REPORT

# Quantification of population exposure to $PM_{2.5}$ and $PM_{10}$ in Sweden 2005

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### Title and subtitle of the report

Quantification of population exposure to PM<sub>2.5</sub> and PM<sub>10</sub> in Sweden 2005.

### Summary

The population exposure to  $PM_{2.5}$  and  $PM_{10}$  in ambient air for the year 2005 has been quantified (annual and daily mean concentrations) and the health and associated economic consequences have been calculated based on these results. The  $PM_{10}$  urban background concentrations are found to be rather low compared to the environmental standard for the annual mean (40 µg/m<sup>3</sup>) in most of the country. However, in some parts, mainly in southern Sweden, the concentrations were of the same magnitude as the environmental objective (20 µg/m<sup>3</sup> as an annual mean) for the year 2010. The majority of people, 90%, were exposed to annual mean concentrations of  $PM_{10}$  less than 20 µg/m<sup>3</sup>. Less than 1% of the Swedish inhabitants experienced exposure levels of  $PM_{10}$  above 25 µg/m<sup>3</sup>. The urban background concentrations of  $PM_{2.5}$  were in the same order of magnitude as the environmental objective (12 µg/m<sup>3</sup> as an annual mean for the year 2010) in quite a large part of the country. About 50% of the population was exposed to  $PM_{2.5}$  annual mean concentrations less than 10 µg/m<sup>3</sup>, while less than 2% experienced levels above 15 µg/m<sup>3</sup>.

Using a cut off at 5  $\mu$ g/m<sup>3</sup> of PM<sub>10</sub> as the annual mean (roughly excluding natural PM) and source specific ER-functions, we estimate approximately 3 400 premature deaths per year. Together with 1 300 - 1 400 new cases of chronic bronchitis, around 1 400 hospital admissions and some 4.5-5 million RADs, the societal cost for health impacts is estimated at approximately 26 billion SEK per year. For PM<sub>2.5</sub> we estimate somewhat lower numbers, approximately 3 100 premature deaths per year.

The results suggest that the health effects related to high annual mean levels of PM can be valued to annual socio-economic costs (welfare losses) of ~26 billion Swedish crowns (SEK) during 2005. Approximately 1.4 of these 26 billion SEK consist of productivity losses for society. Furthermore, the amount of working and studying days lost constitutes some ~0.1% of the total amount of working and studying 2005.

### Keyword

PM<sub>2.5</sub>, PM<sub>10</sub>, particles, population exposure, health impact assessment, risk assessment, socio-economic valuation

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# Summary

The concentrations of particulate matter (PM) in ambient air still have significant impact on human health, even though a number of measures to reduce the emissions have been implemented during the last decades. The air quality standards are exceeded in many areas, and a recent study estimated that more than 5 000 premature deaths in Sweden per year are due to PM exposure.

IVL Swedish Environmental Research Institute and the Department of Public Health and Clinical Medicine at Umeå University have, on behalf of the Swedish EPA, performed a health impact assessment (HIA) for the year 2005. The population exposure to annual mean concentrations of  $PM_{10}$  and  $PM_{2.5}$  in ambient air has been quantified and the health and associated economic consequences have been calculated based on these results.

Environmental standards as well as environmental objectives are to be met everywhere, even at the most exposed kerb sites. However, for exposure calculations it is more relevant to use urban background data, on which available exposure-response functions are based. The results from the urban modelling show that in 2005 most of the country had rather low  $PM_{10}$  urban background concentrations, compared to the environmental standard for the annual mean (40 µg/m<sup>3</sup>). However, in some parts, mainly in southern Sweden the concentrations were of the same magnitude as the environmental objective (20 µg/m<sup>3</sup> as an annual mean) for the year 2010. The majority of people, 90%, were exposed to annual mean concentrations of  $PM_{10}$  less than 20 µg/m<sup>3</sup>. Less than 1% of Swedish inhabitants experienced exposure levels of  $PM_{10}$  above 25 µg/m<sup>3</sup>.

The modelling results regarding  $PM_{2.5}$  show that the urban background concentrations in 2005 were of the same order of magnitude as the environmental objective (12 µg/m<sup>3</sup> as an annual mean for the year 2010) in a quite large part of the country. About 50% of the population was exposed to  $PM_{2.5}$  annual mean concentrations less than 10 µg/m<sup>3</sup>, while less than 2% experienced levels above 15 µg/m<sup>3</sup>.

Further, in order to reflect the assumption that the relative risk factors for health impact are higher for combustion related particles than for particles from other sources, the total  $PM_{10}$  concentration was also separated into different source contributions by using a multivariate method.

Health impact assessments are built on epidemiological findings, exposure-response functions and population relevant rates, combined with estimated population exposure. We have estimated the yearly mean "background" PM<sub>10</sub>, largely natural, to be approximately 5  $\mu$ g/m<sup>3</sup>, and have used 5  $\mu$ g/m<sup>3</sup> as a lower cut off in our impact assessment scenarios and accordingly defined exposure above 5  $\mu$ g/m<sup>3</sup> as excess exposure resulting in "excess cases". For PM<sub>2.5</sub> the corresponding cut off was set at 4  $\mu$ g/m<sup>3</sup>.

There is currently a focus within the research community on the different types of particles; here are more and more indications that their impact on health and mortality differ. Yet a common view is that current knowledge does not allow precise quantification of the health effects of PM emissions from different sources. Nonetheless, when the impact on mortality is predicted for  $PM_{10}$  exposure, exposure-response functions obtained using  $PM_{2.5}$  are adjusted, usually using the  $PM_{2.5}/PM_{10}$  concentration ratio.

The long-term effect of PM<sub>2.5</sub> on mortality has been assumed to be 6 % for a 10  $\mu$ g/m<sup>3</sup> increment of PM<sub>2.5</sub>, based on a large American study, and often used in the European CAFE studies. For PM<sub>10</sub> the adjusted coefficient 4.3 % has mostly been used, as in the European APHEIS study.

Recent studies have shown that within-city gradients in mortality indicate a stronger effect on mortality than expected from between-city studies. In a study of Los Angeles the relative risk per 10  $\mu$ g/m<sup>3</sup> PM<sub>2.5</sub> was reported to be 17 %, or nearly 3 times larger than in models relying on between-community exposure contrasts. Coarse (PM<sub>10-2.5</sub>) and crustal particles have not been associated with mortality in the cohort studies, and have shown inconsistent results for short-term effects on mortality.

Despite the fact that usually, as in CAFE, all PM regardless of source is considered as having the same effect per mass concentration, we have used a less conservative approach in this study for  $PM_{10}$  and mortality. We have chosen to assume that road dust has a smaller effect and that primary combustion PM has a larger effect than the typical, total mix of particles in the US cohort studies, which were largely composed of secondary particles.

For primary combustion particles we have applied the exposure-response coefficient 17 % per 10  $\mu$ g/m<sup>3</sup>. For road dust we assume only a "short-term" effect on mortality of the same size as PM<sub>10</sub> in general. From the European study APHEA2 we chose to assume a cumulative effect of 1 % increase in all cause non-external mortality per 10  $\mu$ g/m<sup>3</sup>. For PM<sub>10</sub> in general (other sources) we have adopted the exposure-response coefficient 4.3 % per 10  $\mu$ g/m<sup>3</sup> converted from the American PM<sub>2.5</sub> results and in the APHEIS project among others. For PM<sub>2.5</sub> we do not have calculations of the contribution from different sources, so we simply apply the 6 % per 10  $\mu$ g/m<sup>3</sup> as was done by CAFE.

For morbidity we have in this study included only some of the potentially available health endpoints to be selected. We have decided to include some important and commonly used endpoints that allow comparisons with other health impact assessments and health cost studies. The question of whether one should convert ER-functions between  $PM_{2.5}$  and  $PM_{10}$  is here less easy. We have decided to do so for restricted activity days (RADs), but not for hospital admissions and chronic bronchitis.

In order to estimate how many deaths and hospital admissions that depend on elevated air pollution exposure we need to use a baseline rate. For our study of  $NO_2$  (Sjöberg et al, 2007), we used the official national death rates for 2002 and hospital admission rates for 2004. Since these rates change slowly, and for the sake of comparability, we used the same rate in this study.

Using a cut off at 5  $\mu$ g/m<sup>3</sup> of PM<sub>10</sub> as the annual mean (roughly excluding natural PM) and source specific ER-functions, we estimate approximately 3 400 premature deaths per year. Together with 1 300 - 1 400 new cases of chronic bronchitis, around 1 400 hospital admissions and some 4.5-5 million RADs, the societal cost for health impacts is estimated at approximately 26 billion SEK per year. For PM<sub>2.5</sub> we estimate somewhat lower numbers, approximately 3 100 premature deaths per year.

The cut off levels used in this study for  $PM_{10}$  and  $PM_{2.5}$  are rather arbitrary, since we do not exactly know the natural background levels nor the shape of the exposure-response association in different concentration intervals. The commonly used conversion of exposure-response functions between  $PM_{10}$  and  $PM_{2.5}$  is also not very scientific. When the health effect is mainly related to  $PM_{2.5}$  this conversion factor may be relevant, but if coarse particles are as important as fine, this down-scaling of effects is not motivated. According to the literature we can assume that the impact on mortality of anthropogenic  $PM_{10}$  and  $PM_{2.5}$  respectively would be of similar size, while for respiratory morbidity the contribution of the coarse fraction may be greater. However, our presented impact estimates are products of the selected cut off levels and ER-functions, and do not fully reflect statements on impacts related to comparisons of  $PM_{10}$  and  $PM_{2.5}$ .

Our assessment of health impacts using  $PM_{10}$  or  $PM_{2.5}$  as exposure indicators is most valid for the contribution from the regional background particle pollution. Even if the exhaust particles contribute much to the health impacts in cities, it is likely that  $NO_2$  or  $NO_X$  is a better indicator of the local-regional gradients in vehicle exhaust than particle mass as  $PM_{10}$ , for which exhaust particles play a minor role. We thus see our previous assessment using  $NO_2$  as a better indication of the size of the mortality effects from traffic in Sweden, than the estimates for exhaust PM and road dust PM in this assessment. In our previous report we estimated that more than 3 200 deaths per year are brought forward due to such exposure, indicated by modelled nitrogen dioxide levels at home above a cut off at 10  $\mu$ g/m<sup>3</sup> as an annual mean. In order to see the total air pollution impact, it is probably justified to add almost all of the 3 240 excess deaths per year that we attribute to  $PM_{10}$  exposure due to the regional background, wood smoke and the non-specified other sources in this study to the estimated deaths per year attributed to nitrogen dioxide levels in our previous report. Likewise, effects of ozone could be added.

The estimated respiratory and cardiovascular hospital admissions due to the short-term effects of  $PM_{10}$  may seem to be low in comparison with the estimated number of deaths, new chronic bronchitis cases and restricted activity days. However, for hospital admissions we can only estimate the short-term effect on admissions, not the whole effect on hospital admissions following morbidity due to PM.

The health effects related to high concentrations of PM in ambient air are related to socioeconomic costs, as are the costs for abating these high concentrations. It is important for decision makers to use their economic resources in an efficient manner, which furthermore induces the need for assessment of what can be considered as an efficient use of resources. The socio-economic costs related to high levels of PM in air are derived from the cost estimates of resources required for treatment of affected persons, productivity losses from work absence and most prominently from studies on the social willingness to pay for the prevention of health effects related to these high levels of PM.

In our study we have applied results from international socio-economic valuation studies to our calculated results of increased occurrences of hospital admissions and fatalities. The values from the studies have been adapted to Swedish conditions. The application of international results favours comparison with other estimates of economic valuation of health effects related to high levels of PM.

The results suggest that the health effects related to high annual mean levels of PM can be valued to annual socio-economic costs (welfare losses) of ~26 billion Swedish crowns during 2005. Approximately 1.4 of these 26 billion Swedish crowns consist of productivity losses for society. Furthermore, the amount of working and studying days lost constitutes some ~0.1% of the total amount of working and studying days in Sweden during 2005.

A large part of the population is exposed to medium levels of PM. Thus, the highest costs to society are to be found in those regions. Further, most of the costs come from exposure to  $PM_{2.5}$ . This displacement in the distribution of the social costs indicates that a cost effective abatement

strategy for Sweden might be to reduce the medium, rather than the highest, annual levels of PM. Attention should preferably be paid to abatement measures with high abatement potential for  $PM_{2.5}$ .

The socio-economic benefits from introducing maximum limit values of 20  $\mu$ g/m<sup>3</sup> for PM<sub>2.5</sub> would equal a little more than 7 billion SEK<sub>2005</sub> (~1000 avoided fatalities). The introduction of even lower maximum limit values would result in correspondingly higher socio-economic benefits; ~15 billion (~2000 avoided fatalities) for max 15  $\mu$ g/m<sup>3</sup> and ~21 billion (~3000 avoided fatalities) for max of 10  $\mu$ g/m<sup>3</sup>.

Comparison between the calculated  $PM_{10}$  concentrations and monitoring data in urban background show good agreement. Long range transport is the dominating source of particles observed in Sweden. Since it is difficult to estimate this contribution it generally leads to a large uncertainty in particle modelling.

In the 1x1 km grid resolution (also used in the URBAN model) the small scale emission patterns, such as roads, are usually not detectable. Comparison between this approach and modelling with a higher spatial resolution however shows similar results for population exposure of the yearly  $PM_{10}$  means, possibly because not many people live next to roads. The method that uses the URBAN model in combination with a GIS based geographical distribution is thus proved to be accurate enough for calculating the PM exposure on a national level. Future development of the modelling methodology should concentrate on incorporating an improved spatial pattern of emissions. It might also be possible to use concentration maps that are available for larger cities, and to apply the dispersion pattern to the URBAN model.

Another uncertainty is the attempt to separate between different sources for  $PM_{10}$ , where the allocation of the contribution from road dust was shown to be one of the largest difficulties. The multivariate approach used could be further improved by applying weighting factors and/or by including more parameters.

The  $PM_{2.5}$  concentrations were roughly calculated by using the relation to levels of  $PM_{10}$  on a yearly basis. Additional monitoring data for  $PM_{2.5}$  would probably result in a considerable improvement in the estimation of the exposure situation.

# Sammanfattning

Knappt 10% av Sveriges befolkning utsätts för halter av  $PM_{10}$  (partiklar mindre än 10 µm) högre än 20 µg/m<sup>3</sup> i den allmänna utomhusluften. Denna halt motsvarar miljömålet för år 2010, men nivån skall även klaras i mer belastade områden såsom gaturum. För mindre partiklar (PM<sub>2.5</sub>, partiklar mindre än 2.5 µm) visar motsvarande jämförelse med miljömålet (12 µg/m<sup>3</sup> för år 2010) på att drygt 20% av landets invånare exponeras för halter över denna nivå.

Med en nedre gräns vid 5  $\mu$ g/m<sup>3</sup> för effekter tillskrivna årsmedelhalten av PM<sub>10</sub> (motsvarar ungefär att undanta det naturliga bidraget) och antaget källspecifika ER-funktioner, skattar vi ungefär 3 400 förtida dödsfall per år. Med beräknad exponering för PM<sub>2.5</sub> hamnar hälsoskattningarna totalt sett något lägre, cirka 3 100 prematura dödsfall per år.

