollutants are the followings:

- ?? Particulate pollution and respiratory deaths in the post-neonatal period.
- ?? Air pollution and adverse effects on lung function development: both reversible and chronically decreased lung growth, with clearer relationships for particulates and traffic related air pollution.
- ?? Air pollution and aggravation of asthma, mainly to exposure to particulates and ozone
- ?? Bronchitis and cough due to particulate exposure

For children, the most reliable estimates were the results from Lacasaña meta-analysis (2005) for all and respiratory causes, and from Woodruff's (1997) estimate for Sudden Infant Death Syndrome (SIDS). Anderson's meta-analysis (2004) provided a summary RR estimate based on three studies for respiratory hospital admissions in children 0-15 years. CARB (2004) provided a meta-estimate for emergency room visits for asthma in people under 18 years based on four studies. Summary estimates were calculated for children by Ward and Ayres (2004) for lower respiratory symptoms and cough in children 5-17 years.

For general population, Anderson's meta-analysis, APHEA2 and Bell's study gave meaningful results. Concordance between them was quite high, though estimates tend to be bigger in Europe than in the U.S.A. Estimates for all cause mortality from Anderson's meta-analysis were not statistically significant, while APHEA2 (Gryparis, 2004) gives statistically significant estimates for total, cardiovascular and respiratory mortality for the summer period. APHEA2 results on mortality (not included in Anderson's metaanalysis) were deemed to be the most adequate for HIA within ENHIS-1 project. Anderson et al (2004) provided combined estimates for respiratory admissions in 15-64 yr and >64 yr groups.

9. Transferability of E-R functions

The question of transferability of E-R functions is not a matter of concern for short-term exposure since most of the cities are some of the cities where the E-R functions were estimated.

For postneonatal mortality we used the Lacasaña meta-analysis (2005) that provided combine estimates from different regions in the world and for both acute and chronic exposure effects of PM₁₀ on postneonatal mortality for all and respiratory causes. Highly consistent results were found regardless of the different study designs used.

And we used Woodruff's (1997) estimate for SIDS for being an original research based on a very large population results but the question of transferability of estimates between the U.S. and Europe raises uncertainties, since the particulate mixtures and populations can differ between the two continents.

Also relevant for transferability are differences in methods used in the U.S. and Europe for exposure measurement, e.g., PM gravimetric vs automatic methods. We used a correction factor for PM₁₀ observed values to compensate for losses of volatile particulate matter. But, on the other hand, the application of this correction factor may be another source of uncertainty in our HIAs.

10. Statistical tools

For our HIA statistical methods, we used WHO guidelines (WHO 2001) as a starting point and also developed our own standardised statistical and HIA guidelines (Medina et al. 2001). Each centre got an excel spreadsheet and the corresponding guidelines for HIA calculations (Annex 4).

11. Answering key questions

Impact of ozone

In the framework of the CAFE programme, a WHO working group was convened to review systematically the most recent scientific evidence on the adverse effects of particulate matter, ozone and nitrogen dioxide (WHO 2003, WHO 2004). Based on the HIA findings of outdoor air pollution conducted in ENHIS-1 we report and comment some of the questions addressed by this working group and by the US EPA (1997).

1) *Why are children at high risk?*

Our HIA focused on children because they are at high risk of suffering adverse effects of air pollution owing to their potentially high susceptibility:

- *The average adult breathes 13,000 liters of air per day. Children breathe even more air per pound of body weight than adults.*
- *They have an increased ventilation playing and exercising outside.*
- *Because children's respiratory systems are still developing, the development and growth of the airways and alveoli are more vulnerable and they are more susceptible than adults to environmental threats.*
- *The immune system is still immature.*
- *For asthmatics children having an attack, the pathways of the lungs become very narrow and ozone and particulate matter can aggravate asthma, causing more asthma attacks, increased use of medication, more medical treatment and more visits to hospital emergency clinics.*

Our HIA intended to evaluate the impact of air pollution on asthma and respiratory symptoms but the information on these outcomes in the cities covered by the HIA was very weak. In terms of attributable fractions, the reduction of daily 1-hour maximum levels of ozone (all year) by $10 \mu\text{g}/\text{m}^3$ would be associated with a decrease of 1.14% of emergency room visits for asthma <18 years.

