

STOCKHOLM CITY REPORT

**Bertil Forsberg
Lars Modig
Bo Segerstedt**

University of Umea

Sweden

Stockholm city report

Summary of the main findings

The estimations in this study show that a reduction of the annual urban background mean level of PM₁₀ in Stockholm by 5 µg/m³, would as a long term effect decrease the number of deaths with approximately 216 per year (for year 2000). The pollution effect on mortality seen within 40 days would over one year accumulate to 60 deaths fewer, if the level was 5 µg/m³ lower.

The main source of particulate air pollution in Stockholm is road traffic (road dust) and secondary particles (long distance transported particles). The trend for particle concentrations (black smoke) has been strongly decreasing until the beginning of the 90's, but thereafter levels of PM₁₀ have been more constant.

During the study year used for this impact assessment (2000) the mean level of PM₁₀ was 17 µg/m³, measured in urban background, which is less than half the EC limit value. Despite the low urban background concentration measured, high levels of mainly coarse particles are generated at street level in the city centre. Several actions have been taken to lower the levels of air pollution in and around the city centre. One example is the creation of an “environmental zone” around the city, which limits old, high emitting heavy duty trucks and busses. Another action is the planned introduction of a road pricing system, which is expected to decrease total traffic volume in the city centre. No action is planned specifically for coarse particle emissions but one of the main controlling factors discussed is the use of studded tyres.

Background

The Stockholm city report included in APHEIS second year report details the air pollution situation and estimated health benefits of a reduction in particles levels. For example, we estimated that reducing the annual PM₁₀-level by 5 µg/m³, would decrease the number of deaths by 230 per year in a long term perspective. This estimate was done with health data for year 1999. The aim for the current study was to extend and adjust the given information to meet the information needs. During APHEIS third year we have conducted a more detailed health impact assessment (HIA) and followed up the communication of our findings. Since information also on PM_{2.5} is available, the new assessment includes also estimations of long-term mortality effects and years of life lost (YoLL) using urban PM_{2.5} as the pollution indicator. In addition we have new epidemiological results on the short-term association between PM₁₀ and acute respiratory hospital admissions.

We used the same geographical study area and study population and also the same year for air pollution data as in the second year report (2000), but now with health data representing the same year. The number of inhabitants was approximately 1 173 000.

Sources

The principal sources of air pollution were described in detail in the previous APHEIS city report published a year ago (www.apheis.org). The numbers in Table 1 is an update of the estimated main sources of air pollution during 2002.

Table 1. Main local sources of PM10

Source (%)	Road (%)	Heating (%)	Other sources (including industry) (%)
2002	80	10	10

Motor vehicle traffic is 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, but affects also the urban background. Local traffic exhaust emissions have very small impact on PM₁₀ levels. The regional background is mainly determined by long distance transport of particles.

Exposure data

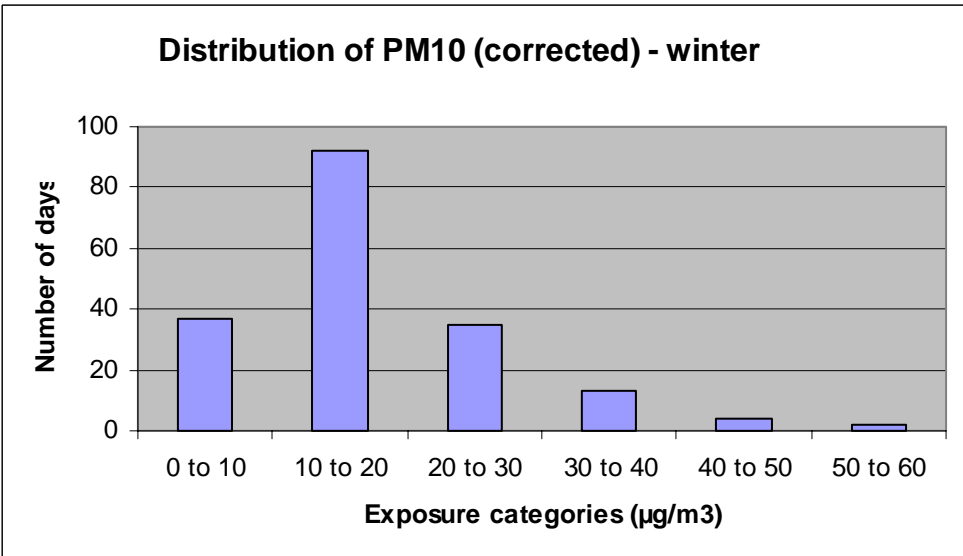
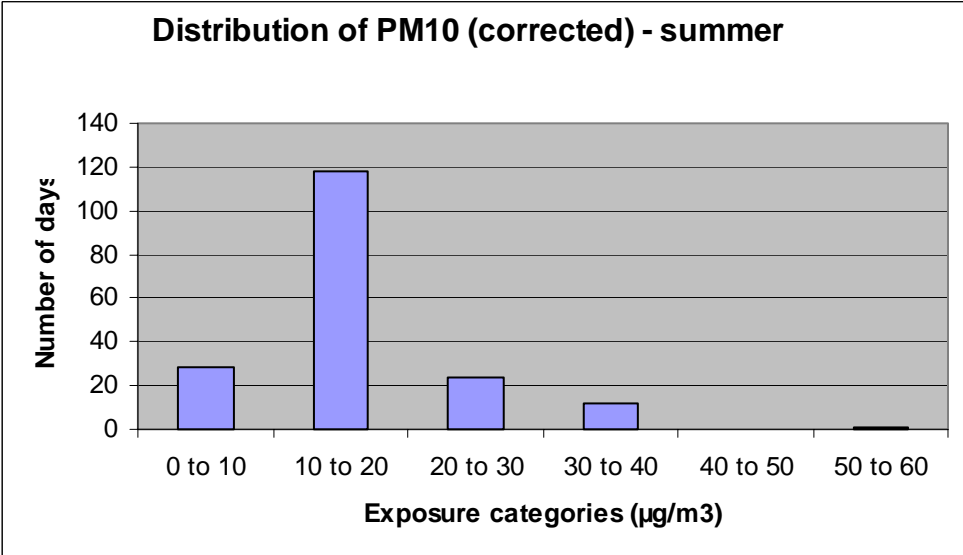
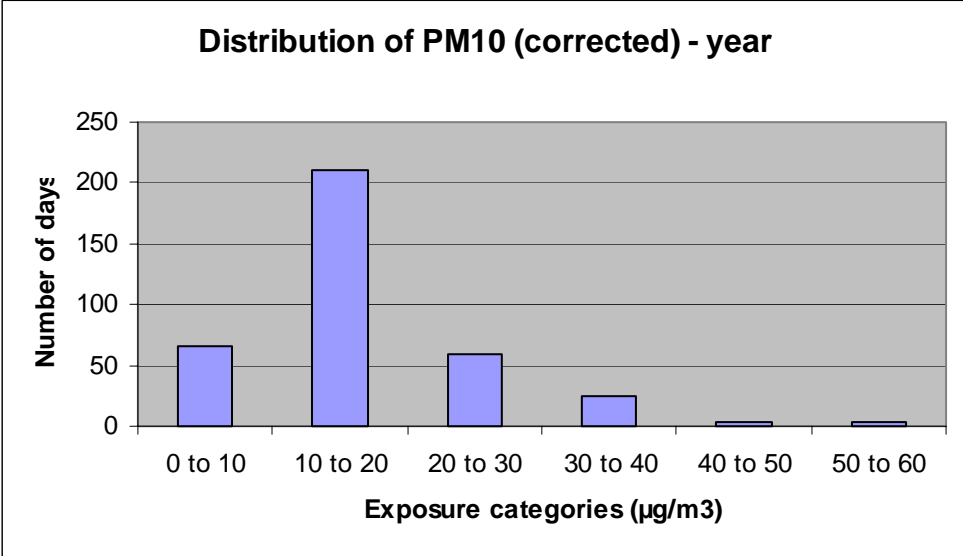
Air quality data are from the same main measuring stations as in the APHEIS second year report. Models and measurements indicate that this central site is representative for the urban background in the greater city. PM₁₀ and PM_{2.5} is measured using a TEOM instrument. For the calculations of short-term effects of PM₁₀ the originally measured levels was used (corresponds to how TEOM data from Stockholm was used to model short-term effects in APHEA2), while for the long-term calculations the TEOM-levels were adjusted by a locally determined correction factor (1.2) to match filter methods used in the long-term studies (Johansson, 2003).