Kostnaden för samhället orsakade av hälsoeffekter relaterade till höga halter av PM värderas till  $\sim$ 26 miljarder svenska kronor per år. Dessa extra kostnader för samhället orsakas av de  $\sim$ 3 400 dödsfallen,  $\sim$ 1 300 – 1 400 fall av kronisk bronkit,  $\sim$ 1 400 sjukhusinläggningar för andnings- och hjärtbesvär samt  $\sim$ 4,5 - 5 miljoner persondagar under vilka normala aktiviteter inte kan genomföras för de drabbade. Den sistnämnda hälsoeffekten orsakar dessutom arbetsbortfall motsvarande strax över 0,1 % av den totala mängden arbetade dagar i Sverige.

I en tidigare studie med avseende på NO<sub>2</sub> har beräknats att drygt 3 200 förtida dödsfall per år beror på lokalt genererade avgaser. För att få fram den totala effekten av luftföroreningar på dödligheten är det sannolikt motiverat att addera fallen som här tillskrivs partiklar från andra källor än lokal trafik (3 240 förtida dödsfall), fall som associerats med NO<sub>2</sub> samt fall tillskrivna ozon.

Haltnivåerna av partiklar (PM) i omgivningsluften har fortfarande en betydande hälsopåverkan, trots att det under de senaste årtiondena har införts ett flertal åtgärder för att minska utsläppen. Miljökvalitetsnormerna för utomhusluft överskrids på många håll, och i en studie som presenterades för några år sedan uppskattades att höga partikelhalter orsakar mer än 5 000 förtida dödsfall i Sverige per år.

På uppdrag av Naturvårdsverket har IVL Svenska Miljöinstitutet och Institutionen för folkhälsa och klinisk medicin vid Umeå universitet kvantifierat den svenska befolkningens exponering för halter i luft av PM<sub>2.5</sub> och PM<sub>10</sub> för år 2005, beräknat som årsmedelkoncentrationer. Även de samhällsekonomiska konsekvenserna av de uppskattade hälsoeffekterna har beräknats.

Angivna miljökvalitetsnormer och miljömål skall klaras överallt, även i de mest belastade gaturummen. För exponeringsberäkningar är det dock mest relevant att använda urbana bakgrundshalter, som även tillgängliga exponerings/respons-samband baseras på. Resultaten visar att den urbana bakgrundshalten av  $PM_{10}$  i merparten av landet var relativt låg i förhållande till miljökvalitetsnormen för årsmedelvärde (40 µg/m<sup>3</sup>). I vissa områden, huvudsakligen i södra Sverige, var haltnivåerna i samma storleksordning som miljömålet (20 µg/m<sup>3</sup> som årsmedelvärde) för år 2010. Merparten av befolkningen, 90 %, exponerades för årsmedelhalter av  $PM_{10}$  lägre än 20 µg/m<sup>3</sup>. Mindre än 1% av landets invånare utsattes för exponeringsnivåer av  $PM_{10}$  över 25 µg/m<sup>3</sup>.

Beträffande PM<sub>2.5</sub> var den urbana bakgrundskoncentrationen år 2005 i samma storleksordning som miljömålet (12 µg/m<sup>3</sup> som årsmedelvärde för år 2010) i en stor del av landet. Ungefär hälften av befolkningen exponerades för årsmedelhalter av PM<sub>2.5</sub> längre än 10 µg/m<sup>3</sup>, medan knappt 2% utsattes för halter över 15 µg/m<sup>3</sup>.

Eftersom forskningsresultat tyder på att de relativa riskfaktorerna för hälsoeffekter är högre för förbränningsrelaterade partiklar än för partiklar från andra källor så separerades också den totala PM<sub>10</sub>-halten på olika källbidrag med hjälp av en multivariat analysmetod.

Hälsokonsekvensberäkningar bygger på samband, s.k. exponerings-responsfunktioner (ERF) från epidemiologiska studier, vilka appliceras på beräknad exponering och typisk frekvens av fall i befolkningen. Beräkningarna utformas ofta så att man uppskattar antal fall som tillskrivs en viss exponering eller exponering över en viss nivå. För PM<sub>10</sub> har vi uppskattat att årsmedelvärdet för den regionala bakgrundshalten, som till avsevärd är del "naturlig", typiskt ligger på cirka 5 µg/m<sup>3</sup>, och vi har därför använt 5 µg/m<sup>3</sup> som en undre gräns för konsekvensberäkningarna. Följaktligen skattar vi antalet fall som kan tillskrivas exponering utöver denna bakgrund. För PM<sub>2.5</sub> har motsvarande avgränsning gjorts vid 4 µg/m<sup>3</sup> utifrån den ungefärliga kvoten PM<sub>2.5</sub>/PM<sub>10</sub>.

Inom forskarvärlden fokuserar luftföroreningsforskningen till stor del på olika typer av partiklar och deras förmodade olika hälsoeffekter relaterade till partiklarnas storlek och egenskaper. Ännu finns dock ingen konsensus om hur olika partikeltyper kan tilldelas olika riskkoefficienter vid konsekvensberäkningar. Vanligt är ändå att när mortalitetseffekter beräknas för PM<sub>10</sub> så används exponerings-responsfunktioner framtagna med PM<sub>2.5</sub> för en kvotbaserad reduktion till en riskkoefficient för PM<sub>10</sub>.

Långtidseffekten på dödligheten beskriven utifrån PM<sub>2.5</sub> i en stor amerikansk kohortstudie (ACS) har ofta använts även i europeiska konsekvensberäkningar som EU-programmet Clean Air For Europé (CAFE). Koefficienten var 6 % per 10  $\mu$ g/m<sup>3</sup> ökning av långtidshalten av PM<sub>2.5</sub>. För PM<sub>10</sub> har den justerade koefficienten 4.3 % vanligtvis använts, exempelvis i det europeiska APHEIS-projektet.

Studier från senare år har dock visat att gradienterna i halter inom en stad tycks ge högre relativ risk per halt än studierna som bygger på jämförelser mellan städer. I en studie enbart inom Los Angeles med data från samma kohort (ACS) blev den relativa risken per  $10 \ \mu g/m^3 PM_{2.5}$  hela 17 %, eller cirka 3 gånger högre än i huvudstudien som jämförde mortaliteten mellan deltagare från olika städer karaktäriserade av en "stadens medelhalt". För grova partiklar har man inte funnit någon säkerställd effekt på dödligheten kopplad till långtidshalterna, och studierna av korttidshalterna har givit varierande resultat för grovfraktionen (PM<sub>10-2.5</sub>).

Trots det faktum att man vanligtvis, som i CAFE, antar att allt PM oavsett källa har samma effekt, har i denna analys använts en mindre konservativ ansats och antagits att avgas- och förbränningspartiklar har en högre effekt på mortaliteten än den typiskt antagna, att vägdamm har en lägre effekt och att sekundära partiklar har den typiskt antagna effekten.

För primära partiklar har vi i denna studie använt exponerings-responssambandet 17 % ökad dödlighet per 10  $\mu$ g/m<sup>3</sup>. För PM<sub>10</sub> i form av vägdamm har vi antagit enbart en korttidseffekt på mortaliteten med samma storlek som för PM<sub>10</sub> i allmänhet. Baserat på den europeiska multicenterstudien APHEA2 har vi valt att använda 1 % ökning av total dödlighet per 10  $\mu$ g/m<sup>3</sup>. För PM<sub>10</sub> i övrigt har vi valt den justerade ERF på 4.3 % per 10  $\mu$ g/m<sup>3</sup> som baseras på amerikanska resultat erhållna med PM<sub>2.5</sub>, och som tidigare används av bl.a. det europeiska APHEIS-projektet. För PM<sub>2.5</sub> har vi inte beräknat bidraget från olika källor och använder resultatet från ACS på 6 % ökad dödlighet per 10  $\mu$ g/m<sup>3</sup> som gjordes i CAFE.

Beträffande mortalitet har vi i denna studie inkluderat bara några av de potentiellt tillgängliga effekterna. Vi beslutade att inkludera bara några viktiga och vanligt använda hälsoutfall som medger

jämförelser med andra hälsokonsekvensberäkningar och hälsoekonomiska beräkningar. Frågan huruvida ER-samband för sjuklighet som framtagits med PM<sub>10</sub> ska justeras uppåt i beräkningen baserad på halter av PM<sub>2.5</sub> är inte enkel. Vi beslöt att göra så för sjukdagar (restricted activity days) men inte för akuta inläggningar på sjukhus respektive uppkomst av kronisk bronkit, eftersom det där saknas tillräcklig grund för en justering.

För att beräkna hur många dödsfall och sjukhusinläggningar som beror på exponering över vissa nivåer, behöver man också använda en grundfrekvens av fall. I våra beräkningar för NO<sub>2</sub> (Sjöberg et al, 2007), tillämpade vi officiella nationella tal, för dödlighet 2002 års frekvens och för sjukhusinläggningar frekvenser för 2004. Eftersom denna typ av tal förändras långsamt, och för jämförbarhetens skull, använde vi samma grundfrekvenser i den tidigare beräkningen med NO<sub>2</sub>.

Med en nedre gräns vid 5  $\mu$ g/m<sup>3</sup> för effekter tillskrivna årsmedelhalten av PM<sub>10</sub> (motsvarar ungefär att undanta det naturliga bidraget) och antaget källspecifika ER-funktioner, skattar vi ungefär 3 400 förtida dödsfall per år. Sammantaget med 1 300 - 1 400 nya fall av kronisk bronkit, ungefär 1 400 sjukhusinläggningar och omkring 4.5-5 miljoner sjukdagar, blir samhällskostnaderna för hälsokonsekvenserna ungefär 26 miljarder kronor per år. Med beräknad exponering för PM<sub>2.5</sub> hamnar hälsoskattningarna totalt sett något lägre, cirka 3 100 prematura dödsfall per år. De nedre haltgränser som används vid beräkning av hälsokonsekvenser i denna studie är dock ganska godtyckligt antagna, eftersom vi inte mera säkert känner den naturliga bakgrunden eller ER-kurvans form i olika koncentrationsintervall.

Denna konsekvensberäkning utifrån beräknade halter av PM<sub>10</sub> och PM<sub>2.5</sub> som exponeringsmått bör resultera i de mest tillförlitliga mortalitetsskattningarna för bidraget som har mindre lokal karaktär, eftersom det var skillnader mellan städers urbana bakgrundsstationer som användes i ACS-studien. Även om lokalt emitterade avgaspartiklar bidrar mycket till hälsokonsekvenserna i städerna, så beräknas konsekvenserna av det lokala avgasbidraget sannolikt mycket bättre utifrån de resultat som erhållits utifrån gradienter i halten av kväveoxider inom städer, än med samband utifrån skillnader i PM-halter mellan städer, för vilka avgaspartiklar har mindre betydelse. Vi anser därför att våra tidigare beräkningar med NO<sub>2</sub> som indikator ger bättre skattningar av effekterna på mortaliteten på grund av trafikavgaser, än de mindre effekter för avgaspartiklar och vägdamm som här skattats. Vi har tidigare beräknat att drygt 3 200 förtida dödsfall per år beror på lokalt genererade avgaser. För att få fram den totala effekten av luftföroreningar på dödligheten är det sannolikt motiverat att addera fallen som här tillskrivs partiklar från andra källor än lokal trafik, fall som associerats med NO<sub>2</sub> samt fall tillskrivna ozon.

Antalet akuta inläggningar på sjukhus som beräknas på grund av exponeringen kan förefalla få jämfört med antal dödsfall, fall av kronisk bronkit och antal sjukdagar. Detta beror dock på att det bara är korttidseffekterna av föroreningarna på antal inläggningar som beräknas, inte hur mycket partikelhalterna ökar antalet inläggningar totalt sett.

Både hälsoeffekter, orsakade av höga halter av luftföroreningar, och åtgärder för att minska dessa halter är oundvikligen kopplade till samhällskostnader. Eftersom det är viktigt för beslutsfattare att använda skattepengar och andra finansiella resurser på mest effektiva sätt blir det även viktigt att göra ordentliga bedömningar av vad som är att räkna som effektivt användande av resurser. Till detta hör en bedömning om värdet för samhället att slippa hälsoeffekter orsakade av höga halter av luftföroreningar. I den ekonomiska delen av denna rapport har genomförts en ekonomisk värdering av de hälsoeffekter som hänger ihop med höga halter av PM i luft.

Internationellt har det skett mycket arbete kring värdering av hälsoeffekter och vi har i denna studie valt att använda de värderingar som skett i tidigare internationella studier som grund för värdering av svenska samhällskostnader kopplade till höga halter av PM. Detta gynnar jämförelse med andra resultat inom området kring ekonomisk värdering av hälsoeffekter.

Resultaten från vår studie visar att negativa hälsoeffekter relaterade till höga nivåer av PM kan värderas till årliga samhällsekonomiska kostnader (välfärdsförluster) på ~26 miljarder svenska kronor under 2005. Ungefär 1,4 av dessa 26 miljarder utgörs av produktivitetsförluster i samhället. Detta motsvarar en förlust i antalet arbets- och studiedagar motsvarande lite mer än 0,1 % av den totala mängden arbets- och studiedagar under 2005.

En stor andel av befolkningen exponeras för medelhöga haltnivåer av PM, vilket medför att de högsta kostnader för samhället återfinns för områden. Dessa kostnader härrör främst från exponering för PM<sub>2.5</sub>.Denna fördelning av samhällskostnader indikerar att kostnadseffektiva åtgärdsstrategier i Sverige kan utgöras av åtgärder riktade mot medelhöga, snarare än de högsta, haltnivåerna. Uppmärksamhet bör främst ägnas åt åtgärder med stor potential att minska haltnivåerna av PM<sub>2.5</sub>.

Den samhällsekonomiska nyttan av att introducera maximala gränsvärden för PM<sub>2.5</sub> motsvarande max 20  $\mu$ g/m<sup>3</sup> skulle resultera i en samhällsekonomisk nytta motsvarande ca 7 miljarder svenska kronor (2005 års värde) (~1 000 dödsfall undvikta). Om man skulle sätta gränsvärdena lägre så skulle detta resultera i ännu högre nytta för samhället, ca 15 miljarder (~2 000 dödsfall undvikta) i samhällsekonomisk nytta skulle nås om gränsvärdet sattes till max 15  $\mu$ g/m<sup>3</sup>, och ca 21 miljarder (~3 000 dödsfall undvikta) skulle nås om gränsvärdet sattes till max 10  $\mu$ g/m<sup>3</sup>.

En jämförelse mellan de beräknade PM<sub>10</sub>-koncentrationerna och mätdata i urban bakgrundsluft visar på en bra överensstämmelse. Den dominerande källan till förekommande haltnivåer av partiklar i Sverige är långdistanstransporten, framför allt från källområden på den europeiska kontinenten. De stora osäkerheter som idag finns vid all partikelmodellering beror till stor del på att det är svårt att uppskatta detta regionala bakgrundsbidrag.

