

HEALTH IMPACT ASSESSMENT OF AIR POLLUTION

ENHIS-1 PROJECT: WP5 HEALTH IMPACT ASSESSMENT

LOCAL CITY REPORT

Stockholm

Summary of main findings for Stockholm

In 2002 the PM_{10} annual mean (SD) as an urban background level was 18 (12) $\mu\text{g}/\text{m}^3$, which is below the 1999/30/EC Directive limit value for 2010 (20 $\mu\text{g}/\text{m}^3$), and below that established limit value for 2005 (40 $\mu\text{g}/\text{m}^3$). For the summer period of the same year, the mean (SD), P5 (5th percentile) and P95 of the maximum daily 8-hour moving average concentration of ozone (O_3) were 86 (17), 56 and 113 $\mu\text{g}/\text{m}^3$.

Regarding children, infant mortality in Europe is quite low and consequently, the expected attributable number of deaths related to air pollution is also very low. All other things being equal, the reduction of the annual average levels of PM_{10} by 5 $\mu\text{g}/\text{m}^3$ would statistically prevent 0.28 postneonatal deaths per year. Reducing PM_{10} daily mean values by 5 $\mu\text{g}/\text{m}^3$ would also prevent 9 hospital respiratory admissions per year within the age group 0-15 years.

As far as short-term effects of O_3 in summer are concerned, all other things being equal, each reduction by 10 $\mu\text{g}/\text{m}^3$ of the daily maximum 8-hour moving average concentrations would prevent 16.3 preterm deaths per year in the general population in the study area, 10.5 from cardiovascular diseases, and 4.7 from respiratory causes. In terms of hospital admissions, this reduction would represent 1.3 respiratory admissions in the adult population below and 11.7 in the population above 64 years of age.

Summary of HIA of outdoor air pollution in Stockholm in ENHIS-1

| Health outcome | Population | Pollutant | Period | Mean type | RR (for 10 µg.m ³ increase) | References | Number of attributable cases by scenario ¹ | | |
|---|--------------------|--|---------------------|---------------------|---|---------------------|---|--|---------------|
| Mortality | | | | | | | Ozone: Reduction by 10 µg.m ³ | PM10: Reduction by 5 µg/m ³ | |
| Total mortality excluding external causes (ICD9 < 800 - ICD10 A00-R99) | All ages | O ₃ 8h max | Summer ² | Daily | 1.0031 (1.0017-1.0052) | Gryparis et al 2004 | 16.29 | | |
| Cardiovascular mortality (ICD9 390-459 - ICD10 I00-I99) | | | | | 1.0046 (1.0022-0.0073) | | 10.53 | | |
| Respiratory mortality (ICD9 460-519 - ICD10 J00-J99) | | | | | 1.0113 (1.0074-1.0151) | | 4.69 | | |
| Total postneonatal mortality | 1 month- 1 year | Corrected PM ₁₀ ³ | Year | Annual | 1.048 (1.022-1.075) | Lacasaña et al 2005 | | 0.28 | |
| Postneonatal respiratory mortality (ICD9 460-519 - ICD10 J00-J99) | | | | | 1.216 (1.102-1.342) | | | 0.10 | |
| Postneonatal Sudden Infant Death Syndrom Mortality (ICD9 798.0 - ICD10 R95) | | | | | 1.12 (1.07-1.17) | Woodruff 1997 | | 0.00 | |
| 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) | CARB 2004 | 3.44 | | |
| Cough | < 18 years | Measured PM ₁₀ | | | 1.0407 (1.0202-1.0511) | Ward and Ayres 2004 | | | not available |
| Lower respiratory symptoms LRS | < 18 years | Measured PM ₁₀ | | | 1.0407 (1.0202 -1.617) | Ward and Ayres 2004 | | | not available |
| Hospital respiratory admissions (ICD9 460-519 - ICD10 J00-J99) | < 15 years | Measured PM ₁₀ | | | 1.010 (0.998-1.021) | Anderson et al 2004 | | | 8.67 |
| Hospital respiratory admissions (ICD9 460-519 - ICD10 J00-J99) | 15 - 64 years | O ₃ 8h max | Summer | 1.001 (0.991-1.012) | 1.28 | | | | |
| Hospital respiratory admissions (ICD9 460-519 - ICD10 J00-J99) | > 64 years | | | 1.005 (0.998-1.012) | 11.69 | | | | |

¹ For ozone: absolute reduction by 10 µg/m³. For PM₁₀ absolute reduction by 5 µg/m³.