2) *Is there new scientific evidence to justify reconsideration of the current WHO Guidelines for ozone (O₃)?*

The current WHO Air quality guidelines (AQG) (WHO, 2000) for O₃ provide a guideline value of $120 \mu\text{g}/\text{m}^3$ (60 ppb), based on controlled human exposure studies, for a maximum 8-hour concentration. The AQG also provide two concentration-response tables, one for health

effects estimated from controlled human exposure studies and one from epidemiological studies. No guideline for long-term effects was provided. Since the time these guidelines were agreed, there is sufficient evidence for their reconsideration. Issues to be considered are: the averaging time(s) for the short-term guidelines and their associated levels, the concentration-response functions used in the tables, the outcomes included in the concentration-response tables, whether a long-term guideline and/or complementary guidelines (e.g. restricting personal activity) should be adopted. Recent epidemiological studies have strengthened the evidence that there are short-term O₃ effects on mortality and respiratory morbidity and provided further information on exposure-response relationships and effect modification. There is new epidemiological evidence on long-term O₃ effects and experimental evidence on lung damage and inflammatory responses. There is also new information on the relationship between fixed site ambient monitors and personal exposure, which affects the interpretation of epidemiological results.

Our HIA confirms the need for reconsideration of the WHO Guidelines for ozone. All other things being equal, in the 30 cities that could provide ozone measurements, reducing the daily 8-h maximum levels of ozone to 120 µg/m³ would prevent respectively 80, 48 and 21 premature deaths for total, cardiovascular and respiratory mortality in the general population, while an absolute reduction by 10 µg/m³ would increase considerably these numbers, respectively 567, 333 and 174 deaths that could be prevented in the 30 cities totalling more than 45 million inhabitants.

Very recently, in the July issue of Epidemiology, three original articles on ambient ozone levels and mortality relationships have been published (Bell et al, 2005; Ito et al, 2005; Levy et al, 2005). They are three meta-analyses including an extend amount of data from different countries. The studies have been performed by three different teams commissioned by EPA (The Editors. Epidemiology 2005). Using different, but not exclusive, data sets and different statistical approaches the authors found similar results of the impact of ozone on mortality: a clear effect in the summer period, but not in winter, and also an independent effect from particulates, with comparable estimates to the ER functions used in our HIA (Gryparis et al, 2004). As stated in the accompanying editorial of this July's issue of Epidemiology (Bates, 2005) this amount of evidence, point to an urgent need to develop effective actions to reduce public exposure to ozone.

Regarding hospital respiratory admissions, the attributable fractions when reducing the daily 8-h maximum levels of ozone to 120 µg/m³ would be 0.02% for patients 15-64 years and 0.08% for patients over 64 years. An absolute reduction by 10 µg/m³ would lead, all other things being equal, to a reduction of 0.10% for the patients 15-64 years. It would be 0.5% for patients over 64 years. These quite low figures for hospital admissions are the result of the non significant ERFs chosen from the meta-analysis of Anderson et al. 2004.

3) Are the current limit values sufficient to ensure no adverse health effects?

The WHO review reconfirmed that exposure to particulate matter and ozone poses a significant risk to human health at concentration levels common in Europe today. Thus, it can be concluded that further reductions in air pollution will have significant health benefits, even in regions where levels are well below current European Union (EU) limit values for PM and target values for ozone. Current air quality standards are to a large extent based on the concept of an effect threshold, below which significant health effects are not likely to occur. As stated above, no such threshold is evident for PM and ozone. Therefore, even if the limit /target value is not exceeded significant health impacts, including a substantial reduction in