Med en grid-storlek på 1x1 km (som i URBAN-modellen) återspeglas vanligtvis inte det småskaliga emissionsmönstret, så som vägar. En jämförelse mellan det här presenterade angreppssättet och modellering med en högre geografisk upplösning visar trots detta på jämförbara resultat för befolkningsexponeringen med avseende på årsmedelvärden för PM<sub>10</sub>. Detta beror troligen på att andelen personer som bor i direkt anslutning till vägar är relativt begränsad. Metoden att använda URBAN-modellen i kombination med GIS-baserad geografisk fördelning, för såväl haltuppskattning som befolkningsfördelning, har därmed visats ge tillfredsställande resultat för kvantifiering av partikelexponering på nationell skala. För att modellen bättre skall kunna spegla situationen även i mer lokal skala skulle man kunna förbättra beskrivningen av det geografiska emissionsmönstret, exempelvis genom att i URBAN-modellen inkludera resultat från mer detaljerade spridningsberäkningar för områden där detta finns tillgängligt.

Ytterligare en osäkerhet ligger i fördelningen av  $PM_{10}$  på olika källbidrag, där allokeringen av uppvirvlat vägdamm visades vara en av de stora svårigheterna. Det multivariata angreppssättet bör kunna förbättras genom att applicera olika viktning på ingående parametrar och/eller inkludera fler parametrar.

Halterna av  $PM_{2.5}$  beräknades utifrån relationen till förekommande haltnivåer av  $PM_{10}$  på årsbasis. Tillgång till ytterligare mätdata för  $PM_{2.5}$  skulle sannolikt kunna förbättra uppskattningen av exponeringssituationen avsevärt.

### Contents

Sı	ımmary	2
1	Introduction	11
2	Background and aims	11
3	Methods	12
	3.1 PM <sub>10</sub> concentration calculations	13
	3.1.1 Regional background	13
	3.1.2 Urban background	14
	3.1.2.1 Population distribution	15
	3.1.3 Separation of particle source contributions	15
	3.1.3.1 Small scale domestic heating	15
	3.1.3.2 Traffic induced particles	17
	3.1.3.3 Dispersion parameters	19
	3.1.3.4 Multivariate data analysis	21
	3.2 PM <sub>2.5</sub> concentration calculations	23
	3.3 Health impact assessment (HIA)	24
	3.3.1 Exposure-response function (ERF) for mortality	25
	3.3.1.1 Selected exposure-response functions	27
	3.3.2 Exposure-response function for morbidity	28
	3.3.2.1 ERF for hospital admissions	28
	3.3.2.2 ERF for chronic bronchitis	29
	3.3.2.3 ERF for restricted activity days	29
	3.3.3 Selected baseline rates for mortality and admissions	29
	3.4 Socio-economic valuation	30
	3.4.1 Quantified results from the literature	30
4	Results	34
	4.1 Calculation of PM concentrations	34
	4.1.1 National distribution of PM <sub>10</sub> concentrations	34
	4.1.2 Separation of PM <sub>10</sub> sources	35
	4.1.3 National distribution of PM <sub>2.5</sub> concentrations	41
	4.2 Population exposure	42
	4.2.1 Exposure to $PM_{10}$	42
	4.2.2 Exposure to PM <sub>2.5</sub>	45
	4.3 Estimated health impacts	46
	4.3.1 Mortality	46
	4.3.2 Morbidity effects	48
	4.4 Socio-economic cost	51
	4.4.1 Results of socio-economic valuation	51
	4.4.2 Sensitivity Analysis	53
	4.5 Consequence analysis of reduced PM <sub>2.5</sub> concentrations	55
_	4.6 Model evaluation	57
5	Discussion	60
6	Reterences	64

# 1 Introduction

The concentrations of particulate matter (PM) in ambient air still have significant impact on human health, even though a number of measures to reduce the emissions have been implemented during recent decades (Sjöberg et al., 2007; Miljömålsrådet, 2008; Persson et al, 2007). The air quality standards are exceeded in many areas, and a recent study estimated that more than 5 000 premature deaths in Sweden per year were due to PM exposure (Forsberg et al., 2005b).

On behalf of the Swedish Environmental Protection Agency, IVL Swedish Environmental Research Institute and the Department of Public Health and Clinical Medicine at Umeå University have quantified the population exposure to annual mean concentrations of  $PM_{10}$  and  $PM_{2.5}$  in ambient air for the year 2005. Based on these results the health and associated economic consequences have also been calculated.

# 2 Background and aims

The highest concentrations of nitrogen dioxide (NO<sub>2</sub>) and PM in a city are normally found in street canyons. However, for studies of population exposure to air pollution it is customary to use the urban background air concentrations, since these data are used in dose-response relationship studies and health consequence calculations.

 $NO_2$  has been monitored on a regular basis for a long time in Sweden, and the number of people exposed to ambient air concentrations of  $NO_2$  in excess of the air quality standards have been investigated earlier (Sjöberg et al, 2007). Measurements of  $PM_{10}$  have been carried out for less than 10 years. The available data on  $PM_{2.5}$  in urban areas is even more limited. No exposure studies for PM have been performed on a national basis. However, in an assessment of the health impact of particulate air pollutants Forsberg et al (2005b) estimated more than 5 000 premature deaths on a national basis.

Exposure studies using dispersion models to simulate the  $PM_{10}$  concentrations on an urban scale have been performed in various cities in the world, such as Lissabo (Borrego et al. 2006), Oslo (Oftedal et al. 2008) and in a smaller scale of a few blocks in Vancouver (Ainsliea et al. 2007). The method to calculate human exposure using both a simplified Stochastic (regression) and a Gaussian model in combination with a GIS based system have also been used by Cyrys et al. (2005). Particle exposure due to local emissions and the related external costs haves also been quantified for the Stockholm area (Johansson & Eneroth, 2007).

Ambient concentrations of air pollutants show strong variability at a fine scale (1x1 km or even less) due, for example, to local meteorological conditions. These variations are difficult to reflect using dispersion models on a national basis, due to scaling problems both according to emission inventories and type of models.

Urban background air pollution levels related to health effects have been studied for more than 20 years in about one third of the small to medium sized towns in Sweden.  $PM_{10}$  has been included in the monitoring program since the year 2000. The monitoring is undertaken within the framework of the urban air quality network, a co-operation between local authorities and IVL Swedish Environmental Research Institute (Persson et al., 2007). An empirical statistical model for air quality assessment, the so-called URBAN model, was developed based on the monitoring data, as a

screening method to estimate urban air pollution levels in Sweden (Persson et al., 1999; Persson and Haeger-Eugensson, 2001). It has since been further improved, by applying local meteorological parameters, to be used for quantification of the general population exposure to ambient air pollutants on a national level (Haeger-Eugensson et al., 2002; Sjöberg et al., 2004).

The possibility to perform health impact assessments based on the calculated exposure to air pollutants and exposure-response functions for health effects, has also been previously demonstrated (Forsberg and Sjöberg, 2005a; Forsberg et al., 2005b; Sjöberg et al, 2007).

The purpose of this study has been to calculate the excess exposure to yearly mean concentrations of  $PM_{10}$  (total as well as different source contributions) and  $PM_{2.5}$  on a national scale and to assess the associated long-term health impact as well as the related economic consequences.

# 3 Methods

The method applied for calculation of ambient air concentrations and exposure to air pollutants has been described earlier (Sjöberg et al., 2007). The empirical statistical URBAN model is used as a basis. Urban background monitoring data and a local ventilation index (calculated from mixing height and wind speed) are required as input information for calculating the air pollution levels.

The concentration pattern of  $PM_{10}$  over Sweden was calculated with a 1x1 km grid resolution by using the model, based on the relationship  $NO_2/PM_{10}$  in urban background air for the year 2005 (see further Chapter 3.1.2). This kind of approach has earlier been applied by e.g. Muri (1998). However, the relationship between the two parameters in that study was not applicable for Swedish conditions since it was assumed to be site dependent. To reflect the seasonal variation in the particle load the calculated yearly means were based on concentrations calculated with a resolution of 2 months.

The concentration distribution in urban background air within cities was estimated assuming a decreasing gradient towards the regional background areas. The calculated  $PM_{10}$  levels are valid for the similar height above ground level as the input data (4-8 m) in order to describe the relevant concentrations for exposure.

The calculation of PM<sub>2.5</sub> concentrations was based on a defined ratio to PM<sub>10</sub> in different types of areas; central urban, suburban and regional background.

The quantification of the population exposure to  $PM_{10}$  (estimated as the annual mean of total  $PM_{10}$  as well as separated for different source contributions) and  $PM_{2.5}$  (annual mean) was based on a comparison between the pollution concentration and the population density. Population density data was used with a grid resolution of 1\*1 km. By over-laying the population grid to the air pollution grid the population exposure to a specific pollutant is estimated for each grid.

To estimate the health consequences, exposure-response functions for the long-term health effects were used, together with the calculated PM exposure. For calculation of socio-economic costs, results from economic valuation studies and other cost calculations were used. These cost estimates were combined with the estimated quantity of health consequences performed in this study to give the total social cost of high levels of PM in ambient air.

### **3.1 PM**<sub>10</sub> concentration calculations

The  $PM_{10}$  concentrations were calculated based on i) regional background levels, and ii) local source contributions to the urban background concentrations. For each urban area the contribution from the regional background  $PM_{10}$  concentration was calculated, and subtracted from the urban  $PM_{10}$  concentration to avoid double counting.

### 3.1.1 Regional background

Monitoring of  $PM_{10}$  in regional background air is carried out at three sites in Sweden, within the national environmental monitoring programme financed by the Swedish Environmental Protection Agency (hosted by www.ivl.se). The basis for calculating a reasonable realistic geographical distribution of  $PM_{10}$  concentrations over Sweden is thus limited. Therefore, calculated distribution patterns by the mesoscale dispersion model EMEP on a yearly basis were used, in combination with the existing monitoring data (Figure 1) (EMEP, 2005).

To separate the regional and local  $PM_{10}$  contributions it was necessary to divide the regional background concentrations into two-month means. This was done by using data for the three monitoring sites, and applying similar conditions between the annual and monthly distribution of the calculated  $PM_{10}$  concentrations from the EMEP model.



**Figure 1** Regional background concentrations of PM<sub>10</sub> in Sweden (the EMEP model in combination with monitoring data).

### 3.1.2 Urban background

Two-month means were calculated for the urban areas where data were available for both  $PM_{10}$  and  $NO_2$  for the years 2000-2005. The regional estimated background concentrations of  $NO_2$  and  $PM_{10}$  were subtracted, and seasonal ratios of  $PM_{10}/NO_2$  for the remaining local contribution were derived and analysed with respect to the latitude, see Figure 2. Thus, different equations for each season were derived for the graphs presented in Figure 2. It was not statistically relevant to calculate a standard deviation of the ratios for each season since there were not enough data. The maximum and minimum spreads of the ratios for each season, presented in Appendix A, were rather small during the winter season (Nov-Dec, Jan-Feb) at all latitudes. However, the variability increased, especially in southern Sweden during spring, summer and autumn.



**Figure 2** Latitudinal and seasonal variation of the functions based on the locally developed ratios (PM<sub>10</sub>/NO<sub>2</sub>) in urban background air.

According to the calculated functions of the ratio  $(PM_{10}/NO_2)$  there are large seasonal differences both in the northern and southern part of Sweden. For the southern part the largest difference was found in May-June and the smallest in January-February. In the north the differences were very small compared to the situation in the south.

The earlier calculated NO<sub>2</sub> concentrations (Sjöberg et al., 2007) underlie the calculated functions for estimation of two-month means of  $PM_{10}$  in the 1890 most densely population areas in Sweden. Consequently, monitoring data are replaced by calculated urban background concentrations in towns where measurements take place. The derived functions were further used for the calculations of annual mean  $PM_{10}$  concentration in ambient urban background air. Due to a limited number of data in July–August, the function for May-June was also applied for those months.

When comparing the national annual means of calculated and monitored urban background concentrations of  $PM_{10}$  it becomes clear that the calculated concentrations are overestimated by about 10%. Further, the overestimation is larger in southern Sweden (about +15%) and in the northern Sweden there is an underestimation (about -15%). In the area around Stockholm the calculations are very accurate ( $\pm 2$ %). The reason for this non-linear "error" is assumed to arise from the interpolation of the regional background concentrations. Since the urban background concentration constitutes between 50-70% of regional background concentration an error in this calculation shows a reasonably good agreement between measured and calculated urban background concentrations.

### 3.1.2.1 Population distribution

The  $PM_{10}$  concentration distribution methodology in urban areas is dependent on the size of the urban area. The size of the urban area is calculated from diameter information gathered from Statistics Sweden (www.scb.se) from 80 towns in Sweden. It was found that there was a strong relationship between the diameter and the number of inhabitants (Sjöberg et. al., 2007). The urban areas were divided into 4 different groups dependent on number of inhabitants; 200 - 2500 inhabitants, 2500 - 5000 inhabitants, 5000 - 10000 inhabitants and >10000 inhabitants.

The current population data applied for exposure calculations in this study are derived from EEA (European Environment Agency) and was produced by JRC (the Joint Research Centre). The method applied by JRC to disaggregate the population statistics at 100 x 100 m is found in Gallego and Peedell (2001). The EEA population density grid is based on 2001 data, and in total, 8,899,724 inhabitants were recorded within the Swedish borders. The 100 x 100 m grid was aggregated into 1 x 1 km grid resolution.

### 3.1.3 Separation of particle source contributions

Since it is assumed that the relative risk factors for health impact are higher for combustion related particles (WHO, 2007; see further Chapter 3.3.1) the total  $PM_{10}$  concentration was also separated into different source contributions by using a multivariate method (se further Chapter 3.1.3.4).

### 3.1.3.1 Small scale domestic heating

In order to evaluate the proportion of  $PM_{10}$  from small scale domestic heating (wood fuel burning exclusively) the statistics of domestic energy consumption on municipality level in 2003, further divided into consumption of wood fuel, were used (SCB, 2007). Figure 3 - Figure 4 present the distribution of energy consumption on a county level. The proportion is governed by the air temperature and the supply of wood.

The energy consumption from wood burning for each of the 1 890 densely built-up areas in Sweden were drawn from the information presented in Figure 5.



Figure 3 Percentage of total energy consumption from wood (red bars) and biomass (blue bars) per county in 2003.



Figure 4 Energy consumption from wood burning (GWh)/inhabitant, county.



Figure 5 Energy consumption from wood burning (GWh)/inhabitant in each municipality in Sweden.