² Definition of summer period : 01 April – 30 September

³ 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.

Introduction

The study area, metropolitan Stockholm, includes 41 parishes and has the population of 1.18 million inhabitants (2002). 17% of the population are between 0-14 years of age while approximately 15% is above 64 years of age.

Stockholm has an sub-continental climate, and is situated next to the Baltic sea, 13% of the total area being water lakes or ocean. The mean temperature during the winter half year is approximately 2°C, and the mean temperature during the summer is 13°C.

The main cause of death in the adult population in Sweden is cardiovascular disease, with approximately 45% of the men and 44% of the woman dying with this cause of death. Since 1990 the trend has been positive with a decreasing number deaths in cardio-vascular disease, which has resulted in an increase in expected life length from 1990 until 2004 with 3 years among men and 2 years among woman.

From an international point of view, children's health is very good in Sweden. Vaccination programmes have been successful, resulting in the disappearance of a number of serious infectious diseases such as tetanus, diphtheria, polio, measles, German measles and whooping cough. Since many years Sweden is one of the countries with the lowest infant mortality in the world. Health in Swedish children has also been improved by almost complete elimination of conditions such as malnutrition and vitamin deficiency.

However, the number of children and adults with allergies has more than doubled in Sweden over the last few decades, as in other parts of Europe. More than one in four children today suffer from an allergic disease with symptoms. Most children with allergic diseases have their onset in the first four-five years of life.

The main sources of air pollution in the city is vehicle traffic, while heating and industry contributes more moderately. Long distance transport of air pollutants from other parts of Europe is also important, especially for the urban background levels of fine particles and ozone.

Previous corresponding health impact assessments (HIA) has been carried out for this area within the framework of the APHEIS year 2 and 3. Similar calculations has also been made for more specific traffic projects, and national studies (Forsberg et al, 2005).

This report presents the descriptive data on air pollution and health outcomes together with the results from the HIA calculations made within the work package WP5 of ENHIS-1 project (www.enhis.com).

Sources of air pollution

In the beginning of the last century heating was a considerably large source of pollution due to many small wood and coal heated furnaces. The 1960's mean levels of sulphur dioxide in the central parts of Stockholm exceeded $100 \mu\text{g}/\text{m}^3$, and during the winter months the 24-hour concentration could raise above $200 \mu\text{g}/\text{m}^3$. The largest decrease in SO_2 -concentration was seen between 1965 and 1972, in some parts of the city the levels decreased by approximately $100 \mu\text{g}/\text{m}^3$. This dramatical decrease was a result of the introduction of district heating, and through regulating the amount of sulphur in heavy oils used for heating.

Meanwhile emissions from heating related combustion sources decreased, traffic was becoming the main source of air pollution in the city of Stockholm. The urban background levels of NO_2 were rather stable during the period 1982-1989, despite the increasing traffic, and the yearly urban background averages were approximately $30 \mu\text{g}/\text{m}^3$. In 1989, the Swedish government decided to implement restrictions concerning exhaust fume emissions on newly built cars. In practice, this meant restrictions against new cars without catalytic equipped engines. This had positive effects on the NO_x emissions, and the urban background levels fell.

These actions have had positive effects on the air pollution levels in Stockholm, not only on the levels of NO_x and SO_2 but also on the concentrations of black smoke, CO and carbon hydrates.

Today vehicle traffic is still the major contributor to local emissions of particle mass and nitrogen oxides. Mechanically generated particles due to wear of roads and possibly also tyres is important especially at street level. Local traffic exhaust emissions have very small impact on PM_{10} levels. The regional background is mainly determined by long distance transport of particles. Long distance transport is also the major contributor to ozone in Stockholm.

In residential areas emission due to wood burning can be important. Even though, there are very few wood boilers in Stockholm, there are several tens of thousands wood stoves (stoves, fireplaces, small open furnaces). Long-range transported particles contribute with a large part of the total mass concentration in the urban background, especially for the finer $\text{PM}_{2.5}$ fraction.

In 1996 an environmental zone was created around the city centre. This means that trucks and busses that do not fulfil predetermined demands on age (max 8 years) and emissions, not are allowed to travel through the city centre. The result has been lowered emissions of particles and nitrogen dioxide, but also decreasing levels of noise. Nevertheless, measurements has shown exceedances of the EC limit values for NO_2 on street stations in the city center, why the county recently has worked out a program with suggestions how to take measures to improve the current situation.