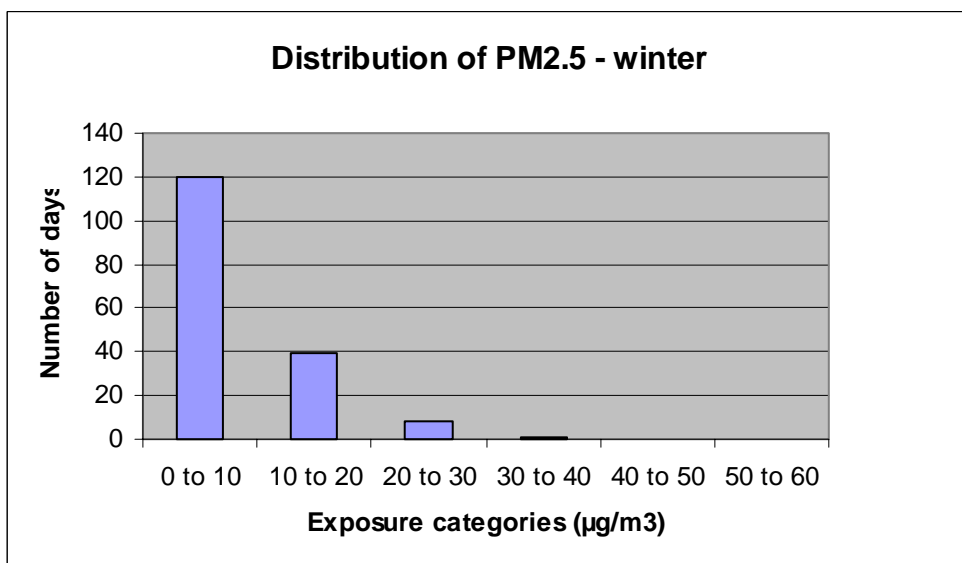
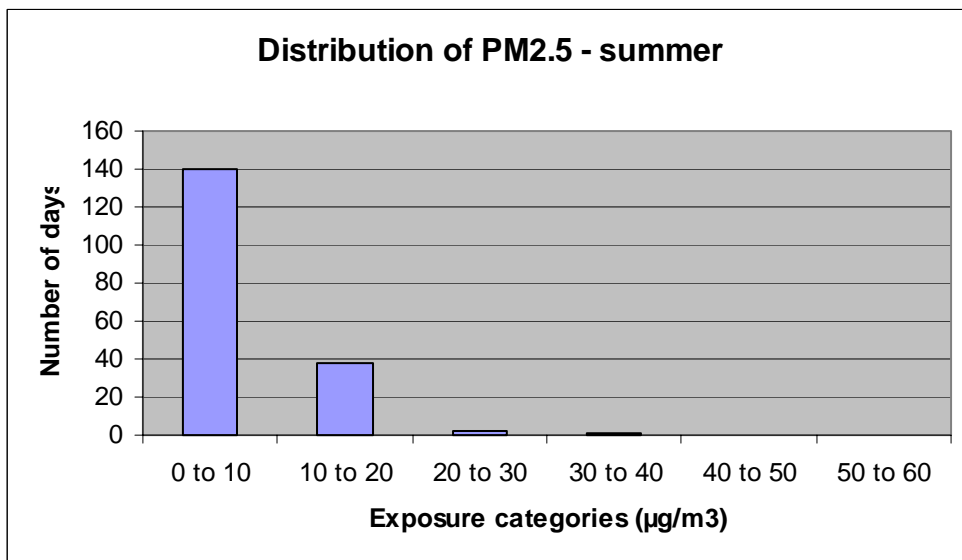
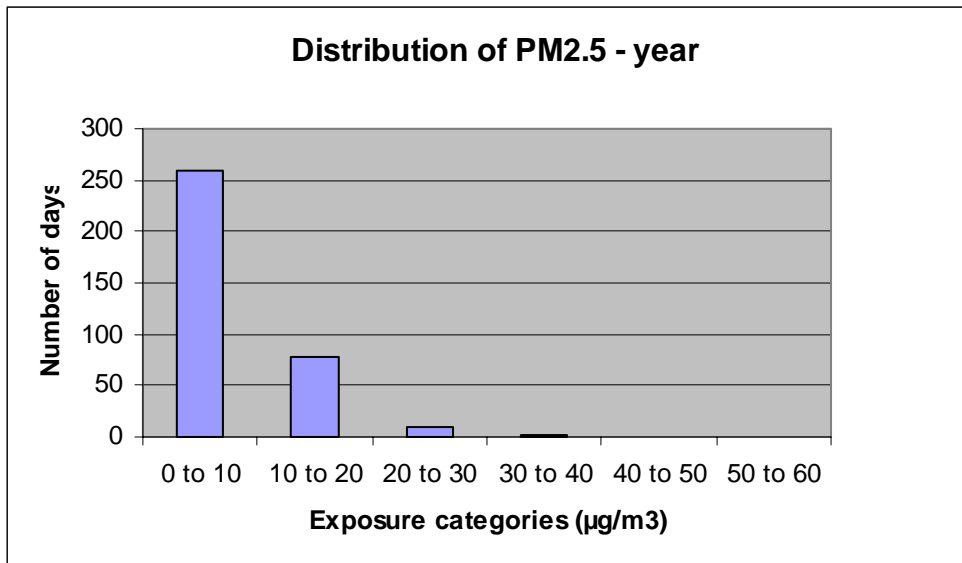
- Year of air pollution data was 2000.
- Daily mean levels (SD) of PM₁₀ and PM_{2.5} were 16.6 (8.9) µg/m³ and 9.0 (4.4) µg/m³, respectively.
- The levels of PM₁₀ reached during the days with the lowest (5th percentile) and the highest (95th percentile) were 6.6 µg/m³ and 34.3 µg/m³, respectively. For PM_{2.5} the same values were 4.6 and 18.4 µg/m³.
- The numbers of days when air pollutants exceeded limit values and scenario levels are shown in Table 2.

Table 2 . Number of days when air pollutants exceeded limit and scenario levels

Air pollutant	Short term	Long term	
	PM ₁₀	PM ₁₀	PM _{2.5}
Number of days above	20 µg/m ³	20 µg/m ³	15 µg/m ³
	91	91	29
Number of days above	50 µg/m ³	40 µg/m ³	20 µg/m ³
	3	7	12

The following six figures (Figure 1 a-f) describe the distribution of PM₁₀ and PM_{2.5} during all year, summer and winter respectively. PM₁₀ is presented as corrected values (TEOM multiplied with the correction factor 1.2) while PM_{2.5} is based on measured values.





Figures 1 a-f (above) show the distribution of PM_{10} and $\text{PM}_{2.5}$ in Stockholm during all year, winter and summer over different exposure categories.

The urban background levels may be elevated due to inversions, emission of road dust and intransportation of polluted air masses. Road dust affects 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.

Health data

- The Center for Epidemiology (EpC) is a part of the Swedish National Board of Health and Welfare and is responsible for the national registers used in APHEIS, the Cause of Death Registry and the Hospital Discharge Registry.
- Age-standardised mortality rate (per 100 000 inhabitants) for Stockholm was 769. The calculation was based on the distribution of the Total European population (both sexes combined) for year 2000 ¹

Table 3. Daily mean number and annual rate per 100 000 of deaths and hospital admissions

Health outcome	ICD9	ICD10	Daily mean number (SD)	Number of cases per 100 000
Short term HIA				
All causes mortality*	<800	A00-R99	28.3 (6.4)	2.4
Cardiovascular mortality	390-459	I00-I99	13.5 (4.1)	1.2
Respiratory mortality	460-519	J00-J99	2.3 (1.7)	0.2
Cardiac mortality	390-429	I00-I52	8.9 (3.2)	0.8
Cardiac hospital admissions	390-429	I00-I52	35.1 (8.8)	3.0
Respiratory hospital admissions	460-519	J00-J99	20.7 (7.6)	1.8
Long term HIA				
Total mortality	0-999	A00-Y98	29.3 (6.6)	2.5
Cardiopulmonary mortality	401-440	I10-I70		
	460-519	J00-J99	15.2 (4.4)	1.3
Lung cancer mortality	162	C33-C34	1.1 (1.1)	0.1

* For short and long term scenarios

Health impact assessment

Different scenarios were used to evaluate the impact of short and long-term exposure to particulate pollution. For Stockholm, these scenarios were built for two indicators of particulate pollution: PM₁₀ and PM_{2.5}. The estimated health impacts of these indicators may overlap, and caution is recommended in the interpretation of findings: under no circumstances should one add estimates for these indicators because they represent the same type of particle pollution.