The outdoor air temperature is also an important parameter governing the use of wood for domestic heating. A method for describing the requirement of indoor heating is to calculate an energy index (I<sub>e</sub>). The index is based on the principle that the indoor heating system should heat up the building to  $+17^{\circ}$ C, while the remaining part is generated by radiation from the sun and passive heating from people and electrical equipment. The calculation of I<sub>e</sub> is thus the difference between  $+17 \,^{\circ}$ C and the outdoor air temperature. For example, if the outdoor temperature is  $-5^{\circ}$ C the I<sub>e</sub> will be 22. During spring, summer and autumn the requirement of indoor heating is less than wintertime (November – March). Thus, during those months, the outdoor temperature is calculated

with a baseline specified in Table 1. The energy index calculations are based on monitored (by SMHI) outdoor temperature as means for 30 years at 535 sites located all over Sweden and result in monthly national distribution of the energy indices, see Figure 6.

Table 1The base line for the outdoor temperature for calculation of Ie during April - October.

Months	Baseline outdoor temperature (°C)
April	+ 12
May-July	+ 10
August	+ 11
September	+ 12
October	+ 13



Figure 6 The calculated energy index (I<sub>e</sub>) for Sweden i January, April, July, October.

Based on these interpolated maps, two-month means of  $I_e$  were extracted for each of the 1 890 towns in Sweden. These results were used to determine the contribution to the  $PM_{10}$  concentration from wood burning to the energy consumption per inhabitant in each town.

### 3.1.3.2 Traffic induced particles

Traffic contributes to the total concentration of  $PM_{10}$  both directly through exhaust emissions from vehicles and secondarily through re-suspended dust from roads. Traffic related particle concentrations are associated with the NO<sub>2</sub> concentration in urban areas, why the earlier calculated NO<sub>2</sub> concentrations for all densely built-up areas (Sjöberg et al., 2007) were used in the multivariate analysis to determine this source. However, since road dust arises mainly from wear of the road surface (i.e. due to use of studded tyres) as well as from brakes and tyres, a valuation of the use of studded tyres was also included as a parameter (see below) analysed with the multivariate method.

The largest contribution from resuspension mainly occurs during late winter and spring as a result of the drying up of the road surfaces. The accumulated road dust goes into suspension in the air, as a result of traffic induced turbulence as well as wind. Suspension of dust and soil from non-vegetated land surfaces also occurs in springtime when soil surfaces dries up and before vegetation season starts, mainly in the southern part of Sweden.

One parameter that regulates the amount of road dust is proved to be the number of cars using studded tyres (Gustafsson et al., 2005). Unfortunately, there are no such information available with a monthly resolution. However, in Malmö in the south of Sweden the number of cars using studded tyres at parking lots in the region has been manually calculated during January, February, March and April 2005 (Sjöberg and Ferm, 2005). The Swedish Tyre Industry Information Board (Däckbranchens informationsråd, 2008) supply annual information on the number of cars with studded tyres in February (Figure 7) in the seven different road administration regions (Figure 8). The national data have been combined with the information from Malmö and the regional scale meteorological conditions, in order to derive a monthly based usage of studded tyres were calculated for each of the 1 890 densely built-up areas in Sweden to be further used in the multivariate analysis.



**Figure 7** The usage of different types of tyres in February within the seven road administration regions in Sweden (visualized in Figure 8).



Figure 8 The seven road administration regions of Sweden (www.vv.se).



**Figure 9** The monthly distribution, in percent, of the usage of studded tyres in the seven road administration regions.

### 3.1.3.3 Dispersion parameters

Meteorology also influences the air pollution concentrations. This can be defined in many ways, but a so called mixing index  $(V_i)$  has been shown to capture both local (such as topographical and coastal effects) and regional variations (such as location of high/low pressures).  $V_i$  is determined by multiplying the mixing height and the wind speed.  $V_i$ 's have been calculated for the whole of Sweden by using an advanced meteorological dispersion model, TAPM (see further Haeger-Eugensson et. al. 2002). In Figure 10 the mean values of  $V_i$  have been calculated in groups of every 1000 steps of the local coordinates.



**Figure 10** Two-month means of V<sub>i</sub> calculated in groups of every 1000 steps of the local coordinates (from south to north) in all towns in Sweden.

According to results presented in Chen et. al (2000) the calculation of the mixing height and wind speed by the TAPM model is well in accordance with measurements. During winter  $V_i$  decreases with latitude from  $V_i$  about 2000 in the south to 1000 at the level of about Gävle (between 6838000 and 6938000 in Figure 10), indicating better dispersion facilities in the south. In Sweden different weather systems are dominant in the northern and southern parts during winter, influencing the  $V_i$ , and thus the dispersion of air pollutants, differently. However, this latitudinal pattern is very much levelled out during spring and summer, whereas other local differences, such as topographical effects, become more important to the dispersion pattern. In Figure 11 the east-westerly distribution of  $V_i$  is shown.



# Figure 11 East-westerly profiles of calculated two-month means of V<sub>i</sub> for each 1000 longitude for 2005 in all towns. The data is divided into two sections a) south of Gävle and b) north of Gävle.

In southern Sweden the difference of V<sub>i</sub>'s between the months is larger than in the north. If comparing the means of January-February, the V<sub>i</sub>, and thus the dispersion, is more efficient in the south. In the north it is also a difference from east to west with the highest V<sub>i</sub>'s in the mountains (east) and close to the sea (west). The southern east-westerly profiles show the effect of the coast (i.e. sea and land breeze), by higher V<sub>i</sub>'s, especially during September-December. Generally, in the inland part of the northern profile, especially during the winter months, the pattern is very varying (zigzagged). This is possible due to locally induced factors, such as limited dispersion in valleys, which is indicated by low V<sub>i</sub>'s. The terrain in northern Sweden is characterized by distinct topography which provides favourable conditions for inversions to develop. In the southern part the terrain is mainly more smooth and therefore the variation of inland V<sub>i</sub>'s is less.

### 3.1.3.4 Multivariate data analysis

MVDA is a tool that can be used for many types of data. In this project it has been used to separate different shares of the total  $PM_{10}$  concentration based on six parameters which represent different sources. The six parameters are presented in Chapter 3.1.3. The data has been evaluated for 1881 communities in Sweden.

Typical examples of MVDA methods are principal component analysis (PCA) and partial least squares (PLS) (Martens and Naes, 1989; Wold et al., 1987; Geladi and Kowalski, 1986). Both techniques reduce the multidimensional data set to lower dimensions, by calculating so-called principal components (PCs) that describe the data. A PCA model is based on the X-block (i.e. content or use indicators) and calculated in such a way that it describes as much variance as possible in the data, whilst a PLS model also takes the correlation to the response(s) of interest (here  $PM_{10}$ ) into account. Results from PLS and PCA are often interpreted in score plots and loading plots. Score plots show how the samples are distributed and loading plots display the relationships between three of the six variables (here  $NO_2$ , Studded tyres and Wood fuel burning). Figure 12 below shows a geometric interpretation of PLS.



Figure 12 a) Each observation has a value for each parameter, giving it a coordinate in the n-dimensional space (n = number of variables, in this example, n = 3, in this project, n = 6). Each observation also has a corresponding  $PM_{10}$  value. b) A number of principal components (PCs) are placed in the n-dimensional space in such a way that they describe the data as well as possible. c) The score plot shows the projection of the observations on the PC plane and the loading plot shows the influence of each variable on the PCs. 'S T' means 'Studded tyres' and 'W F B' means 'Wood fuel burning'. The principle is the same with six variables (as in this project), but it is much more difficult to visualise in pictures.

In this project, the data was divided into six different time periods (two months per period), based on the fact that the use of studded tyres and the wood fuel burning contribute less to the  $PM_{10}$ content during the summer and more during the winter, so one generic model representing a whole year, would not give a good prediction of the  $PM_{10}$  content. This resulted in six different PLS models predicting the  $PM_{10}$  content based on the urban background  $NO_2$  concentration, usage of studded tyres, wood fuel burning, energy index, mixing index and the latitude for each community. Three models (month 5-6, 7-8 and 9-10) do not have any contribution from the usage of studded tyres since these types of tyres are not used during the summer in any part of Sweden. This variable is therefore excluded in these three models.

All six models give extremely good predictions of the  $PM_{10}$  content. All models have a performance of over 90%. The maximum possible performance of a model is 100%, which is unrealistic to receive for a model since there are always contributions to the model that can not be explained, the air does not behave exactly the same at all times.

The model performance is here assessed by cross-validation<sup>1</sup>. It was done as described below.

- Dividing the dataset into 8 segments
- Calculating a model on 7 of the 8 segments
- Applying (predicting  $PM_{10}$ -values) the model on the left out segment (1/8)
- Calculating the explained variance for the predicted segment and repeating this procedure until all segments have been predicted.
- Finally pooling the explained variance from all the eight the segments and dividing by the total variance (for PM<sub>10</sub>) to obtain the percentage of the explained variance.

The result presented in Table 2 shows the performance  $(Q^2)^2$  of the models for each time period.

Table 2The performance of the models, measured as cross validated explained variance for PM10.

Model	Performance (%)
Month 1-2	94,9
Month 3-4	97,9
Month 5-6	96,4
Month 7-8	97,2
Month 9-10	96,9
Month 11-12	96,3

Based on the prediction of  $PM_{10}$ , the proportional contribution from each parameter to the  $PM_{10}$  content was also calculated. The result presented in Table 3 shows the average contribution (in percent) from each parameter to the  $PM_{10}$  content for each specific time period, and have been further used for calculating the different source contributions (see further Chapter 4.1.2).

<sup>&</sup>lt;sup>1</sup> **Cross validation:** Parameters are estimated on one part of a data matrix (observations) and the goodness of the parameters tested in terms of its success in the prediction of the rest of the data matrix (observations)

<sup>&</sup>lt;sup>2</sup>  $\mathbf{Q}^2$ : Goodness of prediction, describes the fraction of the total variation of the Y:s that can be predicted by the model according to cross validation (max 1) (in this case  $Q^2$  = performance)

**Table 3**Average contribution (%) to the  $PM_{10}$  content for each variable and time period normalised<br/>to sum up to 100. Other variables, not measured and not presented here, are also affecting<br/>the  $PM_{10}$  content.

Time period	Wood fuel	Energy	Studded	Traffic	Meteorological	Latitude
/Variable	burning	index	tyres	content	index	
Month 1-2	16	6	13	32	17	16
Month 3-4	11	5	43	27	13	1
Month 5-6	19	18	-	31	25	7
Month 7-8	1	1	-	47	42	9
Month 9-10	10	21	-	38	26	5
Month 11-12	9	15	18	31	21	6

### 3.2 PM<sub>2.5</sub> concentration calculations

The estimation of the  $PM_{2.5}$  concentrations in Sweden was performed by using a ratio relation between monitored  $PM_{2.5}/PM_{10}$  on a yearly basis (data from www.ivl.se). This is somewhat rough, since the ratio is likely to vary with season, but as the available monitoring data was very limited it is not possible to adjust for this.

The ratio varies with type of site location, from lower values in city centres to higher values in regional background, where a large proportion of the  $PM_{10}$  concentration consists of  $PM_{2.5}$ . Three different ratios were calculated based on monitoring data; for rural, central urban background and suburban (a mean between the two others) conditions (Table 4).

Type of area	Ratio (PM2.5/PM10)
Central urban background	0.6
Suburban background	0.7
Rural background	0.8

**Table 4**Calculated ratios applied for different types of surroundings, based on monitoring data.

The different ratios in Table 4 were allocated to different city areas based on the population distribution pattern of cities. For the three major cities (Malmö, Göteborg and Stockholm) 40 % of the population was estimated to live in central urban areas and 60 % in suburban areas. For the smaller cities, 55 % of the population was estimated to live in central urban areas and 45 % in suburban areas. Thus, in smaller cities the majority of the population was allocated as suburban areas. These population distribution relations are based on information from cities in the eastern part of USA (Figure 13), as no similar distribution pattern was found for European conditions.





The GIS-methodology applied to allocate the grid cells within each city into the different classes in Figure 13 consists of several steps: At first, the population size estimated to the central areas [pop\_central] was identified (40 or 55 % of the population depending on the size of the city). Secondly, the grid cell with the largest population [pop\_large] in the city was identified and allocated to the central area. The population of that grid cell was then subtracted from the population size of the central areas, i.e. [pop\_central] – [pop\_large]. Then the grid cell with the second largest population was identified. This loop was continued until the population in the central areas [pop\_central] had been allocated to grid cells. The remaining grid cells were allocated to the suburban class, corresponding to the remaining 60 or 45% of the population.

When all grid cells had been allocated to the three classes (central urban, suburban and rural background), the ratio  $(PM_{2.5}/PM_{10})$  in Table 4 was applied to the  $PM_{10}$  map to calculate the  $PM_{2.5}$  map. To ensure that the  $PM_{2.5}$  concentration in the cities is never below the background concentration, a background map of  $PM_{2.5}$  was calculated from the background map of  $PM_{10}$  ( $PM_{10} * 0.8$ ).

### 3.3 Health impact assessment (HIA)

Health impact assessments (HIA) are built on epidemiological findings; exposure-response functions and population relevant rates. A typical health impact function has four components: an effect estimate from a particular epidemiological study, a baseline rate for the health effect, the affected number of persons and the estimated "exposure" (here pollutant concentration).

The excess number of cases per year may be calculated as:

 $\Delta y = (y_0 \cdot pop) \ (e^{\beta \cdot \Delta x} - 1)$ 

where  $y_0$  is the baseline rate, pop is the affected number of persons; ß is the exposure-response function (relative risk per change in concentration), and x is the estimated excess exposure.

We have for  $PM_{10}$  estimated a yearly mean "background", largely natural, to be approximately 5  $\mu g/m^3$  and have used these 5  $\mu g/m^3$  as a lower cut off in our impact assessment scenarios and accordingly defined exposure above 5  $\mu g/m^3$  as excess exposure resulting in "excess cases".

Regarding the lowest concentrations there are not enough data to assess the effects on health and mortality. The lower cut off in HIA has often been a bit higher than in this study (7-10  $\mu$ g/m<sup>3</sup>). For PM<sub>2.5</sub> the corresponding cut off was set at 4  $\mu$ g/m<sup>3</sup>, roughly based on the ratio to PM<sub>10</sub>.

### 3.3.1 Exposure-response function (ERF) for mortality

It has long been recognized that particle concentrations correlate with mortality, both temporally (short-term fluctuations) and spatially based on mortality and survival (WHO, 2003; WHO, 2006a). Usually the short-term associations are seen as included in the long-term effects when the number of excess deaths is estimated. In addition, the potential years of life lost (PYLL or YoLL) due to excess mortality can only be calculated from the long-term (cohort) studies.

There is now within the research community a focus on the different types of particles and a reasoning that it is likely that their impacts on mortality differ (WHO, 2007). However, a common view is that current knowledge does not allow precise quantification of the health effects of PM emissions from different sources; "Thus current risk assessment practices should consider particles of different sizes, from different sources and with different composition as equally hazardous to health" (WHO, 2007). The practice has also been to treat both  $PM_{10}$  and the fine fraction  $PM_{2.5}$  (quite often considered to be more detrimental to health than the coarse fraction of  $PM_{10}$ ) as being equally toxic by mass, irrespective of the origin. When converting exposure-response functions obtained using urban background  $PM_{2.5}$  as the exposure indicator to  $PM_{10}$ , the factor used has often been their mass relation. This relation may however vary between regions and motivate different factors to be used, as discussed by the APHEIS network (Ballester et al, 2008).