Exposure data

The monitoring network in Stockholm includes both roof level and street level measurements and for some pollutants different methods are being used. A complete description of current instrumentation and details on the measurement sites is given in Swedish at www.slb.nu

In this health impact assessment as in APHEIS 2-3, PM₁₀ data for Stockholm comes from the original roof top station *Rosenlundsgatan*, while ozone is measured at a nearby roof top station on *Södermalm*. Measurements of PM₁₀ are performed at four sites in Stockholm, one rooftop site and three street stations, while ozone is measured at one roof top site.

Background data are also available from the measurements performed at Aspöret by ITM at Stockholm University. It has been estimated that on average close to 5 µg/m³ PM₁₀ at roof level in the city centre is of local origin, mainly from traffic.

The PM₁₀ measurements (TEOM) are corrected by a locally determined factor, 1.2.

How indicators used in the HIA have been calculated:

PM₁₀: daily exposure indicator has been calculated as the mean of the daily concentrations of the stations.

Ozone: the daily maximum 1-hour indicator has been calculated as the arithmetic mean of the 1-hour maximum at the station for the whole year. The daily maximum 8-hour moving average of each day have been calculated as the arithmetic mean of the maximum 8-hour moving averages of the stations for the summer period (1st April to 30th September).

The annual mean level (SD) of PM₁₀ in Stockholm was 18 (12) µg/m³, and P5 and P95 of the daily mean values were, respectively, 7 µg/m³ and 41 µg/m³. The mean (SD), P5 and P95 of the daily maximum 8-hour moving average concentrations of O₃ were, respectively, 86, 56 and 113 µg/m³, and those of the daily maximum 1-hour concentrations 76, 39 and 114 µg/m³ (Table 1 and figures 1-3).

Table 1. Descriptive statistics for ozone and PM₁₀ levels in Stockholm 2002.

| | O3 8h – summer | O3 1h max – year | PM10 – year |
|--------------------|-------------------|---------------------|----------------|
| Number | 183 | 363 | 363 |
| Minimum | 47 | 13 | 5 |
| Percentile 5 | 56 | 39 | 7 |
| Percentile 25 | 74 | 61 | 10 |
| Median | 85 | 74 | 14 |
| Percentile 75 | 98 | 90 | 23 |
| Percentile 95 | 113 | 114 | 41 |
| Percentile 98 | 118 | 121 | 56 |
| Maximum | 126 | 145 | 90 |
| Daily mean | 86 | 76 | 18 |
| standard deviation | 17 | 22 | 12 |