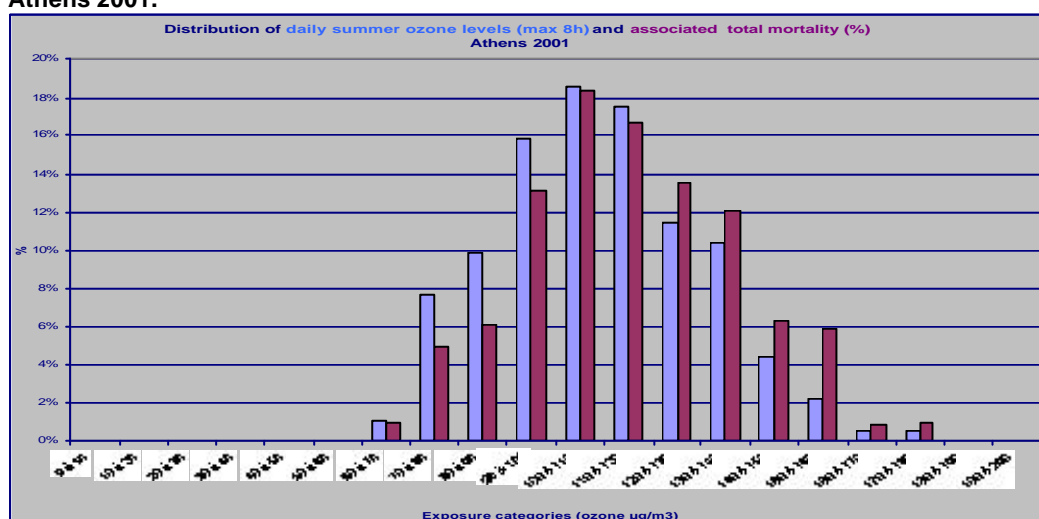
life expectancy, are to be expected. Conversely, a reduction in pollutant concentrations below the current standards should result in health benefits.

4) Should we focus on summer smog ozone peaks?

WHO working group reported that traditionally, the interest of the general public and policy-makers in ambient ozone has focussed on high peak levels, which usually occur during hot, dry periods in the summer. Recent evidence suggests, however, that ozone levels lower than those experienced during episodes of “summer smog” may have considerable effects on human health. Time-series studies have demonstrated linear or near-linear relationships between day-to-day variations in ozone levels and health end-points even at low levels of exposure. As there are usually many more days with mildly elevated concentrations than days with very high concentrations, the largest burden on public health may be expected with the former rather than the latter. Consequently, abatement policies should not only focus on the few days with high peak concentrations but should aim to reduce ozone levels throughout the summer season.

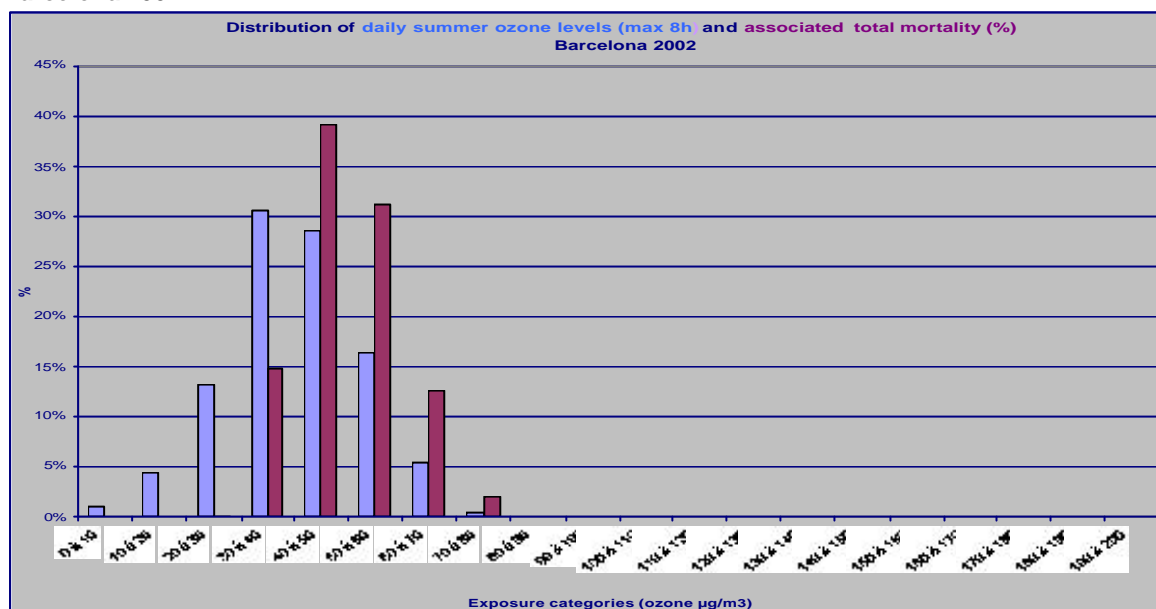
In our HIA, we can illustrate questions 4) and 5) with three examples. In the city with the highest daily 8-h max ozone mean levels, Athens (109 $\mu\text{g}/\text{m}^3$), 30% of the days in the summer period (1 April-1 September) exceeded levels above 120 $\mu\text{g}/\text{m}^3$ and these levels were associated with 40% of the total impact on premature mortality, 60% of which is due to levels that comply with the air quality guidelines (figure 33).