¹ UNITED NATIONS. Population Division Department of Economic and Social Affairs. World Population Prospects: The 2000 Revision.

Different tools and different estimates were used to evaluate the short- and long-term impacts from particulate pollution on health (Table 4).

Table 4. Summary SHORT-TERM Health impact assessment (HIA)

	Health indicator	ICD		Tool	RR (95% IC) For 10 µg/m ³ increase	
Attributable cases		ICD9	ICD10			
	ST HIA for all cities report					
PM10	All ages, all causes mortality (excluding external causes)	< 800	A00-R99	French PSAS-9 Excel spreadsheet	WHO, 2003: 1.006 (1.004 - 1.008)	
	All ages, cardiovascular mortality	390-459	I00-I99		WHO, 2003: 1.009 (1.005 - 1.013)	
	All ages, respiratory mortality	460-519	J00-J99		WHO, 2003: 1.013 (1.005 - 1.021)	
	All ages, cardiac hospital admissions	390-429	I00-I52		Le Tertre et al. 2002: 1.006 (1.003 - 1.009)	
	All ages, respiratory hospital admissions	460-519	J00-J99		Apheis 3: 1.0114 (1.0062 - 1.0167)	
BS	All ages, all causes mortality (excluding external causes)	< 800	A00-R99	French PSAS-9 Excel spreadsheet	WHO, 2003: 1.006 (1.004 - 1.009)	
	All ages, cardiovascular mortality	390-459	I00-I99		WHO, 2003: 1.004 (1.002 - 1.007)	
	All ages, respiratory mortality	460-519	J00-J99		WHO, 2003: 1.006 (0.998 - 1.015)	
	All ages, cardiac hospital admissions	390-429	I00-I52		Le Tertre et al. 2002: 1.011 (1.004 - 1.019)	
	All ages, respiratory hospital admissions	460-519	J00-J99		Apheis 3: 1.0030 (0.9985 - 1.0075)	
PM10 Distributed lag (40 days)	All ages, all causes mortality (excluding external causes)	< 800	A00-R99	French PSAS-9 Excel spreadsheet	Zanobetti et al. 2002: 1.01227 (1.0081 - 1.0164)	
	All ages, cardiovascular mortality	390-459	I00-I99		Zanobetti et al. 2003: 1.01969 (1.0139 - 1.0255)	
	All ages, respiratory mortality	460-519	J00-J99		Zanobetti et al. 2003: 1.04206 (1.0109 - 1.0742)	
Complementary ST HIA for some cities reports						
PM10 with shrunken estimates	All ages, all causes mortality (excluding external causes)	< 800	A00-R99	French PSAS-9 Excel spreadsheet	Apheis 3: RRs and 95% CI of the shrunken estimate for each city	
					RR	
					Athens	1,012 (1,008-1,017)
					Barcelona	1,009 (1,005-1,012)
					Budapest	1,005 (0,999-1,011)
					Cracow	1,004 (0,998-1,009)
					London	1,007 (1,004-1,010)
					Madrid	1,006 (1,002-1,010)
					Paris	1,005 (1,001-1,009)
					Rome	1,011(1,006-1,015)
					Stockholm	1,006 (0,999-1,013)
					Tel-Aviv	1,006 (1,002-1,011)

Table 4 (cont), Summary LONG-TERM Health impact assessment (HIA)						
	Health indicator	ICD 9	ICD10	Tool	RR (95% IC) For 10 µg/m ³ increase	Scenarios
Long term HIA for all-cities report						
Attributable cases						Annual mean
PM10	All causes mortality (excluding external causes)	< 800	A00-R99	French PSAS-9 Excel spreadsheet	Kunzli et al, 2000 1.043 (1.026 -1.061)	Reduction to 40 µg/m ³ Reduction to 20 µg/m ³ Reduction by 5 µg/m ³
PM2.5	All causes mortality Cardiopulmonary mortality LCA	0-999 401-440 and 460-519 162	A00-Y98 I10-I70 and J00-J99 C33-C34	French PSAS-9 Excel spreadsheet	CA III Pope, 2002 1.06 (1.02 - 1.11) 1.09 (1.03 - 1.16) 1.14 (1.04 - 1.23)	Reduction to 20 µg/m ³ Reduction to 15 µg/m ³ Reduction by 3.5 µg/m ³
YoLL						Annual mean
PM2.5	All causes mortality Cardiopulmonary mortality LCA	0-999 401-440 and 460-519 162	A00-Y98 I10-I70 and J00-J99 C33-C34	WHO AirQ software	CA III Pope, 2002 1.06 (1.02 - 1.11) 1.09 (1.03 - 1.16) 1.14 (1.04 - 1.23)	Reduction to 20 µg/m ³ Reduction to 15 µg/m ³ Reduction by 3.5 µg/m ³
Complementary LT HIA for some cities report						
Prospective scenarios on air pollution, prospective scenarios on birth numbers	Local choice	-	-	WHO AirQ software	-	-

Also different approaches were used to describe the impacts:

For PM₁₀, short and long-term findings are expressed in terms of number of attributed deaths per year.

For reductions of PM_{2.5}, long-term effects are estimated in terms of:

- number of attributed deaths per year
- number of expected years of life lost for starting year of simulation.
- gain in life expectancy

Short-term scenarios

We used the following scenarios to estimate the short-term effects of exposure to PM₁₀ on mortality and hospital admissions over one year:

Short term HIA scenarios for PM₁₀

- **Short-term HIA of PM₁₀ effects within 0-1 days and HIA of PM₁₀ cumulative effects within 40 days**

We used three scenarios to estimate the acute health effects of PM₁₀ on 0-1 days and cumulative health effects of PM₁₀ up to 40 days on all causes (excluding external causes), cardiovascular and respiratory mortality over one year:

- reduction of PM₁₀ levels to a 24-hour value of 50 µg/m³ on all days exceeding this value (2005 and 2010 limit values for PM₁₀)
- reduction of PM₁₀ levels to a 24-hour value of 20 µg/m³ on all days exceeding this value (to allow for cities with low levels of PM₁₀)
- reduction by 5 µg/m³ of all the 24-hour values (to allow for cities with low levels of PM₁₀)

- **Combined local and meta-analytic estimates for short-term HIA of PM₁₀**

We used the same scenarios as above and combined local and meta-analytic default estimates to calculate the acute health effects of PM₁₀ on all causes of death (excluding external causes) over one year. This sensitivity analysis was done to study the interest of giving the weight to local estimates in a combined (shrunk) one.

Long-term scenarios

Long-term HIA scenarios for PM₁₀

We used three scenarios to estimate the chronic effects of long-term exposure to PM₁₀ on all causes mortality (excluding external causes) over one year:

- reduction of the annual mean value of PM₁₀ to a level of 40 µg/m³ (2005 limit values for PM₁₀)
- reduction of the annual mean value of PM₁₀ to a level of 20 µg/m³ (2010 limit values for PM₁₀)
- reduction by 5 µg/m³ in the annual mean value of PM₁₀ (to allow for cities with low levels of PM₁₀)

Long-term HIA scenarios for PM_{2.5}

We estimated chronic effects of PM_{2.5} in the population over 30 years old as impacts on mortality due to all causes, due to cardiopulmonary deaths and due to lung cancer deaths.