Figure 1. Distribution of daily O3 8h max in Stockholm. Summer 2002

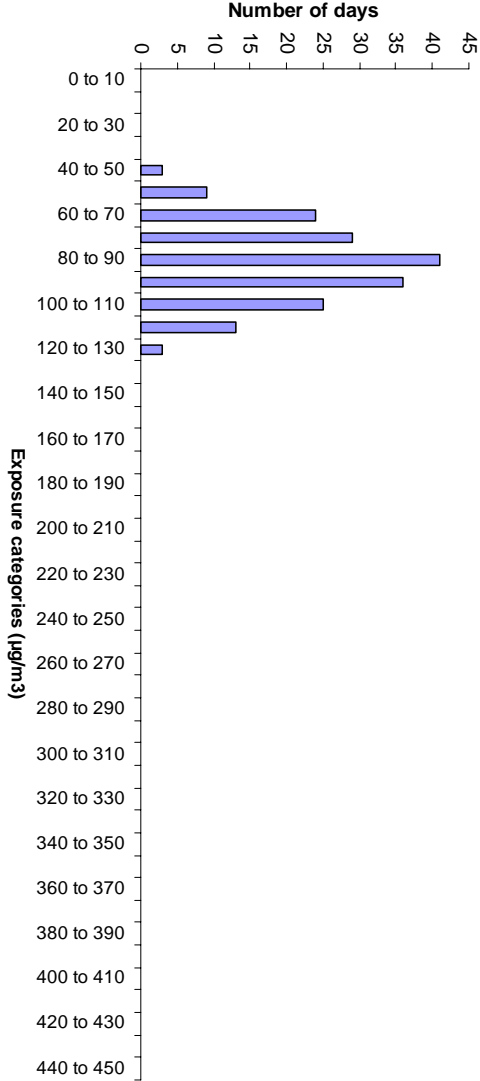


Figure 2. Distribution of O3 1h max in Stockholm. Year 2002

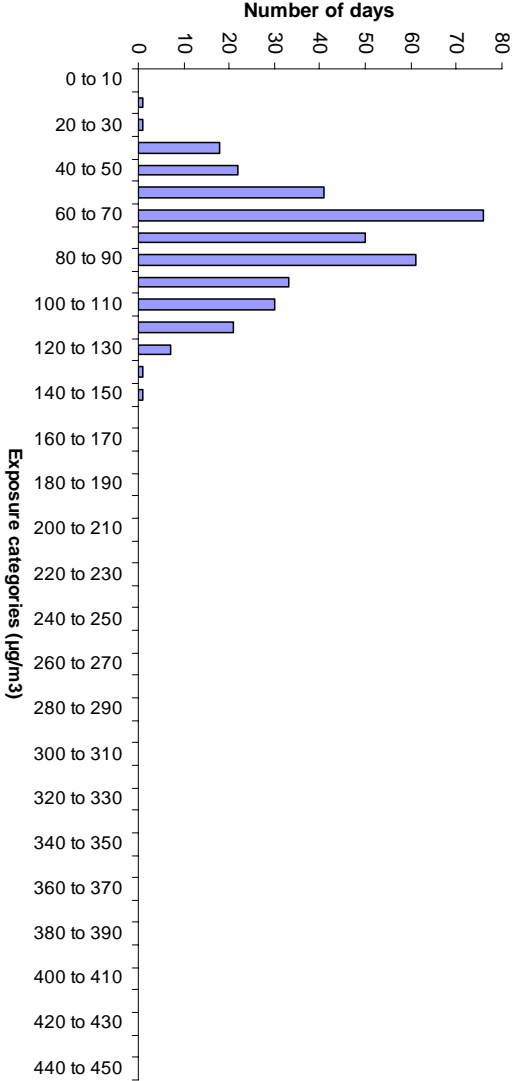
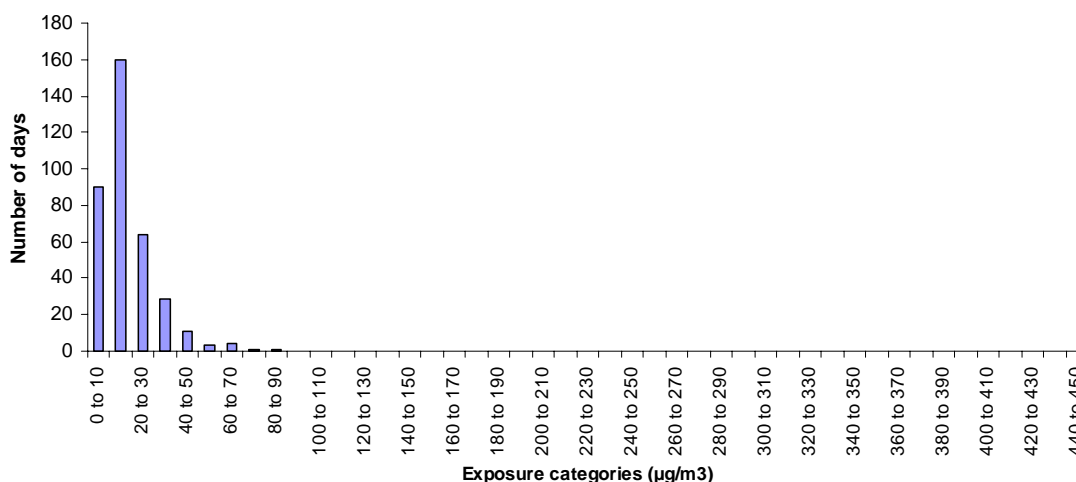


Figure 3. Distribution of daily levels of PM 10 in Stockholm. Year 2002



The urban background levels may be elevated due to inversions, emission of road dust and intranportation of polluted air masses. Road dust has an effect on mainly PM₁₀ and especially the concentration at street level. It is most important in spring, while elevated concentrations of ultrafine particles due to vehicle exhaust emissions are most common in winter. The particle concentration (measured as PM₁₀) has been rather constant during the 1990's. During the 60's, 70's and 80's there has been a reduction in soot particles measured as black smoke. The urban background levels of PM₁₀ did not exceed the limits values during 2002. However, the situation at street level is worse with high levels coarse particles mainly due to road dust.

During 2002 the daily max 8h O₃ levels in central Stockholm exceeded the 2010 limit value for ozone three times. There has been a slightly increasing trend of ozone both in the central parts of Stockholm and in the outskirts of the city. The trend in the city center is most likely a combination of decreasing levels of NO₂ and increasing levels of ozone in the regional background.

