

Quantification of deaths attributed
to air pollution in Sweden using
estimated population exposure to
nitrogen dioxide as indicator

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Summary

In the previous phase of this project a model was provided for quantifying the general population exposure to air pollution. From that work interpolated yearly mean concentrations of nitrogen dioxide were provided for the Swedish population. For the health impact assessment, we identified three studies as potential providers of exposure-response functions for the association between long-term exposure to nitrogen dioxide and mortality. These studies, despite large differences in their design, found very similar associations, 12-14 % increase in mortality per 10 µg/m³ increase in the air concentration of NO₂. To be applied in our assessment we selected an ecological study from Auckland, New Zealand, which reported a 13 % increase in non-accidental mortality (all ages) for 10 µg/m³ increase in NO₂. Based on official national data we assumed a baseline rate of 1010 deaths per 100 000 persons and year at the population weighted mean level of approximately 10 µg NO₂/m³. We then calculated the death rate and the yearly number of deaths expected at the population weighted mean exposure in each of four exposure classes above 10 µg/m³. Using the modelled levels of NO₂ as an indicator of air pollution levels from transportation and combustion, and calculating effects on mortality only above the yearly mean 10 µg/m³, we estimated excess exposure to result in 2837 (95% CI 2400-3273) deaths per year. A recent paper presenting similar calculations estimated the local contribution to urban levels of PM in Sweden to result in around 1800 deaths per year, but the authors questioned the use of risk coefficients for regional PM to assess the effect of local traffic pollutants. The new results obtained, using locally produced nitrogen dioxide as the basis for the risk assessment, resulted in an impact estimate 55 % higher than the published estimate based on PM.

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1. Introduction

Although the outdoor air quality situation in Sweden has improved dramatically during the last decades health impact of exposure to ambient air pollution is still an important issue. Ambient air includes many different components, e.g. particles, ozone, and nitrogen dioxide, which may contribute to a variety of health effects. In many areas the air pollution levels of specific compounds still exceed the health related air quality guidelines and health effects of exposure to air pollutants, even at moderate levels, have been shown in many studies during recent years (WHO 2005).

Consequently, there is an increasing need of tools to estimate the magnitude of the health effects in order to evaluate the number of people exposed to harmful pollution levels and hence to improve the basis for decisions on air pollution control strategies.

Based on an quantification of the general population exposure to nitrogen dioxide (as a yearly mean) in Sweden and exposure-response functions for health effects the Department of Public Health and Clinical Medicine at Umeå University and the Swedish Environmental Research Institute (IVL) have calculated effects on mortality of local air pollutants, using the NO₂ concentration levels as an indicator. The work has been funded by the health related environmental monitoring, Swedish Environmental Protection Agency, administered through the National Board of Health and Welfare (Socialstyrelsen).

The project has been performed in close connection to another research project at IVL within the Swedish National Air Pollution and Health Effects Program (SNAP), financed by the Swedish Environmental Protection Agency. The SNAP part of the project focuses on particles and a final report will be published during the autumn 2005.

2. Background and aims

In the first phase of this project a model was provided for quantifying the general population exposure to air pollution, initially as background nitrogen dioxide exposure at a national level. The aim was to develop a useful tool for exposure assessment, which in the future will fit also other air pollutants. The modelled results from the first phase have been presented in a separate IVL-report; Quantification of general population exposure to nitrogen dioxide in Sweden (Sjöberg K et al, 2004). In this second phase, our aim is to illustrate how to use the modelled exposure data for a health impact assessment. Given the aggregation level of the first set of exposure data, the assessment has been restricted to long-term effects on mortality, which is the most important component in most health impact assessments.

From the first phase of this project, interpolated yearly mean concentrations were provided for the Swedish 1999 population (Table 1). The geographical distribution of the calculated yearly mean background concentrations of NO₂ are presented in Figure 1.

Table 1 The estimated number of persons in Sweden exposed to different levels of nitrogen dioxide according to the first phase of this project.