The following three pollution scenarios were considered:

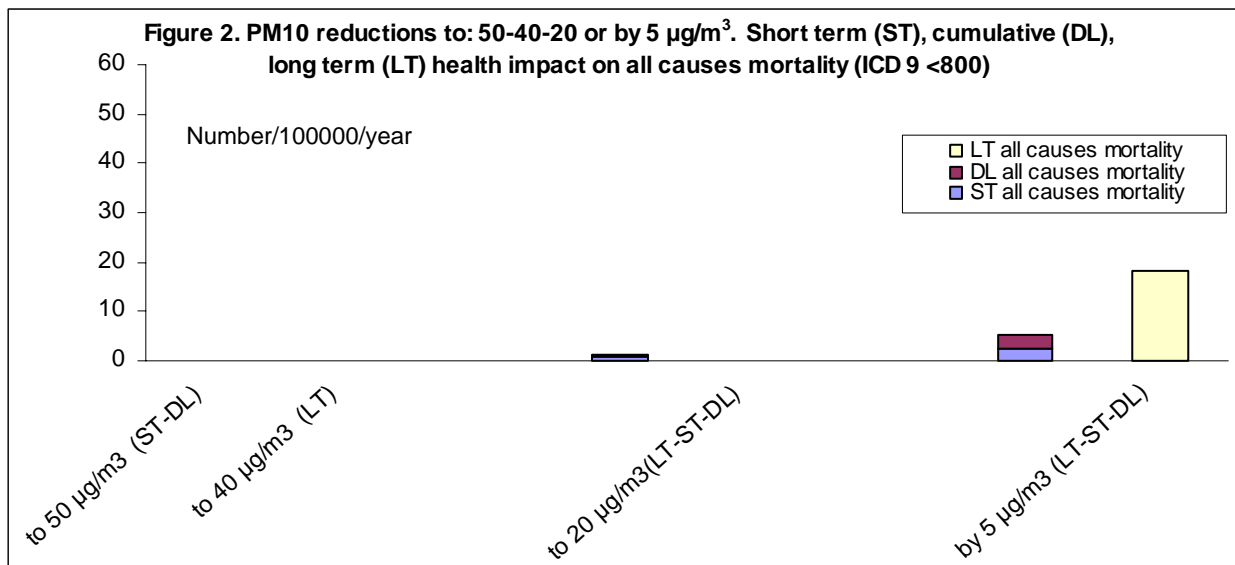
- reduction of the annual mean value of PM_{2.5} to a level of 20 µg/m³
- reduction of the annual mean value of PM_{2.5} to a level of 15 µg/m³
- reduction by 3.5 µg/m³ in the annual mean value of PM_{2.5} (to allow for cities with low levels of PM_{2.5})

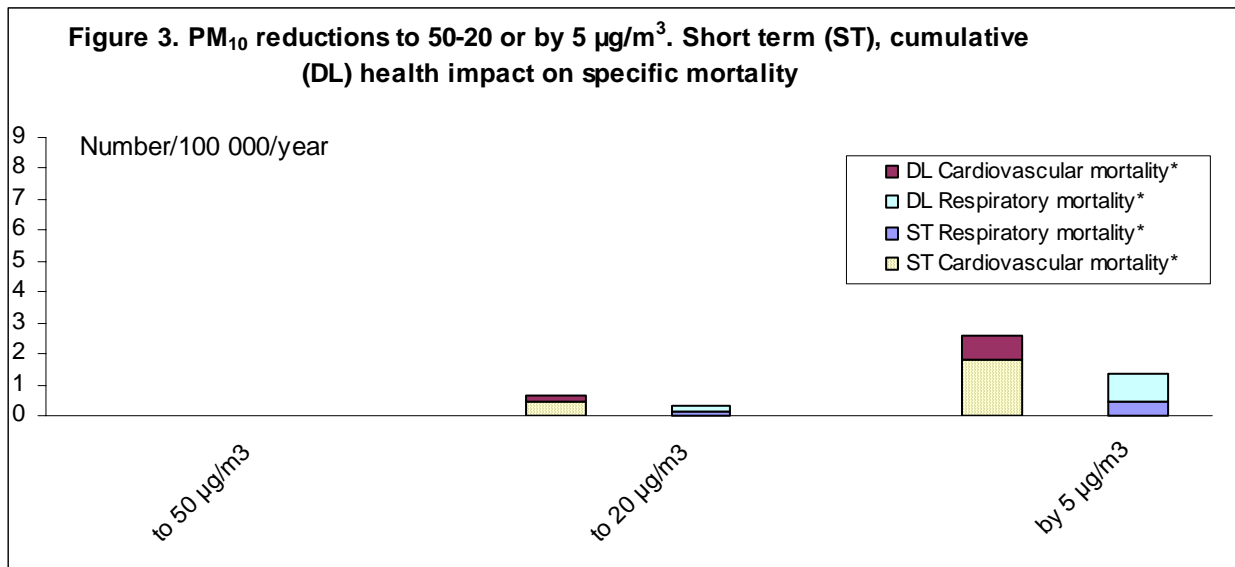
PM₁₀ findings

1. HIA of short-term PM₁₀ effects within 0-1 days, HIA of PM₁₀ cumulative effects within 40 days, and HIA of long term effects of PM₁₀

1.1. Mortality findings

The following graphs show the health impact of PM₁₀ on mortality for different lags: short-term–ST (0-1 day lag), cumulative effect–DL (distributed lag, up to 40 days lag) and long-term–LT (years).





*Cardiovascular mortality (ICD9 390-459), respiratory mortality (ICD9 460-519).

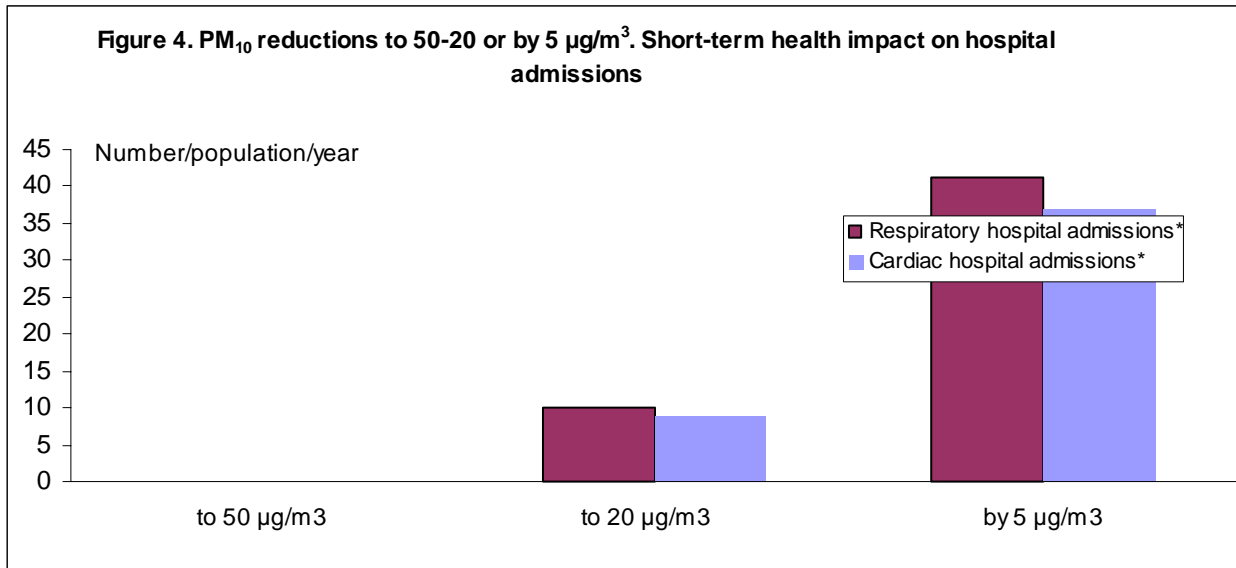
** PM10 data for 2000, mortality data for 2000

Most studies of air pollution and daily deaths have related pollution levels to death in the day or two days immediately following exposure. However, in the 1952 London smog disaster (Her Majesty's stationary service, 1954) deaths remained elevated for several weeks after air pollution levels returned to typical values. A review of other major episodes (H Ross Anderson *in Samet, Maynard*) found this the typical feature of response to air pollution. This suggests that studies that limit themselves to studying deaths within a day or two of exposure may miss some of the deaths attributable to that exposure.

Recently, several studies have looked at longer follow up periods and reported larger estimates. In particular, Zanobetti and coworkers examined up to 40 days of follow up for all causes and cardiovascular and respiratory deaths in the APHEA II study. These reports showed that the cumulative effect was more than twice that found using only two days of follow up. The mentioned results from distributed lag models are the primary basis for our estimates of the short term effects of changes in air pollution concentrations. Results using only two days of follow up are also reported for comparison.