Health data

The Centre for Epidemiology (EpC) is a part of the Swedish National Board of Health and Welfare (<http://www.sos.se/epc/epceng.htm>), and is responsible for national health registers used in this HIA (ENHIS-1 WP5), namely the Cause of Death Registry and the Hospital Discharge Registry.

Statistics Sweden is however entrusted by the EpC with the actual compilation of the mortality statistics. Only 0.8 % of cases are lacking cause of death. Information to the Hospital Discharge Registry is delivered once a year to EpC from each of the 26 county councils in Sweden. The completeness of more than 99% and a low frequency of missing cause (1%) give the register a high quality. Data on hospital emergency visits has only recently been included in the register, and is likely underreported. Frequency data on cough and respiratory symptoms in children is not available in the register.

As obvious from the table the death rate is low, with a total postneonatal mortality of 12 deaths during 2002 which gives an annual rate of 77 per 100 000. The daily number of deaths as total, cardio-vascular and respiratory mortality was 29.4, 13.0 and 2.5.

Table 2. Descriptive statistics for health outcomes in Stockholm 2002.

| Health outcome | ICD9 | ICD10 | Annual deaths | Annual rate (per 100 000) | Daily mean (SD) | Daily rate (per 100 000) | Annual incidence rate (per 100 000) |
|---|---------|---------|---------------|---------------------------|-----------------|--------------------------|-------------------------------------|
| POSTNEONATAL MORTALITY | | | | | | | |
| Total | | | 12 | 77 | | | |
| Respiratory ICD9 460-519 ICD10 J00-J99 | 460-519 | J00-J99 | 1 | 6.4 | | | |
| Sudden infant death syndrome ICD9 798.0 – ICD10 R95 | 798.0 | R95 | 0 | 0 | | | |
| GENERAL POPULATION MORTALITY | | | | | | | |
| Total mortality all causes ICD9 <800 ICD10 A00-R99 | <800 | A00-R99 | | | 29.4 (6.4) | 2.5 | |
| Cardiovascular mortality ICD9 390-459 ICD10 I00-I99 | 390-459 | I00-I99 | | | 13.0 (4) | 1.1 | |
| Respiratory mortality ICD9 460-519 ICD10 J00-J99 | 460-519 | J00-J99 | | | 2.5 (2.0) | 0.2 | |
| MORBIDITY | | | | | | | |
| Cough | | | | | not available | | |
| Lower respiratory symptoms LRS | | | | | not available | | |
| Emergency room visits for asthma - Age < 18 years ICD9 493, ICD10 J45 J46 | 493 | J45-J46 | | | 0.9 (na) | 0.4 | |
| Hospital respiratory admissions - Age < 15 years ICD9 460-519 ICD10 J00-J99 | 460-519 | J00-J99 | | | | | 874.6 |
| Hospital respiratory admissions - Age 15 -64 years | 460-519 | J00-J99 | | | | | 318.6 |
| Hospital respiratory admissions - Age > 64 years | 460-519 | J00-J99 | | | | | 2737.1 |

Health Impact Assessment

Methodology

Health impact of air pollution (AP) has been calculated as the annual number of health events attributable to AP in the target population. A causal relationship between AP and the effects is assumed, and therefore HIA can only be performed for those outcomes with sufficient evidence of causality. Once the effects with sufficient evidence of causal relationship with AP have been determined, the next step is to find the best exposure-response functions (ERFs) for each of the selected outcomes. Table 3 shows the result of a systematic review on these issues carried out by the Bilbao Apheis team¹ for WP5 of ENHIS-1. This table summarizes the health outcomes and ERFs deemed suitable for

¹ Cambra K, Alonso E, Cirarda FB, Martínez-Rueda T. Bilbao APHEIS group. Selection of outcomes and exposure response functions for health impact assessment of particles and ozone. Review of the evidence. ENHIS project. WORK PACKAGE 5. Bilbao, February 2005. Http:

HIA according to the criteria established by WP5 with the advice of the air pollution experts of WP5².