Exposure class ($\mu\text{g NO}_2/\text{m}^3$)	Population weighted mean ($\mu\text{g NO}_2/\text{m}^3$)	Number of persons (N)
0-5	2,3	1863300
5-10	7,1	1559700
10-15	12,2	3503100
15-20	16,4	1368200
20-25	21,2	401450
>25	25,5	40300

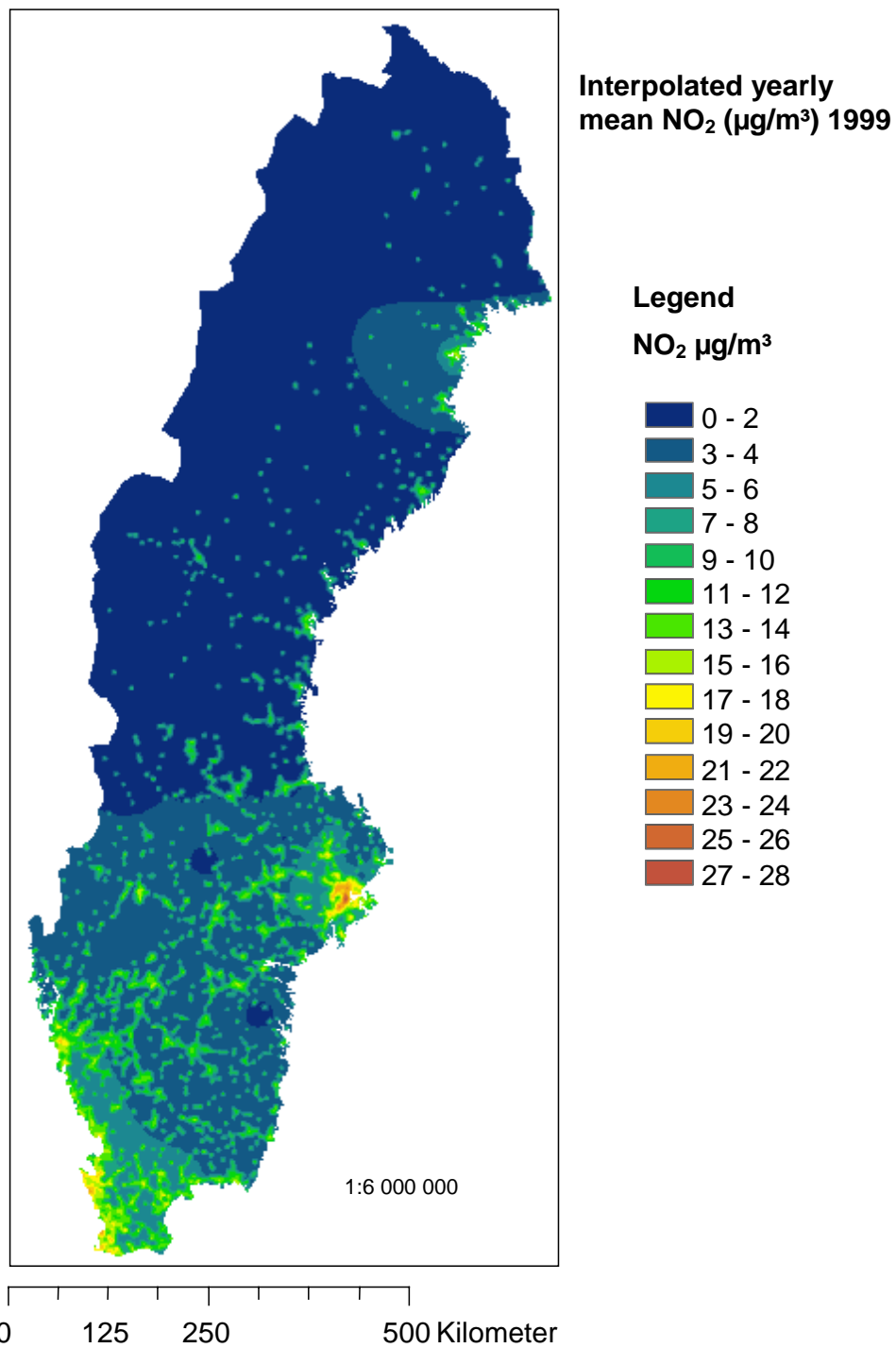


Figure 1 Interpolated yearly mean NO₂ concentrations calculated with the New Urban Model (Sjöberg, K et al., 2004)