1.2. Hospital admissions findings

We estimated the acute effects of short-term exposure to PM₁₀ on cardiac and respiratory hospital admissions over one year.



* Cardiac (ICD9 390-429) and respiratory hospital admissions (ICD9 460-519)

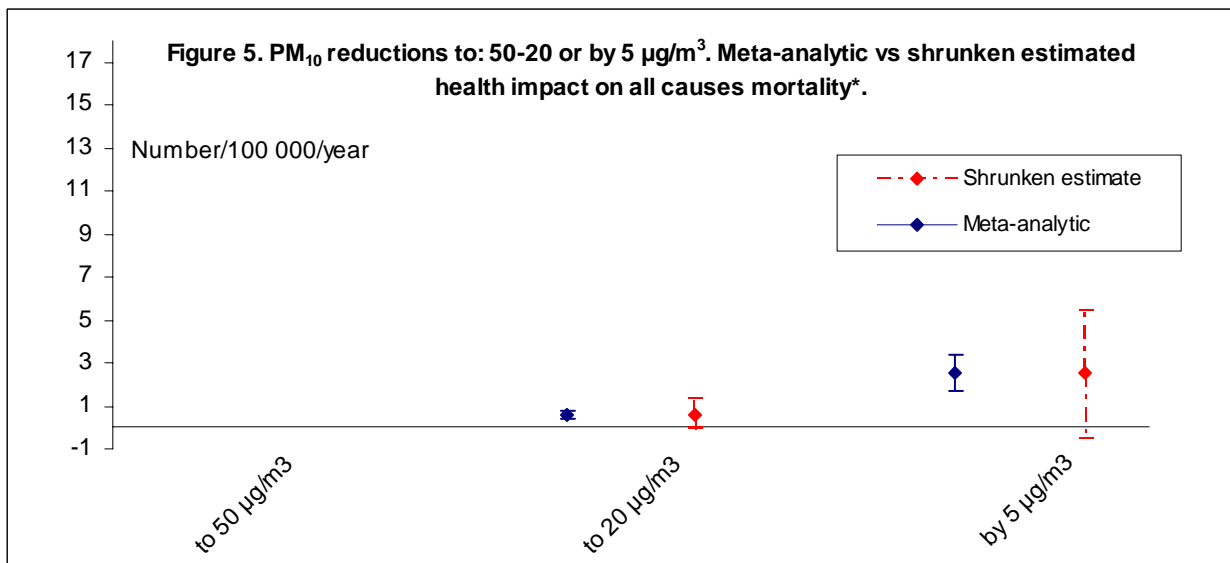
** PM10 data for 2000, mortality data for 2000

The number of attributable hospital admissions may seem low compared to the effects on mortality. However, only the short-term effect on hospital admissions is estimated, and thus the total effect on morbidity and admissions is not reflected by these calculations.

2. Combined local and meta-analytic estimates for the health effects of PM₁₀

We combined local and meta-analytic estimates (shrunken estimates-SE) to calculate the acute health effects of PM₁₀ on all causes of death (excluding external causes) over one year.

The following figure compares the HIA of PM₁₀ effects within 0-1 days using the default (meta-analytic) and the combined (shrunken) estimate with a local adjustment.



* All causes mortality excluding external causes (ICD9 < 800)

** PM10 data for 2000, mortality data for 2000

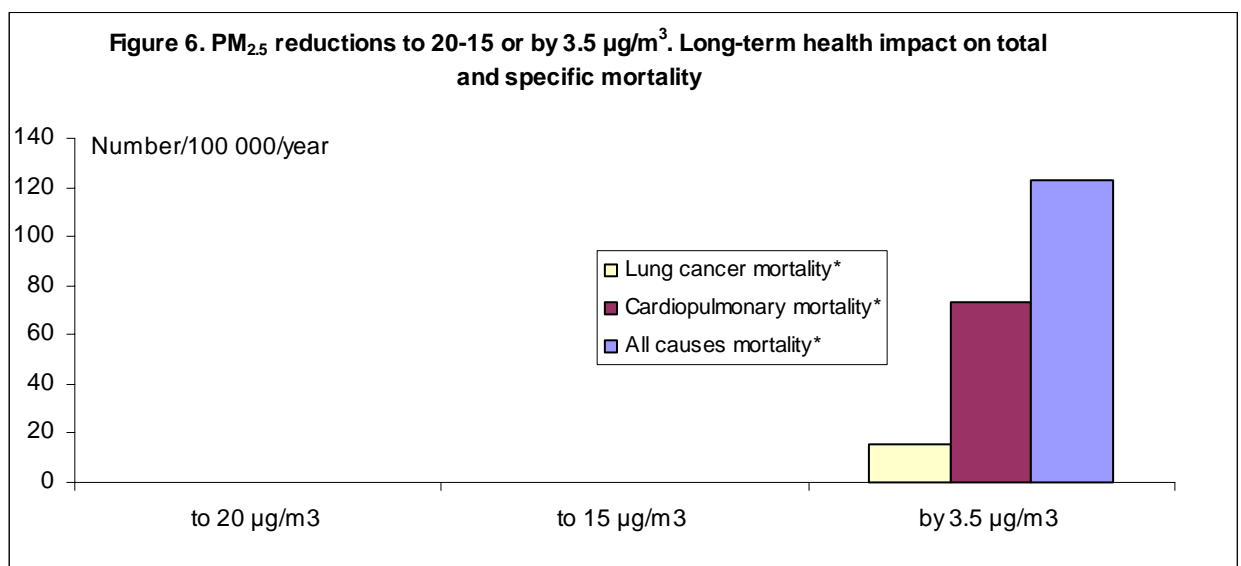
Since the effect size is very similar according to the default meta-analytic estimate and the calculated shrunken estimate, the effect of using a shrunken estimate is mainly decreased precision.

PM_{2.5} findings

1. Number of attributed cases

We also used three scenarios to estimate the chronic effects of long-term exposure to PM_{2.5} on mortality over one year.

The following graph presents the attributable number of all causes, cardiopulmonary and lung cancer deaths expressed as number per 100 000 inhabitants.



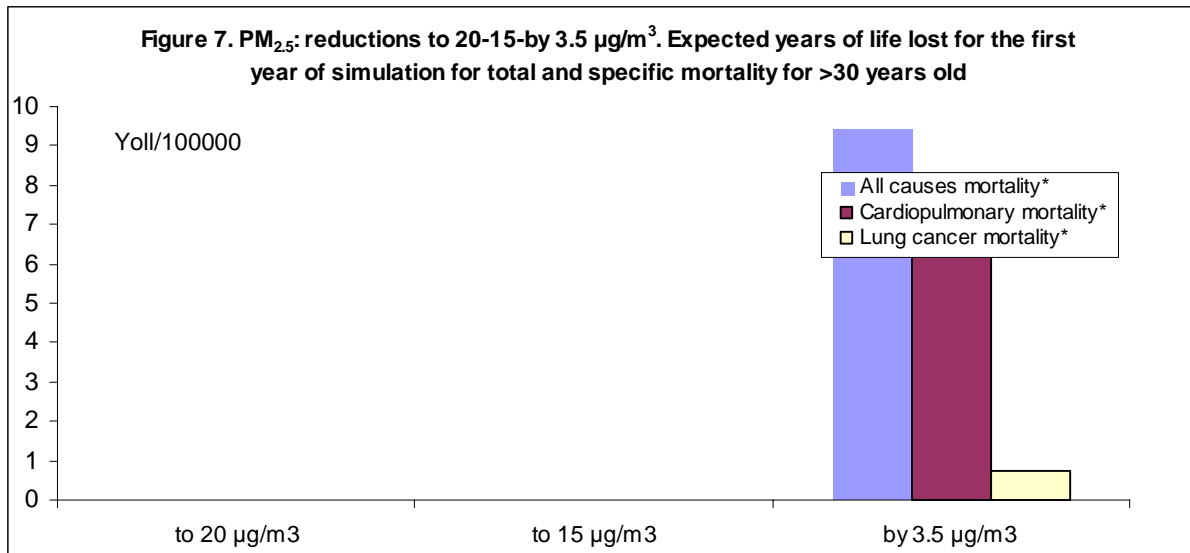
* All causes mortality (ICD9 0-999), cardiopulmonary mortality (ICD9 401-440 and 460-519), lung cancer mortality (ICD9 162)

** PM_{2.5} data for 2000, mortality data for 2000

The figure shows that cardiopulmonary deaths is the major category of mortality related to particulate air pollution, while in comparison, the number of attributable lung cancer deaths is much lower.