Table 3. Health outcomes and Exposure-response functions (ERFs) selected for health impact assessment.

| | OUTCOME | POLLUTANT | ERFs | ORIGINAL SOURCE |
|----------------------------------|--|---------------------------------|---|-------------------------------|
| CHILDREN – PARTICLES | | | | |
| | Total postneonatal mortality (1 month-1 year) | PM ₁₀ Annual Mean | RR=1.048 (1.022-1.075) ↑10µg/m ³ | Lacasaña et al 2005 |
| | Postneonatal respiratory mortality ICD9 460-519 ICD10 J00-J99 | PM ₁₀ Annual Mean | RR=1.216 (1.102-1.342) ↑10µg/m ³ | Lacasaña et al 2005 |
| | Postneonatal Sudden Infant Death Syndrome (SIDS) mortality (normal birth weight ≥2500g) ICD9 798.0 –ICD10 R95 | PM ₁₀ Annual Mean | Adjusted Odds Ratio AOR=1.12 (1.07-1.17) ↑10µg/m ³ | Woodruff et al. 1997 |
| | Cough | PM ₁₀ Daily Mean | OR=1.041 (1.020-1.062) ↑10µg/m ³ | Ward & Ayres 2004 |
| | Lower respiratory symptoms LRS | PM ₁₀ Daily Mean | OR=1.041 (1.020-1.051) ↑10µg/m ³ | Ward & Ayres 2004 |
| CHILDREN – OZONE | | | | |
| | Emergency room visits for asthma <18 Y ICD9 493, ICD10 J45 J46 | Ozone Maximum 1 h | RR=1.0116 (1.0067-1.0165) ↑10µg/m ³ | CARB 2004 |
| ADULTS/GENERAL POPULATION | | | | |
| | Total mortality all causes ICD9 <800 ICD10 A00-R99 | Ozone Maximum 8 h Summer | RR= 1.0031 (1.0017-1.0052) ↑10µg/m ³ | Gryparis et al 2004 (APHEA 2) |
| | Respiratory mortality ICD9 460-519 ICD10 J00-J99 | Ozone Maximum 8 h Summer | RR= 1.0113 (1.0074-1.0151) ↑10µg/m ³ | Gryparis et al 2004 (APHEA 2) |
| | Cardiovascular mortality ICD9 390-459 ICD10 I00-I99 | Ozone Maximum 8 h Summer | RR= 1.0046 (1.0022-1.0073) ↑10µg/m ³ | Gryparis et al 2004 (APHEA 2) |

To be coherent with mortality findings, it was decided, with the experts' advice, to include RRs of 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 could be used, although they were not statistically significant (see Table 2). The rationale for that is that if there is sufficient evidence to accept a causal relationship between air pollution and respiratory mortality both in children-PM and adults-O₃ we should easily accept that there will also be an impact on hospital admissions.

² Ferran Ballester: Valencian School of Health Studies, Valencia, Spain; Sylvie Cassadou: National Institute of Public Health Surveillance, InVS, Toulouse, France; Fintan Hurley: Institute of Occupational Medicine, Edinburgh, Scotland, UK; Nino Künzli: University of Southern California, Division of Occupational and Environmental Health, Los Angeles, CA, USA; Odile Meckel: Institute of Public Health NRW (LOEGD), Bielfeld, Germany; Hans-Guido Mücke: WHO Collaborating Center (Air)-Federal Environmental Agency, Berlin, Germany; Nikolaos Stilianakis: Institute for Environment and Sustainability, European Commission – JRC, Ispra, Italy.

Table 4. Complementary Exposure-response functions (ERFs) for health impact assesment on respiratory hospital admissions for children (particles) and adults (ozone).

| | OUTCOME | POLLUTANT | RR | SOURCE |
|----------------------------------|---|--------------------------------------|--|----------------------------|
| CHILDREN – PARTICLES | | | | |
| | <i>Respiratory hospital admissions 0-14 Y</i> ICD9 460-519 ICD10 J00-J99 | <i>PM₁₀</i> Daily Mean | <i>RR= 1.010 (0.998-1.021)</i> ↑10µg/m ³ | <i>Anderson 2004</i> |
| ADULTS/GENERAL POPULATION | | | | |
| | <i>Hospital respiratory admissions 15-64 Y</i> ICD9 460-519 ICD10 J00-J99 | <i>Ozone</i> <i>Maximum 8 h</i> | <i>RR=1.001 (0.991-1.012)</i> ↑10µg/m ³ | <i>Anderson et al 2004</i> |
| | <i>Hospital respiratory admissions >64 Y</i> ICD9 460-519 ICD10 J00-J99 | <i>Ozone</i> <i>Maximum 8 h</i> | <i>RR=1.005 (0.998-1.012)</i> ↑10µg/m ³ | <i>Anderson et al 2004</i> |

Finally, HIA needs defining the evaluation scenarios, i.e. the hypothetical scenario with which we want to compare the current air pollution situation. We calculate the impact on health of the (current) air pollution levels in the city that are above the pollution level of the evaluation scenario. In other words, the attributable number of health events (deaths, hospital admissions...) calculated for each scenario represents the number of events that would be prevented if, all other things being equal, air pollution levels were reduced to the evaluation scenario level. These evaluation scenarios are based on the objectives and limits established in 1999/30/CE, and 2002/3/CE Directives.