However, the current version of ExternE<sup>3</sup> (2005) includes assumptions about the toxicity of the different PM types which reflect newer evidence that indicates a higher toxicity of combustion particles and especially of particles from internal combustion engines. ExternE treats nitrates as equivalent to half the toxicity of PM<sub>10</sub>; sulfates as equivalent to PM<sub>10</sub>; primary particles from power stations as equivalent to PM<sub>10</sub>; primary particles from vehicles as equivalent to 1.5 times the toxicity of PM<sub>2.5</sub>. The long-term effect on mortality of PM<sub>2.5</sub> has from ACS (see below *Cohort results for long term exposure and mortality*) been assumed to be 1.06 (6 %) for a 10 µg/m<sup>3</sup> increment of annual average PM<sub>2.5</sub>, and the effect of PM<sub>10</sub> to be 0.6 times the effect of PM<sub>2.5</sub> (3.6 % for a 10 µg/m<sup>3</sup> increment). ExternE here assumes a somewhat lower relative risk than the 4.3 % used by the European APHEIS network (www.apheis.net; Medina et al, 2004) after the trilateral study by Künzli et al (2000).

In most urban areas it is the concentration of  $PM_{10}$  that is measured since the current air quality limits are based on this fraction.  $PM_{10}$  includes ultrafine soot particles, fine secondary particles as well as more coarse particles mainly of crustal origin. In many Swedish cities the most important local source of  $PM_{10}$  is road dust, including wear particles, sand etc. Epidemiological studies usually build on measured mass concentrations of  $PM_{10}$  or  $PM_{2.5}$ , both influenced by several sources but not so much determined by motor vehicle exhaust particles. However, nitrogen oxide concentrations are highly correlated with the number of exhaust particles, and therefore  $NO_X$  or  $NO_2$  often are better indicators for the exposure to exhaust particles than is  $PM_{10}$  or  $PM_{2.5}$ , and

<sup>&</sup>lt;sup>3</sup> The ExternE project (www.externe.info, ExternE 2005) is a long lasting research project funded by the European Commission's Directorate-General XII (Science, Research and Development) initiated in 1991. The main purpose of the project was to provide knowledge concerning the external costs of energy production in Europe. The first series of reports were published in 1995, with updates in 1998 and 2005.

excess mortality associated with levels of nitrogen oxides may largely reflect the effect of exhaust particles.

Most assessments of particle exposure and mortality only include effects of long-term exposure and are mostly based on the American Cancer Society (ACS) cohort results (Pope et al, 1995). Since these results are used in the US assessments, by WHO (2006a,b) and by EU in the CAFE program, the ACS study has become the most influential and widely cited study of particles and mortality. The original report, a reanalysis initiated by American Health Effects Institute (HEI) (Krewski et al, 2000) and more recent analyses with longer follow up and improved exposure data (Pope et al, 2002) have all demonstrated associations between PM and all-cause and cause-specific mortality. With the robust relation in the ACS study and only a few other studies of long-term effects available, the ACS study together with the older Six Cities study (Dockery et al, 1993) have also been very influential for setting limit values. Two other small studies, the Southern California study (Abbey et al, 1999) and the Veterans cohort study (Lipfert et al, 2000) found in principle no statistically significant associations with PM.

Table 5 below from a US EPA staff paper (2005) gives a brief summary of the cohort results.

Table 5	Summary of different cohort studies. The relative risks are presented together with the 95%
	confidence interval (CI) in brackets. The concentrations are given as mean values with
	minimum and maximum concentrations in brackets. $NR = not$ reported.

Study	Indicator (Increment)	Relative Risk (95% CI)	Study Concentrations
T 1/T . 1 M	1•. • A 1 1.		(µg/m <sup>3</sup> )
Increased Total Morta	lity in Adults		
Six City <sup>A</sup>	$PM_{15/10} (20 \ \mu g/m^3)$	1.18 (1.06, 1.32)	NR (18, 47)
	$PM_{2.5} (10 \ \mu g/m^3)$	1.13 (1.04, 1.23)	NR (11, 30)
	$SO_4^{=}$ (15 µg/m <sup>3</sup> )	1.54 (1.15, 2.07)	NR (5, 13)
Six City <sup>B</sup>	$PM_{15-2.5} (10 \ \mu g/m^3)$	1.43 (0.83, 2.48)	
ACS Study <sup>C</sup>	$PM_{2.5} (10 \ \mu g/m^3)$	1.07 (1.04, 1.10)	18 <sup>U</sup> (9, 34)
(151 U.S. SMSA)			
	$SO_4^{=}$ (15 µg/m <sup>3</sup> )	1.11 (1.06, 1.16)	11 <sup>U</sup> (4, 24)
Six City Reanalysis <sup>D</sup>	$PM_{15/10} (20 \ \mu g/m^3)$	1.19 (1.06, 1.34)	NR (18, 47)
	$PM_{2.5} (10 \ \mu g/m^3)$	1.14 (1.05, 1.23)	NR (11, 30)
ACS Study Reanalysis <sup>D</sup>	$PM_{15/10} (20 \ \mu g/m^3) (dichot)$	1.04 (1.01, 1.07)	59 (34, 101)
	$PM_{2.5} (10 \ \mu g/m^3)$	1.07 (1.04, 1.10)	20 (10, 38)
	$PM_{15-2.5} (10 \ \mu g/m^3)$	1.00 (0.99, 1.02)	7.1 (9, 42)
ACS Study Extended	$PM_{2.5} (10 \ \mu g/m^3) (1979-83)$	1.04 (1.01, 1.08)	21 (9, 34)
Analyses <sup>E</sup>	$PM_{2.5} (10 \ \mu g/m^3) (1999-00)$	1.06 (1.02, 1.10)	14 (5, 20)
	$PM_{2.5} (10 \ \mu g/m^3)$ (average)	1.06 (1.02, 1.11)	18 (7.5, 30)
Southern California <sup>F</sup>	$PM_{10} (20 \ \mu g/m^3)$	1.09 (0.99, 1.21) (males)	51 (0, 84)
	$PM_{10}$ (30 days/year>100 µg/m <sup>3</sup> )	1.08 (1.01, 1.16) (males)	
	$PM_{10} (20 \ \mu g/m^3)$	0.95 (0.87, 1.03) (females)	51 (0, 84)
	$PM_{10}$ (30 days/year>100 µg/m <sup>3</sup> )	0.96 (0.90, 1.02) (females)	
Southern California <sup>E</sup>	$PM_{2.5} (10 \ \mu g/m^3)$	1.09 (0.98, 1.21) (males)	32 (17, 45)
	$PM_{10-2.5} (10 \ \mu g/m^3)$	1.05 (0.92, 1.21) (males)	27 (4, 44)
Veterans Cohort <sup>G</sup>	$PM_{2.5} (10 \ \mu g/m^3) (1979-81)$	0.90 (0.85, 0.95) (males)	24 (6, 42)

A Dockery et al. (1993) ; BEPA (1996) ; c Pope et al. (1995); d Krewski et al. (2000); E Pope et al. (2002);

F Abbey et al. (1999); G Lipfert et al. (2000); U all years

In a longer follow up of the ACS cohort, Pope et al (2002) found a relative risk (RR) of 1.06 (6 %) due to a 10  $\mu$ g/m<sup>3</sup> increment of average PM<sub>2.5</sub>. This RR is assumed in most assessments, either for all anthropogenic sources without adjustments as in APHEIS, CAFE and the WHO report on LRTAP (WHO, 2006b) or with various modifications as in ExternE mentioned above.

In the ACS study there were also close correlations of the relative risks (RR) with the concentration of sulfates, indicating that the association with mortality may be driven mainly by secondary particles, in particular sulfates. A major shortcoming is that ACS, as well as the Six Cities study, use only community average concentrations from central monitors as particle exposure variables. This type of monitor usually reflects mainly the regional background levels. With their focus on the urban background, these studies are likely to underestimate the effects of more locally elevated concentrations in the vicinity of sources such as traffic. The drawbacks in terms of exposure misclassification have been discussed in an article by Mallick et al. (2002), who performed a hypothetical analysis that attempted to correct for such misclassification in the Six Cities Study, and found two- to three-fold higher effect estimates than those originally reported.

In a subset of ACS subjects all from Los Angeles County, Jerrett et al (2005) extracted health data from the ACS survey for metropolitan LA on a zip code-area scale. Data from 23 monitoring stations was used to assign exposure estimates to 267 zip code areas with a total of 22 905 subjects. For all-cause mortality they found for PM<sub>2.5</sub> alone and control for age, sex, and race, the relative risk per 10  $\mu$ g/m<sup>3</sup> to be 1.24 (95% CI = 1.11–1.37), whereas the RR with adjustments for 44 individual confounders was 1.17 (95% CI = 1.05–1.30). These results suggest that the chronic health effects associated with intraurban gradients in exposure to PM<sub>2.5</sub> may be even larger than previously reported for metropolitan areas. The direct comparison with the ACS main results show effects that results are nearly 3 times larger than in models relying on inter-community exposure contrasts.

The findings with ACS data from Los Angeles are in line with recent evidence (Nafstad et al., 2004) suggesting that intraurban exposure gradients may be associated with even larger health effects than reported in interurban studies.

Coarse (PM<sub>10-2.5</sub>) and crustal particles have not been associated with mortality in the cohort studies, and have shown less evident short-term effects on mortality (Brunekreef & Forsberg, 2005; WHO, 2006). In the literature there are only a few studies of road dust or the coarse fraction when studded tyres are used as in Sweden, and these studies do not focus on mortality. However, there are some studies of coarse and crustal particles. In the Six Cities Study the mass of crustal particles (using Silicon as a tracer) was not associated with daily mortality (Laden et al, 2000). In that study the elemental profile of the crustal factor was qualitatively similar to published chemical analysis of road dust. A study of daily mortality in Phoenix found no increase in mortality (Mar et al, 2000). Also in a later analysis fine particle soil was not associated with increased risks (Mar et al, 2006).

### 3.3.1.1 Selected exposure-response functions

Despite the fact that usually, as in CAFE, all PM regardless of source is considered as having the same effect per mass concentration (WHO, 2007), we have in this study for  $PM_{10}$  and mortality used a less conservative approach. We have chosen to assume that road dust as mainly coarse, crustal particles have a smaller effect than the typical, total mix of particles in the ACS cohort study (Pope at al., 1995), that was largely built up by secondary particles. Furthermore, we assume that primary combustion PM has a larger effect than the typical, total mix of particles.

For  $PM_{10}$  in general we have adopted the exposure-response coefficient 4.3 % per 10 µg/m<sup>3</sup> converted from the American  $PM_{2.5}$  results and used by amongst others the APHEIS project (Medina et al, 2004) after the trilateral study by Künzli et al (2000).

Primary combustion particles are found in the fine fraction (PM<sub>2.5</sub>). Acknowledging the indications of a stronger effect of such particles, we have in this study applied the exposure-response coefficient 17 % per 10  $\mu$ g/m<sup>3</sup> from the intraurban Los Angeles analysis of ACS data (Jerret et al, 2005).

The major part of road dust  $PM_{10}$  is in the  $PM_{10\cdot2.5}$  fraction. Since there is in principle no evidence from the cohort studies for an effect of coarse particles ( $PM_{2.5\cdot10}$ ) on mortality, and weak support for any effect of the crustal fraction, road dust will here be assumed to only have a short-term effect on mortality on the scale that  $PM_{10}$  has in general. Since studies as APHEA2 have shown that the "short-term effect" in fact lasts over several weeks, we choose to assume a cumulative effect of 1 % increase in all cause non-external mortality per 10 µg/m<sup>3</sup>, based on the meta-regression in the largest European study, APHEA-2, (Zanobetti et al, 2002).

For  $PM_{2.5}$  we do not have calculations of the contribution from different sources, and therefore we simply apply the 6 % per 10 µg/m<sup>3</sup> from the ACS study as was done by CAFE. With the relative risk for  $PM_{2.5}$  long-term exposure and total mortality found in ACS, 6 % per 10 µg/m<sup>3</sup>, and our assumed typical mortality of 1 010 per 100 000 persons and year (see further in 3.3.3), the concentration-response function for  $PM_{2.5}$  could also be expressed as approximately 0.0000606 deaths for a change of 1 µg/m<sup>3\*</sup>person year.

### 3.3.2 Exposure-response function for morbidity

For morbidity we have in this study included only some of the potentially available health endpoints to be selected. We have decided to include some important and commonly used endpoints that allow comparisons with other health impact assessments and health cost studies.

### 3.3.2.1 ERF for hospital admissions

The exposure-response function (ERF) for the short-term effect of  $PM_{10}$  on respiratory hospital admissions has been estimated to 0,0000103 per 1 µg/m<sup>3\*</sup>person year based on results from APHEIS 3 (APHEIS, 2005) and our assumed baseline of 903 hospital admissions per 100 000 persons and year. It is an open question whether this ERF should be modified when PM levels are expressed as  $PM_{2.5}$ .

The ERF for the short-term effect of  $PM_{10}$  on cardiovascular hospital admissions has been estimated to 0,0000078 per 1 µg/m<sup>3\*</sup>person year according to a literature review by the UK expert panel COMEAP (2006) assuming a baseline of 2602 hospital admissions per 100 000 persons and year.

It could be justified to modify these exposure-response functions when PM levels are expressed as  $PM_{2.5}$  instead of  $PM_{10}$ , but for studies of PM10 and acute effects there is no general support to modify the ER functions (Brunekreft and Forsberg, 2005) since these effects may also depend on the relative concentrations. Thus, we have chosen not to convert these relative risk functions from  $PM_{10}$  to  $PM_{2.5}$ .

### 3.3.2.2 ERF for chronic bronchitis

There is very limited data regarding new cases of chronic bronchitis and long-term exposure to PM. The Seventh Day Adventist Study (AHSMOG: Adventist Health Smog; Abbey, 1999) conducted in the US examined people on two occasions approximately 10 years apart, in 1977 and again in 1987-88. In this study chronic bronchitis was defined with the common definition of reporting chronic cough or sputum on most days, for at least three months of the year, for at least two years. New cases were defined as those which reported these symptoms at the follow up in 1987-88 but not in 1977.

Assuming the RR from Abbey at al. (1995) and a background incidence rate (adjusted for remission of chronic bronchitis symptoms) of 0.378% estimated from Abbey et al. (1993, 1995), Hurley et al. (2005) has derived an estimated exposure-response function for new cases of chronic bronchitis in the population aged 27 years or older of 26.5 (95% CI -1.9, 54.1) per 10  $\mu$ g/m<sup>3</sup> PM<sub>10</sub> per year per 100 000 adults, or 0.0000265 new cases for a change of 1  $\mu$ g/m<sup>3</sup>\*person year. It is an open question whether this ERF should be modified when PM levels are expressed as long-term concentration of PM<sub>2.5</sub>, as it is done the other way for long-term mortality effects seen with measured levels of PM<sub>2.5</sub>.