2. Years of life lost

We estimated the years of life lost attributable to the chronic effects of PM_{2.5} using the data for 2000. Figure 7 presents the years of life lost due to all causes, cardiopulmonary and lung cancer deaths for persons 30 years of age or older in the population of Stockholm.



* All causes mortality (ICD9 0-999), cardiopulmonary mortality (ICD9 401-440 and 460-519), lung cancer mortality (ICD9 162)

** PM_{2.5} data for 2000, mortality data for 2000

For all causes of deaths, all other things being equal, a reduction of PM_{2.5} by 3.5 µg/m³ would in the year 2000 save almost 111 years of expected life for starting year of simulation in people older than 30 years in the city of Stockholm. For cardiopulmonary mortality, this number is estimated to 84 and for lung cancer mortality, approximately 9.

The following figure presents the findings in terms of gained life expectancy.

Table 5. Life expectancy (years) and its estimated increase following a reduction of PM_{2.5} by 3.5 µg/m³ in Stockholm

Age	Life expectancy	Expected gain in life expectancy		
		Mean	Low estimate	High estimate
At birth	79.69	0.21	0.05	0.36
30	50.50	0.21	0.05	0.36
65	18.67	0.16	0.04	0.29

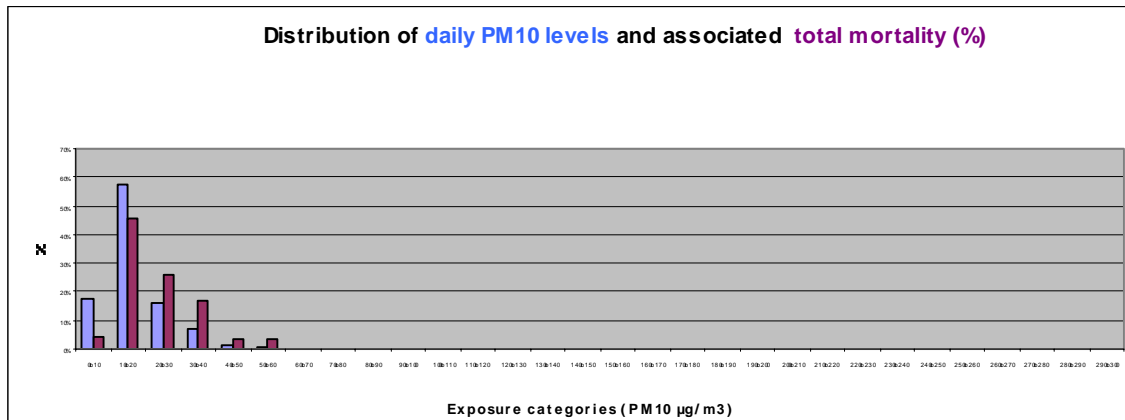
In terms of life expectancy, all other things being equal, if annual mean PM_{2.5} levels (11 µg/m³ based on PM₁₀) would be reduced 3.5 µg/m³, the years of life expectancy in a person of 30 years old would be increased by 0.2 years, due to reduced risk of death from all causes in the city of Stockholm.

Interpretation of findings

Although the particles levels in the city are low in a European perspective, the population would still benefit from further improvements. A reduction of the annual mean concentration of PM₁₀ by 5 µg/m³ or the annual mean concentration of PM_{2.5} by 3.5 µg/m³ would in the long run reduce the number of deaths by 216-217 per year according to the point estimates based on PM₁₀ and PM_{2.5}, respectively. This corresponds to an estimated loss of life expectancy of approximately 2.5 months. The intermediate effect on mortality (within 40 days) would over a year correspond to 60 less fatalities, calculated from a 5 µg/m³ reduction in the urban background concentration of PM₁₀. The “real” short-term effect is about half as

large. For hospital admission we only calculate a short-term effect (admissions related to air pollution the same day or yesterday), which of course must cause an underestimation of the impact. It is more likely that the number of excess hospital admissions due to air pollution is larger than the number of excess deaths. From the figure below it is obvious that days with particle levels between 10 and 20 $\mu\text{g}/\text{m}^3$ are most frequent, and consequently also the category with the largest impact on health.

Short term distribution of PM₁₀ levels and associated percentage of cases



General comments and conclusions

The local pollution situation

In Swedish cities exceedances of the limit values for PM₁₀ (and NO₂) occur only at street sites in major streets. According to measurements in Stockholm comparing rooftop levels (urban background levels used in APHEIS) and traffic side levels, the average PM₁₀ concentration at street level is almost 300 % higher than the urban background level (rooftop site). This means that at the same time as the City of Stockholm and other Swedish cities in international comparisons and studies like APHEIS are presented as having low air pollution levels (referring to the urban background), the cities have problems to meet the limit values not to be exceeded from 2005. Even though the levels are low on an annual basis they are very high close to roads compared to other cities in Europe when the 90 and 98 percentiles of the daily values are considered (see CAFE document on PM). These two pictures may cause some confusion. For PM₁₀ levels close to streets in the city centres the main cause of exceedances of the limit value is re-suspension of road dust. Until this year health aspects have not been used to motivate actions to reduce road dust emission in Stockholm or other Swedish cities. 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 PM₁₀ is almost negligible. However, these particles correlate well with NO_x and NO₂, and for NO₂, the exceedances of the limit value occurs in about the same streets as the exceedances of PM₁₀ levels.

In the largest Swedish cities there are now environmental zones in the city centres. The main purpose with these zones is to encourage the use of cleaner vehicles, to 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 in Stockholm have been reduced by about 40% as compared to the situation without the zone.

But compared to the total PM₁₀ emissions and the contribution to the total PM₁₀ concentration, the environmental zone has negligible impact (Johansson & Burman, 2001).

The political situation surrounding the local pollution situation (the stakes, the interests, the players)

Since traffic is the main reason for air quality problems in the main cities in southern part of Sweden, reduction of traffic related emissions are in focus. The PM₁₀ limit values in Sweden are based on the EC values. In addition there are more strict national environmental goals for future clean air. These and the new national limit values for NO₂ are currently exceeded in some busy streets and may not be accomplished in the future. The Swedish government has asked the County administration in Stockholm County to report on what is needed to meet these limit values after 2005. The Swedish National Road Administration has recently funded two projects on resuspension of road dust, one focussing on health risks and one on potential reductions from new cleaning methods.

The introduction of a congestion fee system is planned and thus very much debated in Stockholm. There will later be a local referendum. Recently the effects on air pollution levels and health have been estimated (by the Swedish APHEIS centre). Traffic models, a dynamic emission database and an air quality dispersion model have been used to calculate the effect on air pollutant levels in Stockholm. The exposure calculations have included effects on PM₁₀ and other indicators. A scenario without the road pricing system has been used as reference. Air pollutant concentrations have been combined with spatial distribution of the population to obtain population weighted means and extreme values. The modelled exposure reductions have been combined with reported exposure-response functions (for PM₁₀ and mortality the same as in APHEIS) and local base-line frequencies, to quantify health benefits expected from the pricing system.

A major part of the Swedish research funding for air pollution and health research comes from the Swedish EPA to a large programme "SNAP". Smaller projects and collaborative projects, like APHEA2, have recent years contributed important results in terms of the conclusion that even the relatively low levels in Sweden cause acute and chronic effects on health.

Information wanted in the future

During all the phases of the APHEIS project the relevant national and local authorities have been represented in the Swedish APHEIS committee. There has been a continuous exchange of information and views. The major limitation raised by the representatives of national bodies, has been the limited number of cities included in the APHEIS project. They would like to see at least an estimate for the rest of the major cities. Another question has been the possibilities to include more and less severe endpoints, i.e. emergency visits and asthma attacks.

Information given

The APHEIS project and the results in the HIA report have been presented to the local environment administrations in Stockholm and Gothenburg, at some meetings/seminars and at a press conference in Gothenburg. The main HIA report has also been distributed printed or as a file to agencies concerned with environment and health.

The results for Stockholm and Gothenburg have been published in a separate Swedish report sent to relevant national and regional agencies, and summarised in two Swedish medical journals. The press conference resulted in a large number of articles, several radio interviews and two TV interviews.