HIA scenarios

1 - HIA scenarios for PM₁₀

1.1.- Scenarios for HIA on **short-term** effects of PM₁₀ and **cough, lower respiratory symptoms** in people under 18 year (<18), and **hospital respiratory admissions** in people under 15 year (< 15)

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

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

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

1.2.- Scenarios for HIA on **long-term** effects of PM₁₀ and **postneonatal mortality** (total, respiratory and sudden infant death syndrome-SIDS)

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

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

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

2.- HIA scenarios on short-term effects of Ozone

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

1.2.1.1 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/CE Directive)

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

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

1.2.2.1 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/CE Directive)

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

Findings

The annual number of postneonatal deaths attributable to a reduction of PM₁₀ levels by 5 µg/m³ was 0.28 (95%CI: 0.1-0.4), which is equivalent to an annual rate of 1.79 deaths per 100 000 (95%CI: 0.8-2.8).

Table 5. Potential benefits of reducing PM₁₀ levels. Absolute numbers and rates (per 100 000 children) (95% confidence limits) attributable to the health effects of PM₁₀.

| | PM10 reduction | Number of attributable cases per year | Annual rates (per 100.000) |
|---------------------------------------|-------------------------|---------------------------------------|-----------------------------|
| POSTNEONATAL MORTALITY | | Annual mean levels | |
| Total | by 5 µg/m ³ | 0,28 (0,13-0,43) | 1.79 (0.83-2.76) |
| | to 20 µg/m ³ | 0,0 (0,0-0,0) | 0.0 (0.0-0.0) |
| | to 40 µg/m ³ | 0,0 (0,0-0,0) | 0.0 (0.0-0.0) |
| Respiratory | by 5 µg/m ³ | 0.10 (0.05-0.16) | 0.64 (0.32-0.1.02) |
| | to 20 µg/m ³ | 0,0 (0,0-0,0) | 0.0 (0.0-0.0) |
| | to 40 µg/m ³ | 0,0 (0,0-0,0) | 0.0 (0.0-0.0) |
| SIDS | by 5 µg/m ³ | 0,0 (0,0-0,0) | 0.0 (0.0-0.0) |
| | to 20 µg/m ³ | 0,0 (0,0-0,0) | 0.0 (0.0-0.0) |
| | to 40 µg/m ³ | 0,0 (0,0-0,0) | 0.0 (0.0-0.0) |
| MORBIDITY | | Daily levels | |
| Cough <18 y | by 5 µg/m ³ | Not available | Not available |
| | to 20 µg/m ³ | | |
| | to 50 µg/m ³ | | |
| LRS <18 y | by 5 µg/m ³ | Not available | Not available |
| | to 20 µg/m ³ | | |
| | to 50 µg/m ³ | | |
| Hospital respiratory admissions <15 y | by 5 µg/m ³ | 8.67 (-1.74-18.16) | 4.29 (-0.86-8.99) |
| | to 20 µg/m ³ | 4.04 (-0.8-8.53) | 2.00 (-0.39-4.22) |
| | to 50 µg/m ³ | 0.26 (-0.05-0.54) | 0.13 (-0.02-0.27) |

Regarding short-term effects of O₃, each reduction by 10 µg/m³ of daily maximum 8-hour moving average concentrations would delay 16.3 (95%CI: 8.9-27.3) deaths per year in the study area, 10.5 (95%CI: 5.0–16.7) from cardiovascular diseases, and 4.7 (95%CI: 3.1- 6.3) from respiratory causes.

Table 6. Potential benefits of reducing ozone daily levels. Absolute numbers and rates (per 100 000 inhabitants) (95% confidence limits) attributable to the health effects of ozone.