Figure 33. Distribution of daily summer ozone levels (max 8h) and associated total mortality (%) in Athens 2001.



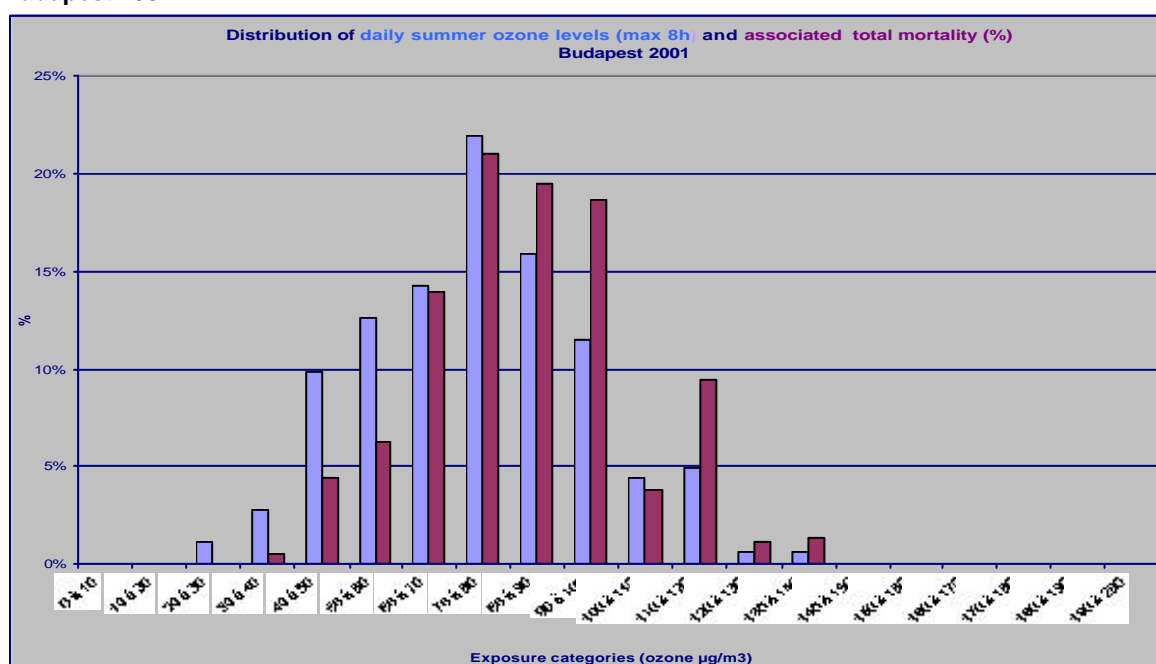
On the other hand, in Barcelona, the city showing the lowest daily 8-h max ozone mean levels ($40.7 \mu\text{g}/\text{m}^3$), no exceedances above $120 \mu\text{g}/\text{m}^3$ were observed in the summer period (1 April-1 September) and 100% of the total impact on premature mortality was observed for levels between 30 and $80 \mu\text{g}/\text{m}^3$ that comply with the air quality guidelines (figure 34).

Figure 34. Distribution of daily summer ozone levels (max 8h) and associated total mortality (%) in Barcelona 2002.



Finally, in a city with daily 8-h max ozone mean levels of $74 \mu\text{g}/\text{m}^3$, Budapest, 2% of the days in the summer period (1 April-1 September) exceeded levels above $120 \mu\text{g}/\text{m}^3$ and these levels were associated with only 2% of the total impact on premature mortality, 98% of which is related to levels that comply with the air quality guidelines (figure 35).