3. Methods

3.1 Selection of exposure-response function

Given the exposure data estimated by The Swedish Environment Research Institute (IVL) in the first phase of this project, a literature survey was conducted to find a relevant exposure-response assumption for the association between air pollution levels (indicated by the annual nitrogen dioxide concentration) and mortality. This process identified only three studies with exposure data and a study design that made them potential as providers of exposure-response functions for the association between long-term exposure to nitrogen dioxide and mortality. The three studies, despite large differences in their design, found very similar associations. A Dutch cohort study reported a 12 % increase in all-cause mortality per 10 µg/m³ increase in NO₂ (Hoek et al, 2002), a French cohort study reported a 14 % increase in non-accidental mortality for 10 µg/m³ increase in NO₂ (Filleul et al, 2005), and an ecological study from Auckland, New Zealand, reported a 13 % increase in non-accidental mortality for 10 µg/m³ increase in NO₂ (Scoggins et al, 2004). A Norwegian cohort study used NO_x as exposure indicator and could not be used for this impact assessment (Naftstad et al, 2004).

3.1.1 The Netherlands Cohort Study

The ongoing cohort study “The Netherlands Cohort Study (NLCS) on diet and cancer” (Hoek et al, 2002) has been used to study the association between traffic related air pollution and mortality. The baseline data collection took place in 1986, when subjects aged 55–69 years were included and information was collected about a large number of risk factors besides diet for the development of cancer. The study sample (n= 120 852) was recruited from 204 municipalities that had computerized population registries in 1986, and were sufficiently covered by cancer registries. The exact address of all study subjects in 1986 is known. A random sample of 5000 participants has been followed up every two years for migration and vital status. In the air pollution study, this sub sample from the NLCS cohort has been analyzed. 4492 persons had answered the questionnaires, and out of these, the geographical coordinates for the addresses were identified for 4466 subjects. 5% lived close to a major road and 3% within 100 m of a freeway. 3464 subjects had information enough for full adjustment for potential confounders. 489 participants in the sub sample died during the follow up 1986-1994, most from natural causes.

Exposure was estimated using the 1986 home address and residential history information to generate indicators, on an individual basis, of long-term exposure to traffic related air pollutants. About 90% of the study population lived 10 years or more at its 1986 home address, supporting the use of the estimated concentration at the 1986 address as a relevant exposure variable.

The long-term average exposure was considered to be determined by the regional background, additional pollution from urban sources (resulting in an urban background), and for a small proportion of the subjects, additional pollution from local sources (major roads and freeways). Two traffic related air pollutants, nitrogen dioxide (NO₂) and black smoke (BS) were used as indicator pollutants. The regional component at the home address was estimated using interpolation of measurements at regional background stations. There were 24 sites for NO₂. A regression model relating degree of urbanization to air pollution was used to allow for differences between different

towns and neighbourhoods of cities. If only the regional scale is taken into account, the range in NO₂ exposure between the 10th and 90th percentile was about 67%. When differences in urbanization degree were taken into account, the difference in exposure between the 10th and 90th percentile became 76% for NO₂.

Distance to major roads was calculated to characterize the local contribution from traffic, using a Geographic Information System. The quantitative estimates of the contribution of living 50 m away from a freeway to the concentration was 11 and the contribution from major inner-city roads was estimated to be 8 µg/m³ NO₂, respectively. These estimates were assigned to each "exposed" address, independent of the actual distance to the road. However, the local contribution added this way increased only by 0,5 µg/m³ the estimated average regional + urban concentration, to 36,6 µg/m³. For NO₂ the exposure range was 14,7 – 67,2 µg/m³, including the contribution from local traffic.