| | OZONE reduction | Number of attributable cases per year | Annual rates (per 100.000) |
|---|----------------------------|--|--|
| MORTALITY | Daily 8-h max | | |
| Total excluding external causes | by 10 µg/m ³ | 16.29 (8.93- 27.33) | 1.37 (0.75-2.30) |
| | to 120 µg/m ³ | 0.10 (0.05-0.16) | 0.008 (0.004-0.013) |
| Cardiovascular | by 10 µg/m ³ | 10.53 (5,04- 16,71) | 0,89 (0,43-1,41) |
| | to 120 µg/m ³ | NA | NA |
| Respiratory | by 10 µg/m ³ | 4.69 (3.07- 6,27) | 0,40 (0,26-0,53) |
| | to 120 µg/m ³ | NA | NA |
| MORBIDITY | Daily 1-h max | | |
| Emergency room visits for asthma <18 y | by 10 µg/m ³ | 3.44 (2.00-4.90) | 1.38 (0.80-1.97) |
| | to 180 µg/m ³ | | |
| | Daily 8-h max | | |
| Hospital respiratory admissions 15-64 y | by 10 µg/m ³ | 1.28 (-11.51-15.35) | 0.16 (-1.43-1.90) |
| | to 120 µg/m ³ | 0.01 (-0.07-0.09) | 0.001 (-0.009-0.011) |
| Hospital respiratory admissions > 64 y | by 10 µg/m ³ | 11.69 (-4.67-28.05) | 6.60 (-2.64-15.8) |
| | to 120 µg/m ³ | 0.07 (-0.03-0.17) | 0.04 (-0.02-0.10) |

NA: Not applicable if air pollution levels are lower than the scenario level

Discussion

Only one urban background monitoring station for PM_{10} is used to estimate the exposure for Stockholm in this study. It is the same station which has been used in the epidemiological APHEA2 study, generating some of the applied exposure-responses functions. Since there are no important point sources, the above roof level is not expected to vary much across the city. The level of PM_{10} in a busy street is however found to be 3 times higher on average. Due to small spatial variations, the used station for ozone can be regarded as representative for the whole study area.

Stockholm has already low air pollution levels from an international point of view. Diesel cars are much less common than in many other parts of Europe, and the vehicle fleet is kept in a good condition. However, findings from this HIA show that even small reductions in PM_{10} levels would lead to improvements in health. A reduction of the annual PM_{10} levels by $5 \mu g/m^3$ would decrease the number of respiratory hospital admissions among children below 15 years of age with approximately 9 visits per year, and save one case of post neonatal mortality per 4 years. The number of emergency visits are likely underestimated. The result of reduced particle levels on these outcomes are not as quantitatively dramatical as the long-term effect estimated on the total mortality, but the expected effects are still notable.

For PM_{10} levels close to streets in the city centre the main cause of exceedances of the limit value is re-suspension of road dust. At present, no actions have been taken to reduce road dust re-suspension in Stockholm. At the other end of the size spectrum, the ultra-fine particles are mainly due to local road traffic exhaust. There is no limit value for these particles; their contribution to PM_{10} is almost negligible.

In Stockholm there is now an environmental zone in the city centre. The main purpose with the zone was to encourage the use of cleaner vehicles, improve air quality and to some degree reduce noise. The zones target trucks and busses. Calculations have shown that particulate matter emissions due to heavy-duty vehicle emissions within the zone have been reduced by about 40% as compared to the situation without the zone. But the corresponding reductions of the atmospheric mass concentrations are only up to 9% for $PM_{0.2}$. However, the influence of heavy-duty vehicles on the number concentration of particles is more important than if the mass concentration is considered.

The importance of wood burning in the outskirts is not known. Calculations indicate that it can be a very important source in residential areas, but the lack of knowledge on the amount of fuel actually used together with uncertainties concerning emission factors make these estimates very tentative. New stoves and boilers have much lower emissions as compared to old ones normally used today. But the number of installations of stoves in residential areas tends to increase. So far, the local authorities have run information campaigns to make people aware of the problems associated with wood burning in densely populated areas.

This HIA shows that a reduction of the daily 8-hour max summer levels of ozone with $10 \mu g/m^3$ would decrease the total number of preterm deaths with 16 per year. Further it would prevent 3.4 emergency room visits for asthma per year among those younger than 18 years, and 12 hospital respiratory admissions among those above 64 years. Most of the ozone effects would exist also after such a reduction, and occur even in winter. The ozone levels seen in Stockholm are mainly due to long distance transport from other parts

of Europe. The local production of ozone is only margin and has in Gothenburg been estimated to approximately $3 \mu\text{g}/\text{m}^3$ under normal circumstances, which also should be representative for Stockholm.

Actions taken to lower air pollution levels in the cities can have the opposite effect on the levels of ozone, especially when lowering the levels of NO. The trend in Stockholm show increasing levels of ozone both in the city and in the outskirts, which could be explained by decreasing levels of NO in the city center and higher background levels of ozone.

Consequently, in this part of Europe with very low net production of ozone, the work within EU is of great importance for lowering current levels.

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