Figure 35. Distribution of daily summer ozone levels (max 8h) and associated total mortality (%) in Budapest 2001.



Hence, in these examples, we can see that, although behaving differently among the cities, the impact of air pollution episodes is not the main issue in terms of public health.

5) To what extent is mortality being accelerated by long- and short-term exposure to O₃ (harvesting)?

Long-term O₃ effects have been studied in two cohort studies. There is little evidence of an independent long-term O₃ effect on mortality so that no major loss of years of life is expected. The issue of harvesting, i.e. the advancement of mortality by only relatively few days, has not been addressed in short-term exposure studies of O₃.

Our HIA could not evaluate the long-term impacts of ozone exposure.

Impact of PM₁₀

Regarding PM₁₀, Apheis-3 (Medina et al., 2005) answered the following questions. We complete them based on our findings for ENHIS-1.

1) What's more important: Long-term or short-term? Number of deaths, attributable fractions or gain in life expectancy, others?

Long-term vs. short-term

When interpreting the findings on annual mortality, we saw that the main effects of air pollution are associated with long-term exposure. Most of the acute effects on mortality are included in effects of long-term exposure and represent around 15% of these chronic effects, when judged in terms of the number of attributable cases. But not all short-term health impacts are included in the long-term impacts (Medina et al 2004, Kunzli et al. 2001). It was interesting to note that the cumulative short-term impact over up to 40 days was more than twice that found using only 2 days of exposure follow-up (Zanobetti et al. 2002), showing that air pollution does not simply displace mortality by a few days. Consequently, omitting E-R functions from time series would lead to under-estimating the short-term impact on mortality (Table 15).

Table 15. Apheis-3 findings for PM₁₀.

Air pollution indicator	Health indicator	HIA scenario	Potential reduction in the number of deaths					
			Very short-term		Cumulative short-term		Long-term	
			Number of deaths	Number of deaths/ 100 000/ year	Number of deaths	Number of deaths/ 100 000/ year	Number of deaths	Number of deaths/ 100 000/ year
PM ₁₀	All causes mortality*	Reduction to 50 ** µg/m ³ /40** µg/m ³	559	2	1150	3	8550	24
		Reduction to 20 µg/m ³	2580	7	5240	15	21385	60
		Reduction by 5 µg/m ³	868	2	1739	5	6143	17
	Cardiovascular mortality	Reduction to 50 µg/m ³	412	1	877	2		
		Reduction to 20 µg/m ³	1741	5	3458	10		
		Reduction by 5 µg/m ³	527	1	897	2		
	Respiratory mortality	Reduction to 50 µg/m ³	87	0.2	288	1		
		Reduction to 20 µg/m ³	429	1	1348	4		
		Reduction by 5 µg/m ³	162	0.5	489	1		

Our HIA in ENHIS-1 focussed on short-term effects of PM₁₀ and ozone except for postneonatal mortality, where mortality occurring within the first year of life is considered. This is in line with the definition of postneonatal mortality. The epidemiological studies that establish the association between air pollution and postneonatal mortality integrate, by design, the (not further specified) combination of short-term and potential sub-acute cumulated effects.

Attributable Fractions/Number of deaths/Gain in life expectancy/Other indicators

Attributable cases are often interpreted as the preventable fraction, meaning those that would have been prevented had exposure been removed. However, caution should be used with such an interpretation. First, the benefit of removing a particular exposure can only rarely be estimated. The benefit may be achieved much later than predicted, or not to the full extent predicted. In our case, lower air pollution levels would take years to be fully achieved. Second, the attributable risk estimation does not take competing risks into account. Removing one risk factor, e.g., air pollution, will increase the relative importance and contribution of other risks and causes of morbidity and mortality. Accordingly, for multicausal diseases it is well known that the sum of attributable cases across several risk factors does not add up to 100% but may be larger. Nevertheless, recent intervention studies (Heinrich et al. 2002, Hedley et al. 2002, Clancy et al. 2002, Friedman et al. 2001) do indicate the reduction in mortality and morbidity after decreases in air pollution.