How decision makers and influencers used this information

The first APHEIS results have been published on the homepage of the Swedish EPA, The National Institute for Public Health and the two involved cities. The results have also been cited in journals, newsletters and status reports produced by bodies represented in the Swedish APHEIS committee.

A member of parliament who got the results by email, later referring to the recent results on health consequences of particles published by APHEIS, in a debate in parliament asked the Minister of environment a question on the health risks related to particles emissions from traffic.

Further communication and use of the information

The articles in news papers and medical journals, as well as the interviews in TV and radio, have resulted in a number of contacts from local politicians, journalists and NGO:s, who would like to transfer the results to other cities.

Did we meet the information needs (information type, level, quality, completeness, presentation; what was lacking).

There is no doubt that the produced information was useful for those responsible for air quality and public health. It was seen as strength that a common European approach was established, based on a large group of experts from many countries.

It was seen as a bit inconsistent that the reduction scenarios used were not completely identical for long-term effects on mortality and for hospital admissions. Also less severe effects (asthma emergency visits, attacks) were of interest for many users, and were asked for.

Since air quality already has improved a lot in Sweden, one type of questions asked was how many cases that are saved (totally or yearly) due to the improvements in air quality achieved in the two studied cities.

References

Johansson, C., 2003. TEOM – IVL's filtermetod, en metodjämförelse. SLB rapport 1:2003. Box 38 024, 100 64 Stockholm.

Johansson, C. and Burman, L., 2001. Swedish experience with low emission zones. Third international conference on health effect of vehicle emissions and pre-conference seminar on noise. November 2001, Birmingham, England.

Appendix

A1.1. Health effects of PM₁₀ on 0-1 days

Tables 1, 2, 3 present the attributable number of all causes, cardiovascular and respiratory deaths expressed as absolute numbers and as rates per 100 000 inhabitants. Table 4 presents the results for cardiac and respiratory hospital admissions.

Table 1. Deaths all causes (ICD9 < 800) (2000). Potential benefits of reducing daily PM₁₀ levels (2000) above 20 to 20 µg/m³, above 50 to 50 µg/m³ and all days by 5 µg/m³. Absolute number and number per 100 000 inhabitants (95% confidence limits) attributable to the acute effects of PM₁₀

Scenarios	Number of days per year exceeding 20 and 50 µg/m ³	Attributable cases per year					
		N° of deaths central	N° of deaths lower	N° of deaths upper	N° of deaths per 100 000 central	N° of deaths per 100 000 lower	N° of deaths per 100 000 upper
		20 µg/m ³	7.2	4.8	9.6	0.6	0.4
50 µg/m ³	-	-	-	-	-	-	
By 5 µg/m ³	NA*	29.7	19.8	39.6	2.5	1.7	3.4

*NA: not applicable

Table 2. Cardiovascular deaths (ICD9 390-459) 2000. Potential benefits of reducing daily PM₁₀ levels 2000 above 20 to 20 µg/m³, above 50 to 50 µg/m³ and all days by 5 µg/m³. Absolute number and number per 100 000 inhabitants (95% confidence limits) attributable to the acute effects of PM₁₀

Scenarios	Number of days per year exceeding 20 and 50 µg/m ³	Attributable cases per year					
		N° of deaths central	N° of deaths lower	N° of deaths upper	N° of deaths per 100 000 central	N° of deaths per 100 000 lower	N° of deaths per 100 000 upper
		20 µg/m ³	5.2	2.9	7.5	0.4	0.3
50 µg/m ³	0	0	0	0	0	0	
By 5 µg/m ³	NA*	21.3	11.8	30.7	1.8	1.0	2.6

*NA: not applicable

Table 3. Respiratory deaths (ICD9 460-519) 2000. Potential benefits of reducing daily PM₁₀ levels (2000) above 20 to 20 µg/m³, above 50 to 50 µg/m³ and all days by 5 µg/m³. Absolute number and number per 100 000 inhabitants (95% confidence limits) attributable to the acute effects of PM₁₀

Attributable cases per year							
Scenarios	Number of days per year exceeding 20 and 50 µg/m ³	N° of deaths	N° of deaths	N° of deaths	N° of deaths per 100 000	N° of deaths per 100 000	N° of deaths per 100 000
		central	lower	upper	central	lower	upper
20 µg/m ³		1.3	0.5	2.0	0.1	0.04	0.2
50 µg/m ³		0	0	0	0	0	0
By 5 µg/m ³	NA*	5.1	2.0	8.2	0.4	0.2	0.7

*NA: not applicable

Table 4. Cardiac (ICD9 390-429) and respiratory (ICD9 460-519) hospital admissions (2000). Potential benefits of reducing daily PM₁₀ levels (2000) above 20 to 20 µg/m³, above 50 to 50 µg/m³ and all days by 5 µg/m³. Absolute number (95% confidence limits) attributable to the acute effects of PM₁₀

Attributable cases per year				
Scenarios	Number of days per year exceeding 20 and 50 µg/m ³	N° of deaths	N° of deaths	N° of deaths
		central	lower	upper
Hospital admissions for cardiac diseases (all ages)				
20 µg/m ³		9.0	4.5	13.5
50 µg/m ³		0	0	0
By 5 µg/m ³	NA*	37.0	18.5	55.4
Hospital admissions for respiratory diseases (all ages)				
20 µg/m ³		10.1	5.5	14.8
50 µg/m ³		0	0	0
By 5 µg/m ³	NA*	41.1	22.4	60.1

*NA: not applicable

A1.2. Cumulative health effects of PM₁₀ up to 40 days

Tables 5, 6, 7 present the attributable number of all causes, cardiovascular and respiratory deaths expressed as absolute numbers and as rates per 100 000 inhabitants.

Table 5. Cumulative health effects of PM₁₀ up to 40 days and all causes of deaths (ICD 9 < 800) (2000). Potential benefits of reducing daily PM₁₀ levels (2000) above 20 to 20 µg/m³, above 50 to 50 µg/m³ and all days by 5 µg/m³. Absolute number and number per 100 000 inhabitants (95% confidence limits) attributable to the acute effects of PM₁₀

Scenarios	Number of days per year exceeding 20 and 50 µg/m ³	Attributable cases per year					
		N° of deaths		N° of deaths per 100 000		N° of deaths per 100 000	
		central	lower	upper	central	lower	upper
20 µg/m ³		14.8	9.8	19.8	1.3	0.8	1.7
50 µg/m ³		0	0	0	0	0	0
By 5 µg/m ³	NA*	60.4	39.9	80.6	5.2	3.4	6.9

*NA: not applicable

Table 6. Cumulative health effects of PM₁₀ up to 40 days and cardiovascular deaths (ICD9 390-459) (2000). Potential benefits of reducing daily PM₁₀ levels (2000) above 20 to 20 µg/m³, above 50 to 50 µg/m³ and all days by 5 µg/m³. Absolute number and number per 100 000 inhabitants (95% confidence limits) attributable to the acute effects of PM₁₀

Scenarios	Number of days per year exceeding 20 and 50 µg/m ³	Attributable cases per year					
		N° of deaths		N° of deaths per 100 000		N° of deaths per 100 000	
		central	lower	upper	central	lower	upper
20 µg/m ³		7.5	5.3	9.7	0.6	0.5	0.8
50 µg/m ³		0	0	0	0	0	0
By 5 µg/m ³	NA*	30.2	21.3	39.0	2.6	1.8	3.3

*NA: not applicable

Table 7. Cumulative health effects of PM₁₀ up to 40 days and respiratory deaths (ICD9 460-519) (2000). Potential benefits of reducing daily PM₁₀ levels (2000) above 20 to 20 µg/m³, above 50 to 50 µg/m³ and all days by 5 µg/m³. Absolute number and number per 100 000 inhabitants (95% confidence limits) attributable to the acute effects of PM₁₀

Scenarios	Number of days per year exceeding 20 and 50 µg/m ³	Attributable cases per year					
		N° of deaths	N° of deaths	N° of deaths	N° of deaths per 100 000	N° of deaths per 100 000	N° of deaths per 100 000
		central	lower	upper	central	lower	upper
20 µg/m ³		4.2	1.07	7.4	0.4	0.1	0.6
50 µg/m ³		0	0	0	0	0	0
By 5 µg/m ³	NA*	16.0	4.2	28.0	1.4	0.4	2.4

*NA: not applicable

A1.3. Combined local and meta-analytic estimates for the health effects of PM₁₀

Table 8 presents the attributable number of all causes of deaths expressed as absolute numbers and as rates per 100 000 inhabitants.