In the analyses, two types of models for exposure calculations were used with regard to how the local contribution was added to the urban background. One type of models had a qualitative indicator variable for living near a major road, the other added the estimated contribution from living near a major road as a concentrations.

Adjustment was in the analyses made for a large set of potential confounding variables at individual level; age, active smoking, passive smoking, education, last occupation, Quetelet index (bodyweight divided by height squared), alcohol intake, fat intake, vegetable and fruit consumption. In addition, adjustment was made for regional indicators of poverty (income distribution, proportion of the population aged 15–64 years on social security).

Before adjustment for confounders, exposure to black smoke and nitrogen dioxide was significantly associated with all-cause mortality. The relative risk associated with an increase in NO₂ of 30 µg/m³ was 1,45 (95% CI 1,05-2,01). After adjustment for confounders, the relative risk became smaller and non-significant, 1,36 (95% CI 0,93-1,98).

The size is a clear limitation in the case of this study, which also resulted in a non-significant adjusted association. On the other hand the magnitude of the effect estimate is still not trivial, and corresponds to a 12% increase in all-cause mortality per 10 µg/m³ of NO₂.

3.1.2 The PAARC Study

In the French PAARC survey long term effects of air pollution on mortality were studied in 14 284 adults who resided in 24 areas from seven French cities (Bordeaux, Lille, Lyon, Mantes la Jolie, Marseille, Rouen, Toulouse) when enrolled in the study in 1974 (Filleul et al, 2005). For six monitoring sites, the NO/NO₂ ratio was suggesting that the exposure measure was heavily influenced by the local traffic and not representative of the mean exposure of the population in these areas. Thus, the main conclusions from this study are based on a subgroup of 18 areas that could be characterised by urban background monitoring stations, defined by a ratio of NO/NO₂ <3.

The choice of areas was based on a three step procedure, initially based on available historic air pollution data for the cities, followed by selection of potential areas and measurement points, taking into account all available information on pollution and feasibility regarding population density in the areas. Each area varied in diameter from 0.5 to 2.3 km. In the final step, air pollution measurements were set up at a centrally located pollution monitoring station in each of these areas, using available

standard methods: sulphur dioxide (specific (SO₂) and acidimetric method (AM)), total suspended particles (TSP, gravimetric method), black smoke (BS, reflectometry), nitrogen dioxide (NO₂, colorimetric analyser), and nitric oxide (NO, colorimetric analyser). Daily measurements were conducted for three years (1974–76). Indicators of air pollution were the mean concentrations during the measurement period, when the area variation for NO₂ was ranging from 12 to 61 µg/m³.

The inclusion criteria for enrolment between 1974 and 1976 were to be a member of a French family household in the area for three years or more, and to be aged 25–59 years. In an interview, a questionnaire was completed which included questions about among other things weight, height, smoking history and occupational exposures. Vital status was first searched for all subjects born in France (17 805 subjects) over three years (1995–98) in each place of birth. In addition, searches through a national register were performed. Loss to follow up was primarily related to sex, with significantly more unknown vital status for women due to changes in surname, but was unrelated to air pollution. Vital status was available until June 2001 (2533 deaths, 11 753 alive, and 2619 unknown), and cause of death until December 1998. Causes of death were obtained through the specialised department (SC8) of the National Institute of Health and Medical Research (INSERM) and for 96% of subjects.

Cox proportional hazards models were used for the analysis, controlling for individual confounders (smoking, educational level, body mass index, occupational exposure), in addition frailty models were used to take into account spatial correlation.

Models were run before and after exclusion of the six areas with monitors influenced by local traffic. After exclusion of these areas, analyses showed that non-accidental mortality increased by 14% (95% CI 3-25%) for 10 µg/m³ increase in NO₂. In particular, cardiopulmonary mortality was associated with NO₂, the increase associated with a 10 µg/m³ change in the concentration was 27 % (95% CI 4-56%).

A problem in this study is that people tended to move a lot, why the analysis was also restricted to deaths during the first 10 years of follow up (until 1986), which resulted in the same results in the estimated associations between air pollution and mortality, although with wide CIs according to the smaller number of deaths.