For the time being, expressing mortality findings in terms of “premature” deaths per year is still an easy-to-understand way of communicating health/mortality impacts. It gives a picture at one point in time. Expressing mortality findings in terms of expected gain in life expectancy provides a more dynamic picture.

Our HIA expressed the findings in terms of “premature” or anticipated deaths per year but because it was recently done in Apheis-3, we did not calculate the expected gain in life expectancy. For those outcomes for which baseline frequency measures were not available or were not comparable, a more general measure of the impact was used, the attributable fraction that expresses the findings in percentages and do not allow providing the actual numbers in each city.

In future HIAs, besides the attributable numbers, fractions and gain in life expectancy, we should consider the possibility of calculating also disability adjusted life years used by WHO to assess the global burden of diseases associated with different causes (Murray et al. 2002, de Hollander et al 1999). This metric is a variant of the quality adjusted life years (QALYs) that measure morbidity as a reduction of quality of life over a period of life. A new metric suggested by Hubbell (2005) at the USEPA, the “fair QALYs”, aggregates life years saved and improvements in quality of life.

2) Implications for policy making: particulate pollution indicators and limit values

PM vs. BS

There is substantial toxicological and epidemiological evidence of the effects of PM on mortality and morbidity. And it has been highlighted that primary, combustion-derived particles have the highest toxicity (WHO 2004).

PM₁₀ levels are regulated by the European Commission. Unfortunately, black smoke regulation has ceased, and no European Directive is planned for BS by 2005 or by 2010. Nevertheless, this air-pollution indicator, which has been measured for many years in most European cities, represents small black particles (less than 4 µm in size) with measurable health effects and may be considered as a good proxy for traffic-related air pollution closely related to diesel engine exhaust in urban areas (WHO 2003).

Our HIA focussed on PM₁₀ in children, and we consider it as an indicator of the particulate exposure. We could study the impact of air pollution on postneonatal mortality and the findings were not negligible. All other things being equal, reducing the annual mean value of PM₁₀ by 5 µg/m³ in all the cities covered by this HIA, totalling almost 46 million inhabitants, would decrease the number of total postneonatal deaths by 23, for respiratory postneonatal deaths the reduction would be of 5 deaths and for Sudden Infant Death Syndrome it would be of 7. Because the scientific evidence is not strong enough, our HIA did not evaluate the effects on birth weight, pre-term births and intrauterine growth retardation.

Given the evidence currently available, policymakers should consider the air-pollution mixture as a whole for setting standards, and not favour some air-pollutant indicators over others.

PM₁₀: Meeting 2005 and 2010 European limit values

In our HIA, the European annual limit value of 40 µg/m³ for PM₁₀ is still exceeded in a few cities in southern and Eastern Europe, although 26 of the 31 cities that measured PM₁₀ already meet the annual cut-off of 40 µg/m³. However, excepting the two Swedish cities, Hamburg and London, the 2010 annual limit value of 20 µg/m³ for PM₁₀ is exceeded in most of the cities.

Conclusion

Following Apehis-3 guidelines, we established a good basis for comparing methods and findings between 31 cities in Europe in ENHIS-1.

To provide a conservative overall picture of the impact of urban air pollution on public health in Europe, like its predecessors Apehis-2 and Apehis-3, the HIA in ENHIS-1 used a limited number of air pollutants and health outcomes for its HIAs.

Our HIA in ENHIS-1 with special emphasis on children, added more evidence to the findings from Apehis-2 and 3 and other HIAs performed in Europe that air pollution continues to pose a significant threat to public health in urban areas in Europe.

The main obstacle to be creating a more complete picture of the health impacts of outdoor air pollution in Europe remains the availability of morbidity data sources. Our study stresses that local, national and European public health authorities should advocate:

- Reducing the time needed to obtain validated total and cause-specific mortality data in some countries
- Producing more-uniform hospital-admissions statistics in Europe
- Accessibility, preferably on a routine basis, to other important morbidity indicators, such as asthma attacks and respiratory symptoms, using standardised methodology.

Our HIA findings continue to demonstrate that incentives to reduce PM₁₀ levels in the short and medium terms are needed to help reduce air-pollution levels further. A coordinated initiative by European legislators and national and local policy-makers could help achieve this goal.

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