Table 8. Combined local and meta-analytic estimates for the health effects of PM₁₀ and all causes of deaths (ICD9 < 800) (2000). Potential benefits of reducing daily PM₁₀ levels (2000) above 20 to 20 µg/m³, above 50 to 50 µg/m³ and all days by 5 µg/m³. Absolute number and number per 100 000 inhabitants (95% confidence limits) attributable to the acute effects of PM₁₀

Scenarios	Number of days per year exceeding 20 and 50 µg/m ³	Attributable cases per year					
		N° of deaths	N° of deaths	N° of deaths	N° of deaths per 100 000	N° of deaths per 100 000	N° of deaths per 100 000
		central	lower	upper	central	lower	upper
20 µg/m ³		7.0	-1.3	15.4	0.6	-0.1	1.3
50 µg/m ³		0	0	0	0	0	0
By 5 µg/m ³	NA*	28.8	-5.5	63.1	2.5	-0.5	5.4

*NA: not applicable

A1.4. Long term HIA for PM₁₀

Table 9 presents the attributable number of all causes of deaths expressed as absolute numbers and as rates per 100 000 inhabitants.

Table 9. Deaths all causes (ICD9 < 800) (2000). Potential benefits of reducing annual mean values of PM₁₀ (2000) to levels of 20 and 40 µg/m³, and by 5 µg/m³. Absolute number of deaths and number of deaths per 100 000 inhabitants (95% confidence limits) attributable to the chronic effects of PM₁₀

Attributable cases per year						
	N° of deaths	N° of deaths	N° of deaths	N° of deaths per 100 000	N° of deaths per 100 000	N° of deaths per 100 000
	central	lower	upper	central	lower	upper
20 µg/m ³	-	-	-	-	-	-
40 µg/m ³	-	-	-	-	-	-
By 5 µg/m ³	215.6	130.9	304.5	18.4	11.2	26.0

A2. Tables for PM_{2.5} findings

A2.1. LT PM_{2.5}: Attributable Cases

Tables 1, 2, 3 present the attributable number of all causes, cardiopulmonary and lung cancer deaths expressed as absolute numbers and as rates per 100 000 inhabitants.

Table 1. Deaths all causes (ICD9 0-999) (2000). Potential benefits of reducing annual mean values of PM_{2.5} (2000) to levels of 15 and 20 µg/m³, and by 3,5 µg/m³. Absolute number of deaths and number of deaths per 100 000 inhabitants (95% confidence limits) attributable to the chronic effects of PM_{2.5}

Attributable cases per year						
	N° of deaths	N° of deaths	N° of deaths	N° of deaths per 100 000	N° of deaths per 100 000	N° of deaths per 100 000
	central	lower	upper	central	lower	upper
15 µg/m ³	-	-	-	-	-	-
20 µg/m ³	-	-	-	-	-	-
3.5 µg/m ³	215.6	56.1	378.0	18.4	4.8	32.2

Table 2. Cardiopulmonary deaths (ICD9 401-440 and 460-519) (2000). Potential benefits of reducing annual mean values of PM_{2.5} (2000) to levels of 15 and 20 µg/m³, and by 3,5 µg/m³. Absolute number of deaths and number of deaths per 100 000 inhabitants (95% confidence limits) attributable to the chronic effects of PM_{2.5}

Attributable cases per year						
	N° of deaths	N° of deaths	N° of deaths	N° of deaths per 100 000	N° of deaths per 100 000	N° of deaths per 100 000
	central	lower	upper	central	lower	upper
15 µg/m ³	-	-	-	-	-	-
20 µg/m ³	-	-	-	-	-	-
20 µg/m ³	164.7	59.1	272.6	14.0	5.0	23.2

Table 3. Lung cancer deaths (ICD9 162) (2000). Potential benefits of reducing annual mean values of PM_{2.5} (2000) to levels of 15 and 20 µg/m³, and by 3,5 µg/m³. Absolute number of deaths and number of deaths per 100 000 inhabitants (95% confidence limits) attributable to the chronic effects of PM_{2.5}

Attributable cases per year						
	N° of deaths	N° of deaths	N° of deaths	N° of deaths per 100 000	N° of deaths per 100 000	N° of deaths per 100 000
	central	lower	upper	central	lower	upper
15 µg/m ³	-	-	-	-	-	-
20 µg/m ³	-	-	-	-	-	-
3.5 µg/m ³	16.9	5.7	28.5	1.4	0.5	2.4

A2.2. LT PM2.5: Years of Life Lost

Tables 4, 5, 6 present the years of life lost for starting year of simulation of all causes, cardiopulmonary and lung cancer deaths expressed as absolute numbers and as rates per 100 000 inhabitants.

Table 4. Deaths all causes >30 years, male and female, for starting year of simulation (ICD9 0-999) (2000). Potential benefits of reducing annual mean values of PM_{2,5} (2000) to levels of 15 and 20 µg/m³, and by 3,5 µg/m³. Years of life lost (YoLL) and YoLL per 100 000 inhabitants (95% confidence limits) attributable to the chronic effects of PM_{2,5}

Years of life lost						
	YoLL	YoLL	YoLL	YoLL	YoLL	YoLL
	central	lower	upper	per 100 000	per 100 000	per 100 000
15 µg/m ³	-	-	-	-	-	-
20 µg/m ³	-	-	-	-	-	-
3.5/m3	111.0	27.8	193.0	9.4	2.4	16.4

Table 5. Cardiopulmonary deaths >30 years, male and female, for starting year of simulation (ICD9 401-440 and 460-519) (2000). Potential benefits of reducing annual mean values of PM_{2,5} (2000) to levels of 15 and 20 µg/m³, and by 3,5 µg/m³. Years of life lost (YoLL) and YoLL per 100 000 inhabitants (95% confidence limits) attributable to the chronic effects of PM_{2,5}

Years of life lost						
	YoLL	YoLL	YoLL	YoLL	YoLL	YoLL
	central	lower	upper	per 100 000	per 100 000	per 100 000
15 µg/m ³						
20 µg/m ³						
3.5 µ/m3	84.3	30.7	136.8	7.2	2.6	11.6

Table 6. Lung cancer deaths >30 years, male and female, for starting year of simulation (ICD9 162) (2000). Potential benefits of reducing annual mean values of PM_{2,5} (2000) to levels of 15 and 20 µg/m³, and by 3,5 µg/m³. Years of life lost (YoLL) and YoLL per 100 000 inhabitants (95% confidence limits) attributable to the chronic effects of PM_{2,5}

Years of life lost						
	YoLL	YoLL	YoLL	YoLL	YoLL	YoLL
	central	lower	upper	per 100 000	per 100 000	per 100 000
15 µg/m ³						
20 µg/m ³						
3.5 µg/m3	8.6	2.9	14.1	0.7	0.3	1.2

Appendix 3

Harmonised compilation of information indicating the exposure relevant area of the city, number of PM10 or BS monitoring sites, and the type, sampling and measurement characteristics of stations selected for the HIA of APHEIS

- | | | |
|-----|--|--|
| 1. | City: | Stockholm |
| 2. | Total area of agglomeration (km ²): | 500 |
| 3. | Area (km ²) covered by the air monitoring network in the city: | approx 500 |
| 4. | Number of population in this (exposure relevant) area: | 1 163 015 |
| 5. | Total number of PM10 monitoring stations in this area: | 3 |
| 6. | Total number of BS monitoring stations in this area: | - |
| 7. | Total number of TSP monitoring stations in this area: | - |
| 8. | Number of selected PM10 monitoring stations for HIA: | 1 |
| 9. | Number of selected BS monitoring stations for HIA: | - |
| 10. | Number of selected TSP monitoring stations for HIA: | - |
| 11. | Measurement interval (please cross) | |
| | continuous <input checked="" type="checkbox"/> | hourly 24 hours weekly 2 weekly |
| 12. | Quality assurance and control (please cross) | |
| | Yes <input checked="" type="checkbox"/> | no do not know |
| 13. | Data quality (please cross) | |
| | validated data <input checked="" type="checkbox"/> | invalidated data |