3.1.3 The Auckland Study

This study (Scoggins et al, 2004) is in opposite to above mentioned studies from The Netherlands and France not a cohort study. This study is an ecological cross-sectional study with the aim to investigate the relation between ambient air pollution levels and mortality in Auckland, New Zealand. Thus, in this study the data analysis was undertaken at the national census area unit level, which means that adjustments for risk factors are not done at the individual level. The census area units (CAU) typically had 3000 inhabitants, and had an average size of approximately 14 km², while in the central urban areas the average size was approximately 2 km².

In the Auckland study urban airshed modelling and GIS-based techniques were used to quantify long-term exposure to air pollution. A comprehensive emission inventory and a climate database were used to simulate air pollution concentrations, which were validated with hourly observations from several air quality monitoring sites. The models were run on a 3 km grid that covered almost the entire Auckland region. The final grid had a total of 1296 grid cells, 36 rows by 36 columns, grid cell size 9 km². The NO₂ modelled concentrations were averaged over the whole year and annual average NO₂ was used as a long-term air pollution exposure indicator. The evaluation with

measured values showed that the urban airshed modelling carried out gave an index of agreement above 0.75 for NO₂ at most sites, which is a good model performance. Modelled annual average NO₂ concentrations were converted from point-based x, y coordinates into 3 km by 3 km polygon grid coverage. Then polygon grid coverage concentrations were converted to census area unit concentrations by calculating an area-weighted average concentration for all individual units that overlapped more than one grid cell.

Mortality data were collected from New Zealand Health Information Service, for the years 1996 to 1999. External causes of mortality (deaths due accidents, violence and suicide) were excluded. The 1996 Census provided information by CAU for the Auckland region on resident population, sex, age and ethnicity.

Logistic regression was used to investigate how air pollution influences the probability of dying, while controlling for potential confounders. A binomial model was applied because of the very small denominator populations in most cells. Relative risks produced from multivariate modelling, were used to estimate the percentage increase in mortality per increase in annual average NO₂. These risk functions were also used to estimate the average annual (1996–1999) number of deaths attributed to air pollution in the Auckland region. A linear increase in mortality risk above each annual average threshold level was assumed. Several different annual threshold levels were tested.

After adjustment for age, sex, ethnicity, socio-economic status, and urban/rural domicile there was a 13% (95% CI: 11–15%) increase in non-external cause mortality per 10 µg/m³ increase in annual average NO₂. There was no significant relationship between annual average NO₂ and external cause mortality, which suggests that the effect on non-external cause mortality is less likely to be due to uncontrolled confounding. NO₂ was however significantly and more strongly related to circulatory and respiratory mortality.

Based on this exposure–response relationship and applying an annual average threshold of 13 µg/m³, the proportion of deaths from non-external causes attributable to air pollution above this level was estimated to 3.9% of all deaths in Auckland. This cut off point was chosen as it represents the mean annual average NO₂ in the Auckland region, and it was assumed that below this level, air pollution has negligible effects on mortality.

Besides the ecological design, one other limitation of this study is that the analytical methods did not control for potential spatial autocorrelations in the data, which may have resulted in an underestimation of the variance and to narrow confidence intervals.

3.2 Selected exposure-response assumption

The use of any of the three identified studies as a basis for the impact calculations would only result in a small difference in comparison with the use of the others, since they all found mortality to increase around 12–14 % per 10 µg/m³ increase in annual average NO₂. The lowest coefficient came from the Dutch study with the local contribution from major roads and freeways added to the urban background. No such street contribution has been included in the Swedish exposure data for this assessment.

We have for our assessment chosen to use the median estimate, which was from the Auckland study, and was 13% (95% CI: 11–15%) increase in non-external cause mortality per 10 µg/m³ increase in annual average NO₂.