### 3.3.2.3 ERF for restricted activity days

Six consecutive years (1976-1981) of the US Health Interview Study (HIS) were used to study restricted activity days (RADs) in adults aged 18-64 (Ostro, 1987; Ostro and Rothschild, 1989). In the multi-stage probability sample of 50,000 households from metropolitan areas of all sizes and regions severity was classified as (i) bed disability days; (ii) work or school loss days and (iii) minor restricted activity days (MRADs), which do not involve work loss or bed disability but do include some noticeable limitation on 'normal' activity.

The weighted mean pollutant coefficient for restricted activity days (RADs) was linked to estimated background rates of, on average, 19 RADs per person per year. From this study there is an exposure-response function of 902 RADs (95% CI 792, 1013) per 10  $\mu$ g/m<sup>3</sup> PM<sub>2.5</sub> per 1,000 adults at age 15-64, or 0.092 RADs for a change of 1  $\mu$ g/m<sup>3</sup>\*person year. In this age group we may see this as work loss days.

For  $PM_{10}$  we have in this study decided to convert this ERF using the same relation used in APHEIS (Medina et al, 2004) for long-term effects on mortality (ERF for  $PM_{10} \approx 0.72$  ERF for  $PM_{2.5}$ ), that means for  $PM_{10}$  close to 0.065 RADs for a change of 1 µg/m<sup>3\*</sup>person year.

### 3.3.3 Selected baseline rates for mortality and admissions

In order to estimate how many deaths and hospital admissions that depend on elevated air pollution exposure we need to use a baseline rate. For our study of NO<sub>2</sub> (Sjöberg et al, 2007), we used the official national rate for 2002 published by the register unit Epidemiologiskt Centrum (EpC), at The Swedish Board of Health and Welfare (www.sos.se). It was 1 063 deaths per 100 000 persons. The national mean rate for non-external causes of death was approximately 1 010. For the sake of comparability, and since these rates change only slowly, we used this same rate in this study.

To estimate the number of life years lost, we have also used life tables for Greater Stockholm and the WHO software AirQ to crudely estimate the average of years lost per excess death. Assuming the same relative increase in mortality in all ages, we found a loss of just over 11 years per death.

For our calculations we have used 1 010 deaths per year from non-external causes per 100 000 persons in the entire population 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, so the impact calculation results only marginally would be changed by such an exclusion.

In our study of NO<sub>2</sub>, we reported that the mean number of hospital admissions for respiratory disease in Sweden 2004 was 975 per 100 000 persons, and out of these 903 per 100 000 persons were acute (not planned) admissions. The mean number of cardiovascular hospital admissions in Sweden 2004 was 2 602 per 100 000 persons. Of these, 2 063 were acute (not planned) admissions. For the sake of comparability we have used the same baseline rates for acute admissions in this study. Official statistics show variations in the baseline rates both for mortality and hospital admissions between counties, but variations within counties are not presented and may be larger than between different counties.

### 3.4 Socio-economic valuation

The method used for socio-economic valuation in this study is identical to the method used in Sjöberg et al. 2007. In this report, relevant updates on health end points are presented. There are two additions to the valuation performed in Sjöberg et al. 2007, valuation of Chronic Bronchitis (CB) and Restricted Activity Days (RAD).

In the ExternE update from 2005 (www.externe.info) it is recognised that the suitable valuation of health related effects consists of three components; Resource costs (costs for medical aid), Opportunity costs (loss in productivity) and Disutility (costs for discomfort etc).

These costs correspond to welfare parameters of relevance for valuation of health effects related to air pollution. They allow for consideration of all economic decision makers in society; individuals (households), firms and government. Ideally, all these costs should be taken under consideration during the valuation of health effects, but it is sometimes difficult to measure and calculate reliable estimates of these cost parameters. It can also be that some methods of valuation aggregate the above mentioned parameters thereby making it difficult to distinguish between the different types of costs.

### 3.4.1 Quantified results from the literature

OECD (2006) summarises the recent developments in the area of Cost Benefit Analysis (CBA) and valuation and presents the results from several studies including the ExternE project and Chilton et al. (2004). Results of valuation of mortality, expressed as the measure Value of Statistical Life (VSL), of relevance for our study are summarised in Table 6 below.

	VSL [\$ million]	Currency year
Hammit 2000	3 – 7	1990
Alberini et al. 2004	1,5 - 4,8 (small risk reduction) 0,9 - 3,7 (large risk reduction)	2000
Krupnick et al. 1999	0,2 - 0,4	1998
Markandya et al. 2004	1,2 - 2,8 0,7 - 0,8 0,9 - 1,9	2002
Chilton et al. 2004	0,3 - 1,5	2002

Table 6	VSL estimates of mortality from previous studies (OECD, 2006).
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The values in the table are used mainly for illustration of the most common ranges of VSL in the literature on the subject. Valuations that are based on risk contexts such as occupational risks (accidents when at work), road traffic, and fires are excluded from this table. The column indicating the currency year is necessary for a potential transfer of the results to other valuation studies. The results from Alberini et al. (2004), Krupnick et al. (1999) and Markandya et al. (2004) are all results from studies on risk reductions for persons in the age class 70-80 years.

Other values of interest for our study are the valuation of mortality expressed as the measure VOLY estimates (Value Of Life Year) comparing Chilton et al (2004) with Markandya et al. (2004), Table 7.

**Table 7**VOLY estimates on mortality from previous studies (OECD, 2006).

	VOLY [£]	Currency Year
Chilton et al. 2004	27630	2002
Markandya et al. 2004	41975	2002

The VOLY given by Markandya et al. (2004) is an indirect estimate derived from the VSL estimate in the study while the VOLY from Chilton et al. 2004 is a direct estimate.

Furthermore, OECD (2006) also indicates morbidity valuations for several different health effects given in the available literature. The values of interest for our study are given in Table 8.

	Study quoted		
Type of Illness (morbidity)	Ready et al. 2004	ExternE 1998	Maddison 2000
Hospital admission for treatment of respiratory disease	€ 490	€ 7870	n.a.
3 days spent in bed with respiratory illness (3 RAD)	€ 155	€ 75	€ 195

**Table 8**Morbidity valuation estimates (OECD, 2006).

In OECD there is no suggestion as to why the ExternE values for hospital admissions is so much higher than in Ready et al. (2004). On the other hand, these ExternE values are then updated in the following update of the ExternE project, see Table 9. The values given by Ready et al. 2004 are identical to the ones in Pearce 2000, which is mentioned above.

The following Table 9 lists the central estimates of monetary values for health effects that are of relevance to our study as they are valued in the latest update of the ExternE project.

Mortality	Value	Unit
Value of a statistical life	1052000	€ <sub>2000</sub> /case
Value of Life year lost	50000	€ <sub>2000</sub> / year
Morbidity		
Chronic Bronchitis	190 000	€ <sub>2000</sub> / case
Cardiovascular Hospital admission, Health Care resource costs	620*	€ <sub>2000</sub> / day in Hospital
Respiratory Hospital admission, Health care resource costs	323	€ <sub>2000</sub> / day in Hospital
Hospital admission, cost for absenteeism from work	82	€ <sub>2000</sub> / day
Hospital admission, WTP for avoided hospitalisation	437* *	€ <sub>2000</sub> / occurrence
Restricted Activity Day (RAD)	46	€ <sub>2000</sub> / day

 Table 9
 Economic values of health effects (ExternE, 2005)

\* The value for Cardiovascular health care resource costs is derived through multiplying the RHA with 1.92, a multiplier provided by ExternE 2005.

\*\* Hospital treatment for respiratory disease lasting three days, followed by five days at home in bed. The value is based on Ready et al. 2004 but differs from the same value given by OECD (2006) due to exchange rates and currency year used.

ABS associates (2000) indicates socio-economic values of several health end points related to high levels of PM, as seen in Table 10.

# Table 10Socio-economic values for health effects related to high $PM_{10}$ levels (ABS Associates 2000).

Health end point	Value	Unit
Mortality		
VSLMorbidity	6 120 000	1999 \$ / case
Morbidity		
Chronic Bronchitis (WTP)	331 000	1999 \$ / case
Work loss day (RAD)	105	1999 \$ / day

As can be seen, all the estimated health effect end points in ABS associates (2000) are higher than in corresponding European studies.

Chilton et al. (2004) performed surveys in 665 households to estimate the willingness to pay for avoided health effects that can be linked to poor air quality. The quantitative values of the two health effects of concern for our study are summarised in Table 11.

	£2002	
Value of a one year gain in life expectancy in normal health	£ 6 040 - 27 630	£ 27 630 is the recommended value for policy use.
Value of avoiding a respiratory hospital admission	£1310-7110	
Value of Prevented Fatality from reduced levels of air pollution*	~£ 241 600 - 1 105 200	
Value of Prevented Fatality in road accidents**	£ 1 250 000	

**Table 11**Economic values of health effects (Chilton et al., 2004).

The value is derived from the value of a one year gain in life expectancy and assumes 40 remaining life years and a 0 % discount rate.

\*\* Value originating from a British study and quoted in Chilton et al. 2004

As a final remark from the three sources used for the analysis in our study one should mention the huge variance in WTP for avoided hospital admission. The value given by ExternE (1999) is  $\epsilon_{1995}$  7870, in Ready et al. (2004) ~ $\epsilon$  490 (different values given by OECD 2006 and ExternE 2005), and in Chilton et al. (2004) the values range between £ 1310 - 7110. When adjustments are made for currency years and exchange rates, the variance becomes even larger. This variance motivates further research in the area of WTP for hospital admissions related to respiratory diseases. The valuation estimates used in our project to calculate socio-economic costs of high levels of PM are presented in chapter 4.4 and Table 26.

## 4 Results

### 4.1 Calculation of PM concentrations

### 4.1.1 National distribution of PM<sub>10</sub> concentrations

The annual mean concentrations of  $PM_{10}$  for 2005, calculated with the URBAN model, are presented in Figure 14. The result is based on calculated two-month means in order to capture the seasonal variations, where higher concentrations of  $PM_{10}$  usually occur during late winter or spring depending on the location in the country.



Figure 14 Calculated annual mean concentrations of PM<sub>10</sub> for 2005.

In Figure 14 it can be seen that the  $PM_{10}$  concentrations on a yearly basis are governed to a large extent by the regional background concentrations. There is a large latitudinal decrease to the north of the regional background concentration, due to the strong influence of long range transport. The urban background concentrations in the largest towns in the southern and western parts of Sweden are calculated to be about 24-28  $\mu$ g/m<sup>3</sup>, while the concentration in Stockholm is estimated to about 19  $\mu$ g/m<sup>3</sup>. Compared to the environmental standard for the annual mean value (40  $\mu$ g/m<sup>3</sup>) there are no exceedances of this limit value exceeded in urban background air in Swedish towns and cities.

The total  $PM_{10}$  concentrations in regional and urban background air in relation to the latitude are shown in Figure 15. Compared to measurement data the regional background concentration, calculated as a yearly mean, is somewhat underestimated in southern Sweden, while there is good agreement in the north (Vindeln). As can be seen in Figure 15 the calculated local contribution decreases with increasing latitude. The monitoring data (as yearly as well as winter half-yearly means) lies well within the interval for the standard deviation of the calculated yearly means.



**Figure 15** Measured concentrations of total PM<sub>10</sub> in regional background (RB) and urban background (UB) air compared to calculated local, regional and total (local+regional) urban background (UB) concentrations. The error bars show the standard deviation.

### 4.1.2 Separation of PM<sub>10</sub> sources

The multivariate method described in chapter 3.1.2.4 was used to separate the local  $PM_{10}$  contributions into different particle sources. The distribution of the locally developed  $PM_{10}$  concentrations is related to the latitude, and the result presented in Figure 16 shows that the local  $PM_{10}$  concentration decreases as the latitude increases. One reason for this is possibly due to the fact that there are more large towns in the south, thus causing a larger local contribution to the concentration of  $PM_{10}$  (Figure 17). However, when comparing towns of similar size at different latitudes the calculated local contribution is still larger in the southern part compared to the northern part.


Figure 16 The latitudinal variation of calculated local  $PM_{10}$  concentrations in urban background air (no regional background concentration included).



**Figure 17** The latitudinal location of towns (from 200 inhabitants) and corresponding population, a) in all urban areas and b) summarized in the defined latitudinal classes.

A similar decreasing pattern of local PM<sub>10</sub> contributions is also visible in measurement data. Figure 18 shows the local contributions calculated from monitoring data, where regional background concentrations have been subtracted from the urban background levels. However, during the spring months, March and April, the pattern is different and therefore these months have been visualized separately (Figure 18a). In Figure 18b the local concentrations decrease with increasing latitude, which is the opposite to locally developed NO<sub>2</sub> concentrations (Figure 18c). This has earlier been explained by poorer dispersion facilities in the north of Sweden. The reason why the local contribution of PM<sub>10</sub> does not follow the same pattern as NO<sub>2</sub> may be because the local PM<sub>10</sub> concentration arises principally from dust/resuspension. It is assumed that the road wear, and thus the origin for dust on roads/resuspension, is small in northern Sweden due to the snow coverage on the roads. The exhaust particles are mainly very small, resulting in a very limited contribution to the PM<sub>10</sub> concentration.





The results from the multivariate analyses are presented as percentage contributions in Figure 19. The shares from "total traffic" and "resuspension" increase with increasing latitude for all months. This is logical since this part is based on the  $NO_2$  concentration, following the same trend (Figure 18). All the other parameters vary with the season. In January-February the other parameters decrease slightly.

In March-April "studded tyres" is the most dominant parameter in southern Sweden, but decreases towards the north. The opposite pattern is shown for "wood" and "dispersion index" but there is quite large variation in southern Sweden, possibly due to the differences in inland/coastal conditions. The proportions from "energy index" and "Y-coordinate" (latitude) are similar in the whole country.

In May-June all the parameters, except "total traffic" and "resuspension", either decrease or are rather similar at all latitudes. There are, however, two extremes in southern Sweden. This is possibly due to worse dispersion at inland locations. The contribution from "studded tyres", and at some locations also "energy index", are zero during this time period.

In July-August most of the contribution to the local  $PM_{10}$  can be explained by "total traffic", "resuspension" and/or "dispersion index". There is also some influence from domestic heating in the north of Sweden ("energy index"). In southern Sweden the share from latitude (Y-coordinate) is not negligible. The shares from "studded tyres", and at some locations also "energy index", are zero.

During September-October the share from "wood" is increasing with increasing latitude. During this period the climatological differences in the country are quite significant. This is visible in both that the part from "energy index", "dispersion index", and also to some extent the "Y-coordinate", perform a larger variety in between the northern and southern part. The contribution from "studded tyres" is still zero.

In November-December the situation is similar to the previous two month period with the addition of "studded tyres"; the effect from "dispersion index" is more or less the same in the whole country.



Figure 19 The percentage contribution from different sources, to the local PM<sub>10</sub> concentration at different latitudes, based on the multivariate analysis divided into two-month means, a) January-February, b) March-April, c) May-June, d) July-August, e) September-October and f) November-December.