3.3 Selected base-line death rate

In order to estimate how much mortality would decrease if air pollution levels were lower we need to use a base-line death rate. The latest national mean rate published (for 2002) by the register unit EpC, at The Swedish Board of Health and Welfare, was 1063,3 deaths per 100 000 persons. If only non-external cause of death is include, the national mean rate was 1009,5. There are variations in the rate between counties and cities, but air pollution data is not presented in such a way that it is possible to used specific base-line rates for specific parts of Sweden or regions within the country.

We have for our calculations taken the latter baseline, 1009,5 deaths from non-external causes per 100 000 persons and year in the entire population, rounded it and are assuming a rate of 1010 deaths per 100 000 persons and year at the population weighted mean exposure level. It is sometimes assumed that there is no effect of air pollution on mortality in younger persons, which could motivate exclusion of deaths below a certain age (often 30 years) in the calculations. However, the number of deaths in age range 0-30 years is less than 40 per 100 000 in Sweden, why the impact calculation results only marginally would be changed by such an exclusion. In addition, the Auckland study included all ages (Scoggins et al, 2004).

3.4 Exposure data and scenarios

Exposure data were produced in the first phase of this project by modelling described in a separate report (Sjöberg et al, 2004). This exposure information is not given stratified for age groups, why we use the entire age range in our calculations (see comments above!). Based on these data we have calculated the population weighted mean exposure to be approximately 10 µg/m³. We then used 10 µg/m³ as the lower cut off in our impact assessment scenarios, and following labelled exposure above 10 µg/m³ as “excess exposure” resulting in “excess mortality”.

Table 2 details the exposure information used for our calculations, where we have used the mean exposure levels in each class to estimate exposure above 10 µg/m³, since we assume the relative risk to be a linear function. Thus, in the calculations all persons in each exposure class are assumed to be at the mean exposure level.

We have calculated excess mortality as the yearly number deaths due to air pollution (brought forward due to air pollution). Since the relative increase in the death rate (and number of deaths) is assumed to be 1,013 (95% CI: 1,011–1,015%), or 1,3%, per 1 µg/m³ increased concentration of NO₂, the corresponding decrease in death rate would be 1-(1/RR) per 1 µg/m³, which corresponds to a decrease of 1,283% or almost 1,3%.

Table 2 Estimated excess exposure above 10 µg/m³ of nitrogen dioxide calculated from results from the first phase of this project.

Exposure class (µg/m ³)	Population weighted mean (µg/m ³)	Number of persons (N)	Mean excess exposure > 10 µg/m ³	Sum of excess exposure (µg/m ³ *pers)
0-5	2,3	1863300		
5-10	7,1	1559700		
10-15	12,2	3503100	2,2	7706820
15-20	16,4	1368200	6,4	8756489
20-25	21,2	401450	11,2	4496240
>25	25,5	40300	15,5	624650

We have then calculated the death rate and the yearly number of deaths expected at the population weighted mean exposure in each exposure class above 10 µg/m³.

4. Results

4.1 Calculated death rates at exposure levels above cut off

The death rates calculated for the exposure classes above our cut off (above a yearly mean of 10 g/m³) are presented in table 3.

Table 3 Calculated death rates in exposure classes above the cut off level of 10 µg/m³ of nitrogen dioxide and the yearly number of deaths.

Exposure class (µg/m ³)	Calculate death rate per 100 000 persons and 1 year	Number of persons (N)	Calculated yearly number of deaths
10-15	1039	3503100	36397
15-20	1094	1368200	14968
20-25	1157	401450	4645
>25	1214	40300	489

4.2 Deaths attributable to exposure levels above cut off

Using the modelled levels of NO₂ above the yearly mean 10 µg/m³ as an indicator of local air pollution contribution from transportation and combustion, and calculating effects on mortality only above the cutoff level of 10 µg/m³, we estimated the excess exposure to result in 2837 (95% CI 2400-3273) deaths per year. The major part of these excess deaths come from the two lower classes (10-20 µg/m³) including most of the persons exposed to levels above 10 µg/m³.

Table 4 Calculated yearly number of death in exposure classes above 10 µg/m³ of nitrogen dioxide at present levels, at a level of 10 µg/m³ and the excess number of deaths.