The concentrations (in  $\mu g/m^3$ ) of each share to the local PM<sub>10</sub> concentration are presented in Figure 20. In this analysis some of the calculated shares have been merged together where they were assumed to contribute to the same specified shares of PM<sub>10</sub> (see Table 12).

Shares of PM <sub>10</sub> , used in the multivariate analyses	Merged shares of $PM_{10}$ (µg/m <sup>3</sup> )	Source
Wood plus Energy index	Domestic heating	Particles from wood in domestic heating.
Total traffic minus resuspension	Traffic combustion	Particles from vehicle exhaust pipes.
Studded tyres + resuspension	Resuspension + dust	Particles from resuspended material due to road and tyre wear and other dust material.
Dispersion index + Y- coordinate	Other sources	Likely to originate from uncertainties in the geographical distribution of regional background levels.

**Table 12**Shares of  $PM_{10}$  used for concentration calculations.

During the first two months (January-February) all PM<sub>10</sub> concentrations are rather low. In March-April the resuspended part is the most dominant and is still relatively high in May-June. During these months the share that originates from domestic heating is large. The large proportion in the southern inland has been pointed out earlier in Figure 19c. In summer (July-August) the most dominant shares are the resuspension and the "rest". The PM<sub>10</sub> concentration from the latter part is mainly dependent on the limited dispersion facilities (V<sub>i</sub>) and the "Y-coordinate". The pattern during the last two time periods (September-December) is similar with a relatively small variation between the different parameters.



**Figure 20** The merged shares of the local  $PM_{10}$  concentration ( $\mu g/m^3$ ) based on the calculated percentage distribution divided into two-month means, a) January-February, b) March-April, c) May-June, d) July-August, e) September-October and f) November-December.

### 4.1.3 National distribution of PM<sub>2.5</sub> concentrations

The annual mean concentrations of  $PM_{2.5}$  for 2005 are presented in Figure 21. The result is based on the previously calculated  $PM_{10}$  concentrations in combination with calculated ratios based on empirical  $PM_{10}/PM_{2.5}$  relationships.



Figure 21 The total annual mean concentrations of  $PM_{2.5}$  for 2005.

# 4.2 Population exposure

The population exposure to different particle concentrations has been calculated based on the calculated air concentrations.

### 4.2.1 Exposure to PM<sub>10</sub>

The estimated number of people in Sweden exposed to different intervals of the total  $PM_{10}$  annual mean concentrations in 2005 are shown in Table 13 and also in Figure 22. Approximately 30 % of the were exposed to  $PM_{10}$  concentrations in each of the concentration intervals 5-10, 10-15 and 15-20  $\mu$ g/m<sup>3</sup>. Less than 5 % of Swedish inhabitants were exposed to  $PM_{10}$  levels higher than 20  $\mu$ g/m<sup>3</sup>.

The number of people exposed to the  $PM_{10}$  concentration contribution from the separate sources is presented in Table 14 – Table 18.

PM <sub>10</sub> concentration	Mean PM <sub>10</sub> [µg m <sup>-3</sup> ] Number of		Percentage of	
[µg m <sup>-3</sup> ]		people	population	
0 - <5	5.0	280	0.0 %	
5 - <10	8.4	2 380 610	26.7 %	
10 - <15	12.4	3 121 580	35.1 %	
15 - <20	16.6	2 789 630	31.3 %	
20 - <25	21.9	563 140	6.3 %	
25 - <30	26.3	44 480	0.5 %	
Total:	13.3	8 899 700	100 %	

Table 13	Distribution of exposure	e levels to total PM <sub>10</sub>	in the Swedish	population in 2005.
I uble IC	Distribution of exposure		m the bweaton	population in 2005.

Table 14Distribution of exposure levels to regional background  $PM_{10}$  in the Swedish population in<br/>2005.

PM <sub>10</sub> concentration	Mean PM <sub>10</sub> [µg m <sup>-3</sup> ]	Number of	Percentage of
[µg m-3]		people	population
0-<5	5.0	280	0.0 %
5-<10	8.5	3 093 110	34.8 %
10 - <15	12.9	4 009 620	45.1 %
15 - <20	16.1	1 785 270	20.1 %
20 - <25	20.6	11 440	0.1 %
Total:	12.0	8 899 700	100 %

Table 15Distribution of exposure levels to  $PM_{10}$  from road dust in the Swedish population in 2005.

PM <sub>10</sub> concentration	Mean PM <sub>10</sub> [µg m <sup>-3</sup> ]	Number of	Percentage of
[µg m <sup>-3</sup> ]		people	population
0	n.a.	4 707 370	52.9%
0 - <0.5	0.28	1 107 080	12.4%
0.5 - <1	0.71	1 295 730	14.6%
1 - <1.5	1.24	488 270	5.5%
1.5 - <2	1.73	437 060	4.9%
2 - <2.5	2.22	453 640	5.1%
2.5 - <3	2.73	232 040	2.6%
3 - <3.5	3.21	120 020	1.3%
3.5 - <4	3.75	35 240	0.4%
4 - <4.5	4.04	13 630	0.2%
4.5 - <5	4.93	9 640	0.1%
Total:	0.54	8 899 700	100 %

in 2005.			
PM <sub>10</sub> concentration	Mean PM <sub>10</sub> [µg m <sup>-3</sup> ]	Number of	Percentage of
[µg m-3]		people	population
0	n.a.	4 707 370	52.9%
0 - <0.5	0.14	4 171 230	46.9%
0.5 - <1	0.55	21 120	0.2%
Total:	0.067	8 899 700	100 %

Table 16Distribution of exposure levels to  $PM_{10}$  from *traffic combustion* in the Swedish populationin 2005

Table 17	Distribution	of exposure levels to	PM <sub>10</sub> from	wood related a	lomestic	heating in	the Swedish
	population in	n 2005.					

PM <sub>10</sub> concentration	Mean PM <sub>10</sub> [µg m <sup>-3</sup> ]	Number of	Percentage of		
[µg m <sup>-3</sup> ]		people	population		
0	n.a.	4 707 370	52.9%		
0 - <0.5	0.22	2 647 470	29.7%		
0.5 - <1	0.73	1 087 190	12.2%		
1 - <1.5	1.16	416 220	4.7%		
1.5 - <2	1.69	41 470	0.5%		
Total:	0.22	8 899 700	100 %		

Table 18	Distribution of exposure levels to PM <sub>10</sub> from other sources than those specified above in the
	Swedish population in 2005.

PM <sub>10</sub> concentration	Mean PM <sub>10</sub> [µg m <sup>-3</sup> ]	Number of	Percentage of		
[µg m <sup>-3</sup> ]		people	population		
0	n.a.	4 707 370	52.9%		
0 - <0,5	0.28	1 361 140	15.3%		
0.5 - <1	0.71	1 203 950	13.5%		
1 - <1.5	1.24	547 530	6.2%		
1.5 - <2	1.75	406 390	4.6%		
2 - <2.5	2.27	321 530	3.6%		
2.5 - <3	2.70	173 110	1.9%		
3 - <3.5	3.27	77 310	0.9%		
3.5 - <4	3.70	55 650	0.6%		
4 - <4.5	4.10	24 620	0.3%		
4.5 - <5	4.52	11 480	0.1%		
5 - <5.5	n.a.	0	0.0%		
5.5 - <6	5.52	9 640	0.1%		
Total	0.50	8 899 700	100%		

### 4.2.2 Exposure to $PM_{2.5}$

The estimated exposure of the total annual mean concentrations of  $PM_{2.5}$  is shown in Table 19. The major part of the population, almost 50 %, were exposed to  $PM_{2.5}$  annual mean concentrations between 5 and 10 µg/m<sup>3</sup>. About 48 % of the people in Sweden were exposed to levels between 10 and 15 µg/m<sup>3</sup> and less than 2 % were exposed to  $PM_{2.5}$  concentrations above 15 µg/m<sup>3</sup>.

Table 19Distribution of exposure levels to total  $PM_{2.5}$  in the Swedish population in 2005.

PM <sub>2.5</sub> concentration	Mean PM <sub>2.5</sub>	Number of	Percentage of
[µg m <sup>-3</sup> ]	[µg m-3]	people	population
0- <4	4.0	280	0.0%
4 - <5	4.7	105 710	1.2%
5 - <6	5.5	520 020	5.8%
6 - <7	6.6	636 280	7.1%
7 - <8	7.5	1 549 180	17.4%
8 - <9	8.5	1 021 250	11.5%
9 - <10	9.5	709 670	8.0%
10 - <11	10.5	792 840	8.9%
11 - <12	11.5	1 544 110	17.4%
12 - <13	12.6	1 165 460	13.1%
13 - <14	13.4	468 540	5.3%
14 - <15	14.5	257 980	2.9%
15 - <16	15.5	84 260	0.9%
16 - <17	16.4	40 620	0.5%
17 - <18	17.2	3 300	0.04%
18 - <19	18.3	190	0.0%
Total	9.8	8 899 700	100%

In Figure 22 it can be seen that the percentage exposure is more evenly distributed between the different concentration classes  $5-20 \ \mu g/m^3$  for  $PM_{10}$  than for  $PM_{2.5}$ , reflecting the variation in concentrations between the two particle fractions.



Figure 22 Percentage distribution of the population exposed to total annual mean concentrations of  $PM_{10}$  and  $PM_{2.5}$ .

# 4.3 Estimated health impacts

Excess mortality, as well as the excess number of people suffering from other health related effects, have only been estimated due to pollution levels corresponding to annual mean concentrations of  $PM_{10}$  above 5 µg/m<sup>3</sup> and of  $PM_{2.5}$  above 4 µg/m<sup>3</sup> since there is less scientific support for effects below these levels.

### 4.3.1 Mortality

Excess mortality has been calculated as the yearly number of deaths due to  $PM_{10}$  and  $PM_{2.5}$  concentrations in concentration classes above 5 and 4  $\mu g/m^3$  respectively.

The calculated yearly numbers of excess deaths in each concentration class and totally for  $PM_{10}$ , separated for each of the estimated source contributions, are given in Table 20. Altogether we estimate almost 3 400 excess deaths per year.

#### Table 20Mortality effects of $PM_{10}$ .

*	) Numbers	in	brackets	refer to	concentration.	with	the cui	t off leve	l of 5	'µg/m <sup>‡</sup>	subtracted,	and are
	the values	use	d for calc	ulation	of human hea	lth im	pact.					

Annual PM <sub>10</sub> class [μg/ m³]	Population (n)	Population weighted annual mean contribution PM <sub>10</sub> [μg/ m <sup>3</sup> ] *	Pop* conc	Excess number of deaths
PM Regional ba	ackground			
0 - 5	280	5 (0)	1 400	0
5 – 10	3 093 110	8.5 (3.5)	10 825 885	470
10 – 15	4 009 620	12.9 (7.9)	31 675 998	1 376
15 – 20	1 785 270	16.1 (11.1)	19 816 497	861
20 – 25	11 440	20.6 (15.6)	178 464	8
Subtotal	8 899 700			2715
PM Road dust				
0	4 707 370	n.a.	0	0
0 < 0.5	1 107 080	0.28	309 982	3
0.5 – 1	1 295 730	0.71	919 968	9
1 – 1.5	488 270	1.24	605 455	6
1.5 -2	437 060	1.73	756 114	8
2 – 2.5	453 640	2.22	1 007 081	10
2.5 – 3	232 040	2.73	633 469	6
3 - 3.5	120 020	3.21	385 264	4
3.5 – 4	35 240	3.75	132 150	1
4 - 4.5	13 630	4.04	55 065	1
4.5 – 5	9 640	4.93	47 525	0
Subtotal	8 899 700			48
PM Traffic exh	aust			
0	4 707 370	n.a.	0	0
0 < 0.5	4 171 230	0.14	583 972	100
0.5 - 1	21 120	0.55	11 616	2
Subtotal	8 899 700			102
				cont.

Annual PM <sub>10</sub> class [µg/ m³]	Population         Population weighted annual           Population         mean contribution PM <sub>10</sub> (n)         [μg/ m³] *		Pop* conc	Excess number of deaths
PM Wood sm	oke			
0	4 707 370	n.a.	0	0
0 < 0.5	2 647 470	0.22	582443	100
0.5 – 1	1 087 190	0.73	793649	136
1 – 1.5	416 220	1.16	482815	83
1.5 – 2	41 470	1.69	70084	12
Subtotal	8 899 700			331
PM10 Other	emissions			
0	4 707 370	n.a.	0	0
0 < 0.5	1 361 140	0.28	381 119	17
0.5 – 1	1 203 950	0.71	854 804	37
1 – 1.5	547 530	1.24	678 937	29
1.5 -2	406 390	1.75	711 182	31
2 – 2.5	321 530	2.27	729 873	32
2.5 – 3	173 110	2.7	467 397	20
3 – 3.5	77 310	3.27	252 803	11
3.5 – 4	55 650	3.7	205 905	9
4 - 4.5	24 620	4.1	100 942	4
4.5 – 5	11 480	4.52	51 890	2
5 – 5.5	0	n.a.	0	0
5.5 - 6	9 640	5.52	53 213	2
Subtotal	8 899 700			194
Total				3 390

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The calculated yearly numbers of excess deaths in each concentration class and totally for  $PM_{2.5}$ , separated for each of the estimated source contributions, are given in Table 21. Altogether we estimate close to 2900 excess deaths per year when no sources are assumed to have exposure-response functions different than the "default" assumptions used in, for example, CAFÉ and APHEIS.

#### Table 21Mortality effects of PM2.5.

Annual PM <sub>2.5</sub> class [µg/ m³]	Population (n)	Population weighted annual mean contribution PM <sub>2.5</sub> [μg/ m <sup>3</sup> ]	Pop* conc	Excess number of deaths
3 – 4	280	4 (0)	0	0
4 – 5	105 710	4.7 (0.7)	73 997	4
5 - 6	520 020	5.5 (1.5)	780 030	47
6 - 7	636 280	6.6 (2.6)	1 654 328	100
7 – 8	1 549 180	7.5 (3.5)	5 422 130	329
8 - 9	1 021 250	8.5 (4.5)	4 595 625	278
9 - 10	709 670	9.5 (5.5)	3 903 185	237
10 – 11	792 840	10.5 (6.4)	5 153 460	312
11 – 12	1 544 110	11.5 (7.5)	11 580 825	702
12 – 13	1 165 460	12.6 (8.6)	10 022 956	607
13 – 14	468 540	13.4 (9.4)	4 404 276	267
14 – 15	257 980	14.5 (10.5)	2 708 790	164
15 – 16	84 260	15.5 (11.5)	968 990	59
16 – 17	40 620	16.4 (12.4)	503 688	31
17 – 18	3 300	17.2 (13.2)	43 560	3
18 – 19	190	18.3 (14.3)	2 717	0
Total:	8 889 700			3 140

\*) Numbers in brackets refer to concentrations with the cut off level of  $4 \mu g/m^3$  subtracted, and are the values used for calculation of human health impact.