Exposure class (µg/m ³)	Number of persons (N)	Calculated yearly number of deaths at present	Calculated yearly number of deaths at 10 µg/m ³	Number of excess deaths
10-15	3503100	36397	35381	1016
15-20	1368200	14968	13819	1149
20-25	401450	4645	4055	590
>25	40300	489	407	82

5. Discussion

Nitrogen dioxide is a good indicator of air pollution from the transport sector (cars, trucks, shipping) and from other types of combustion (*e.g.* power plants). Nitrogen dioxide is a regulated pollutant and is thus frequently measured and modelled. However, this does not mean that it is very important as a causal agent behind the health effects related to air pollution.

For several years there have been different viewpoints on the health effects of nitrogen dioxide at current urban levels. Toxicologists and epidemiologists do not completely agree on how the existing body of evidence should be interpreted. Epidemiological studies have detected associations at low ambient air concentrations, most consistent for the prevalence of respiratory illness in children, but often also for the daily number of hospital admissions and the daily number of deaths. However, it is well known that NO₂ and other combustion related pollutants co-vary in time and space, making it difficult or impossible to separate their effects. Thus, epidemiological studies cannot prove that it is nitrogen dioxide per se which is the causal factor. In addition, a lot of human exposure studies have shown that normal healthy individuals do not show adverse effects to NO₂ below concentrations of about 4000 µg/m³, while subjects with asthma or chronic obstructive lung disease may react to concentrations of about 500 µg/m³, either by alterations in bronchial reactivity or by increased sensitivity to inhaled allergens. For the time being, nitrogen dioxide has to be seen as an indicator of air pollution mainly from the transport sector and other combustion sources. The fact that we in this report are assessing the impact of air pollution on mortality using nitrogen dioxide, should also be viewed in the light of nitrogen dioxide as an indicator. We do not claim that it is nitrogen dioxide per se which cause the estimated several thousands of excess deaths per year, but expect actions that reduce emissions of nitrogen dioxide to reduce the number of deaths from air pollution.

We have estimated more than 2800 deaths per year brought forward due to exposure to a local air pollution concentration indicated by nitrogen dioxide levels above $10 \mu\text{g}/\text{m}^3$ as an annual mean. This cut off, roughly set at the population weighted mean, is rather arbitrary, since we do not know the shape of the exposure-response association in different concentration intervals. There is no evidence of a specific toxicological threshold level at the cut-off level. On the other hand, we know that the regional background level of nitrogen dioxide is lower than $10 \mu\text{g}/\text{m}^3$ in most parts of the country, so the assessment in principle reflects only effects of the local contribution and not always the whole part of it, why a lower cut off could have been used for most of the country.

In a recent paper similar calculations for Sweden were presented using particulate matter (PM_{10} or $\text{PM}_{2.5}$) as the air pollution indicator (Forsberg et al, 2005). In that health impact assessment, the local contribution to urban levels of PM in Sweden was estimated to result in around 1800 deaths per year brought forward, while the impact of long-range transported pollutants was estimated to approximately 3500 deaths annually. However, the authors meant that the effect of particle emissions from local traffic likely were underestimated with the applied risk coefficients for PM from American cohort studies across regions. Our results for locally produced nitrogen dioxide resulted in an impact estimate 55 % higher than the PM estimate, supporting the hypothesis about an underestimation presented in the previous study.

Epidemiological studies as well as the method used in this study to assess health impact of harmful air pollutants have shown that NO_2 is a useful indicator for exposure estimates and calculations of effects on mortality of local air pollutants. However, to be able to use this kind of quantifications on a more routinely basis to i.a. follow up on air pollution control strategies, the framework conditions for the data assessment need to be refined in order to obtain a more comprehensive strategy for the proposed indicator.

There is still a number of issues that need to be clarified: the selection of data to be used; possible extension of the amount of air quality monitoring data; requirement of assessment frequency; application of relevant geographical areas and best degree of resolution to fit with the most valid epidemiological ER-functions; calculation uncertainties etc. These clarifications are likely to require additional evaluation.

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