# 4.3.2 Morbidity effects

We have estimated only a selection of potential morbidity effects that are commonly included and allow comparisons with other studies; respiratory and cardiovascular hospital admissions related to short-term exposure, restricted activity days (RADs) and induction of new cases of chronic bronchitis. Regional background exposure to PM<sub>10</sub> is estimated to result in just over 4 million RADs per year. Local sources are estimated to cause approximately an additional 760 000 RADs per year.

Regional background exposure to  $PM_{10}$  is estimated to induce more than 1100 cases of chronic bronchitis per year. Local sources are estimated to cause more than 200 cases additionally per year.

Regional background exposure to  $PM_{10}$  is estimated to result in about 650 respiratory hospital admissions and almost 500 cardiovascular hospital admissions per year. Local sources are estimated to cause approximately 120 (respiratory) and 90 (cardiovascular) admissions additionally per year.

Annual PM <sub>10</sub> class [µg/ m³]	Chronic Bronchitis (27+)	Respiratory Hospitalisations	RADs (age group 15-64)	RADs (all ages)	Cardiovascular Hospitalisations
PM Regional	background				
0 - 5					
5 - 10	195	112	228 643	692 857	84
10 - 15	571	326	668 997	2 027 264	247
15 - 20	357	204	418 524	1 268 256	155
20 - 25	3	2	3 769	11 422	1
Subtotal	1126	644	1 319 933	3999798	487
PM Road dus	st				
0 < 0.5	6	3	6 547	19 839	2
0.5 - 1	17	9	19 430	58 878	7
1 – 1.5	11	6	12 787	38 749	5
1.5 -2	14	8	15 969	48 391	6
2 – 2.5	18	10	21 270	64 453	8
2.5 - 3	11	7	13 379	40 542	5
3 - 3.5	7	4	8 137	24 657	3
3.5 - 4	2	1	2 791	8 458	1
4 - 4.5	1	1	1 163	3 524	0
4.5 - 5	1	0	1 004	3 042	0
Subtotal	88	43	102 476	310 533	37
PM Traffic ex	chaust				
0 < 0.5	11	6	12 333	37 374	5
0.5 - 1	0	0	245	743	0
Subtotal	11	6	12 579	38 118	5
PM Wood sm	oke				
0 < 0.5	10	6	12 301	37 276	5
0.5 - 1	14	8	16 762	50 794	6
1 – 1.5	9	5	10 197	30 900	4
1.5 - 2	1	1	1 480	4 485	1
Subtotal	34	20	40 740	123 455	16
					cont.

### **Table 22**Morbidity effects of PM10 (number of cases/year or RADs/year)

Annual PM₁₀ class [µg/ m³]	Chronic Bronchitis (27+)	Respiratory Hospitalisations	RADs (age group 15-64)	RADs (all ages)	Cardiovascular Hospitalisations
PM10 Other	emissions				
0 < 0.5	7	4	8 049	24 392	3
0.5 - 1	15	9	18 053	54 707	7
1 – 1.5	12	7	14 339	43 452	5
1.5 -2	13	7	15 020	45 516	6
2 – 2.5	13	8	15 415	46 712	6
2.5 - 3	8	5	9 871	29 913	4
3 - 3.5	5	3	5 339	16 179	2
3.5 - 4	4	2	4 349	13 178	2
4 - 4.5	2	1	2 132	6 460	1
4.5 - 5	1	1	1 096	3 321	0
5 – 5.5	0	n.a.	0	0	0
5.5 - 6	1	1	1 124	3 406	0
Subtotal	81	48	94 788	287 236	36
TOTAL	1 340	761	1 570 516	4 759 140	581

Table 22. Cont.

The effects on hospital admissions and induction of chronic bronchitis are estimated smaller for  $PM_{2.5}$  than  $PM_{10}$  as a result of the combination of exposure distribution and the cut off level when using the same exposure-response assumptions (Table 23). For RADs the assumed exposure-response function was higher for  $PM_{2.5}$  than for  $PM_{10}$ , but the estimated numbers associated with  $PM_{2.5}$  are still smaller as a result of the combination of exposure distribution and the cut off level.

Annual PM <sub>2.5</sub> class [μg/ m³]	Chronic Bronchitis (27+)	Respiratory Hospitalisations	RAD (age group 15-64)	RAD (all ages)	Cardiovascular Hospitalisations
3 – 4	0	0	0	0	0
4 – 5	1	1	2 247	6 808	1
5 – 6	14	8	23 682	71 763	6
6 – 7	30	17	50 225	152 198	13
7 – 8	98	56	164 616	498 836	42
8 – 9	83	47	139 523	422 798	36
9 - 10	70	40	118 501	359 093	30
10 – 11	93	53	156 459	474 118	40
11 – 12	209	119	351 594	1 065 436	90
12 – 13	181	103	304 297	922 112	78
13 – 14	79	45	133 714	405 193	34
14 – 15	49	28	82 239	249 209	21
15 – 16	17	10	29 419	89 147	8
16 – 17	9	5	15 292	46 339	4
17 – 18	1	0	1 322	4 008	0
TOTAL	934	532	1 573 129	4 767 057	403

**Table 23**Morbidity effects of PM2.5 (number of cases/year or RADs/year)

# 4.4 Socio-economic cost

## 4.4.1 Results of socio-economic valuation

#### Central Estimate, PM<sub>10</sub>

The social costs in Sweden caused by health effects that can be linked to high annual ambient air concentrations of  $PM_{10}$  are estimated by adapting the socio-economic values of the considered health effects from available literature to the number of occurrences of the health effects as estimated in this study, see Table 24. In the central estimate on socio-economic costs calculated in this project, the exposure-response function varies for 'road', 'background' and 'other' sources. In the sensitivity analysis we then compare these costs with the costs given by using identical exposure response function for all sources, which is the current approach used by WHO.

LSIIIIale			
	Socio-economic cost of health Effect [SEK <sub>2005</sub> / case]	Health effects in 2005	Socio-economic cost [million SEK <sub>2005</sub> ]
Total Sweden			25 570 million SEK <sub>2005</sub>
Out of which:			
Value of prevented fatality (VSL/VPF) (11 years of prolonged life)	5 691 000	3390 excess death occurrences	19 292
Chronic Bronchitis	1 966 143	1340 excess cases	2 635
Hospitalisation, cardiology	42 738	581 excess cases	25
Hospitalisation, generic (respiration)	25 208	761 excess hospital admissions	19
RAD (age group 15-64)	1 325	1 570 516 excess RAD	2 081
RAD (other age groups)	476	3 188 624 excess RAD	1 518

 Table 24
 Annual socio-economic costs of high long term PM<sub>10</sub> levels in Sweden, 2005 - Central Estimate

As shown, the total annual socio-economic costs related to high  $PM_{10}$  levels is some 26 000 million SEK<sub>2005</sub>, and the absolute majority of these costs relate to loss of life years. This value can serve as a comparison with estimated financial costs for abating high concentrations of  $PM_{10}$ . In these calculations all the estimates from the literature are recalculated into Swedish Crowns at the value in 2005. This is done by adjusting the currencies with respect to Consumer Price Indices (CPI) and Purchase Power Parity (PPP). CPI is used to adjust the values given to year 2005 values while PPP is used to adjust for national differences. In Appendix C all the values from the literature are expressed in Swedish Crowns with the 2005 year value.

### Central estimate, PM<sub>2.5</sub>

The health effects from  $PM_{2.5}$  presented Table 25 in below translates into the following socio-economic value.

	Socio-economic cost of health Effect [SEK <sub>2005</sub> / case]	Health effects in 2005 [cases]	Socio-economic cost [million SEK <sub>2005</sub> ]
Total Sweden			23 341
Out of which:			
Value of prevented fatality (VSL/VPF) (11 years of prolonged life)	5 691 000	3 140 excess death occurrences	17 870
Chronic Bronchitis	1 966 143	934 excess cases	1 836
Hospitalisation, cardiology	42 738	403 excess cases	17
Hospitalisation, generic (respiration)	25 208	532 excess hospital admissions	13
RAD (age group 15-64)	1 325	1 573 129 excess RAD	2 084
RAD (other age groups)	476	3 193 928 excess RAD	1 520

# Table 25Annual sociao-economic costs of high long term PM2.5 levels in Sweden,<br/>2005 – Central Estimate

The socio-economic costs for high levels of  $PM_{2.5}$  are ~23 000 million SEK<sub>2005</sub> for the year 2005. Due to the difference in methodological approach when estimating the health effects, these values are not directly comparable to the socio-economic costs for  $PM_{10}$ , but as expected the socio-economic costs related to  $PM_{2.5}$  are very high compared to  $PM_{10}$ . Since  $PM_{2.5}$  is a part of  $PM_{10}$  these socio-economic costs should not be added to the socio-economic costs for  $PM_{10}$  presented earlier. This again confirms that a large portion of the socio-economic costs related to PM air pollution can be attributed to the smallest fractions of PM emissions.

The central estimates are based on the central values from the 2005 update of the ExternE project. The use of the ExternE project values is mainly due to reasons of comparability with other national and international calculations on health effects. In the table we indicate a VSL value of 5 691 000 SEK<sub>2005</sub> to be used for the valuation. This value is lower than other common estimates of VSL. This is mainly an effect of the adjustment of the VSL value for the fact that the expected life loss amounts to 11 years, as is the estimate in our study. The normal number of years lost when estimating a VSL value is ~40. The VSL estimate in our central estimate is not corrected for the respondents' time preferences (where future costs are valued less than costs taken today) since the origin of the value specifically indicates that annual payments should be made over 10 years, thereby inducing discounted values given by the respondents.

In accordance with the valuation approach recommended by the ExternE update from 2005, the results from the socio-economic valuation of high levels of  $PM_{10}$  are constituted out of three main components when applicable; Resource costs (costs for medical aid), Opportunity costs (loss in productivity) and Disutility (costs for discomfort etc) (see Chapter 3.3.3). The detailed results for each health end point are presented in Table 26.

	Resource costs [SEK <sub>2005</sub> / day]	Opportunity costs [SEK <sub>2005</sub> / day]	Disutility [SEK <sub>2005</sub> / day]	Duration [days]	Socio-economic cost [SEK <sub>2005</sub> / case]
Value of Statistical Life (VSL/VPF) (11 years of prolonged life)	n.a.	n.a.	5 691 000	n.a.	5 691 000
Chronic Bronchitis	n.a.	n.a.	1 966 143	n.a.	1 966 143
Hospitalisation, cardiology	5 592	849	565	6	42 738
Hospitalisation, generic (respiration)	3 342	849	565	5	25 208
RAD (age group 15-64)	n.a.	849	476	1	1 325
RAD (other age groups)	n.a.	n.a.	476	1	476

#### **Table 25**Unit values for considered health effects - central estimate.

However, it must be stressed that the valuation approach chosen for the value of avoided fatalities is based on valuation studies (WTP estimates) and expert opinions, supposedly due to the controversial nature of valuing pre-mature fatality in humans in economic terms.

#### Effect on labour force

The number of RAD:s affecting the Swedish 'working force' (age group 16 - 64 years) linked to high levels of  $PM_{10}$  concentrations are ~1,5 million, which equals ~4 000 full time employments (given 252 working days per year and an unemployment rate of 7.8 %). As a comparison, Volvo AB has approximately 28 000 employees in Sweden during 2007, out of which ~4 000 at Volvo Torslanda. The number of full time occupancy in Sweden during 2005 equals some 3,26 million full time employments (4,26 million persons in the age group 16 - 64 with an occupation, 30.6 average working hours per week) (www.scb.se). A quick comparison of these numbers gives at hand that a bit over 0,1 % of the Swedish working force with an occupation in the age group 16 - 64 are impeded from participating in their occupation due to high levels of PM concentrations.

For  $PM_{2.5}$ , the corresponding effect on the labour force is some 3 500 full time employments foregone due to high levels of  $PM_{2.5}$ , corresponding to ~0,1 % of the Swedish working force. Again, this effect on the labour force from  $PM_{2.5}$  is not to be added on top of the effect from  $PM_{10}$ .

### 4.4.2 Sensitivity Analysis

The central estimates presented in this study are based on an approach where different PM fractions are related to different risk estimates. The internationally accepted method is still to use one identical risk estimate (Exposure Response Function) for all sources of PM. The Table 26 below shows the results on socio-economic costs if the results in our study were to be calculated using identical exposure-response functions (ERF) for all sources of PM. These results can be compared to the results in Table 24 where we use source-specific ERF for the emissions sources.

	Socio-economic cost of health Effect [SEK <sub>2005</sub> / case]	Health effects in 2005	Socio-economic cost [million SEK <sub>2005</sub> ]
Total Sweden			24 654
Out of which:			
Value of Statistical Life (VSL/VPF) (11 years of prolonged life)	5 691 000	3 229 excess death occurrences	18 376
Chronic Bronchitis	1 966 143	1 340 excess cases	2 635
Hospitalisation, cardiology	42 738	581 excess cases	25
Hospitalisation, generic (respiration)	25 208	761 excess cases	19
RAD (age group 15-64)	1 325	1 570 516	2 081
RAD (other age groups)	476	3 188 624	1 518

# Table 26Annual socio-economic costs of high long term $PM_{10}$ levels in Sweden, 2005 - identical<br/>ERF mortality estimates for all PM fractions.

The comparison shows that by not using different risk estimates for different sources of PM, the effects on mortality will be underestimated by 161 occurrences per year (5 %), which in turn leads to an underestimation of the annual Swedish welfare costs of almost 1 billion  $SEK_{20005}$  when including morbidity costs as well.

In order to estimate a plausible range of socio-economic costs related to high levels of PM, some simple sensitivity analyses are performed. Matters of interest are what the results would be if health effect values from other studies were used and what effect a discounting of the VSL value would have on the total socio-economic cost. The VSL estimates quoted in OECD 2006 are not included in this sensitivity analysis since they relate to a larger loss in life expectancy than the 11 years we study. The VSL values given in OECD can be seen in the Appendix C.

First we estimate the effect of different values on VSL as shown in Table 27. These estimates are taken from ExternE (2005).

**Table 27**Low / High Estimates of VSL from ExternE (2005).

	Socio-economic cost related to PM <sub>10</sub> [million SEK <sub>2005</sub> ]
Low estimate VSL	16 789
High estimate VSL	93 101

The analysis shows that our central estimate is on the lower bound of the ExternE estimates.

Furthermore, time preferences is of general interest when valuing health effects. In economic valuation estimates time preferences are considered by introducing a discount rate, thereby reducing the value of future events. For the sake of comparison we discount the VSL values previously used with a 4 % discount rate, which is a common rate used in valuation of health effects related to air pollution, Table 29.

	Socio-economic cost related to PM <sub>10</sub>
	[million SEK <sub>2005</sub> ]
Low estimate VSL	13 320
Central estimate VSL	21 643
High estimate VSL	64 588

Table 28	Discounted Low / Central / High Estimates of ExternE (2005	5).
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From the discounting it can be seen that the values are sensitive to the choice of discount rate, which have been previously mentioned. But it is our opinion that the values we use from ExternE (2005) should remain undiscounted in the central estimate since they are valued with a method that allows the survey respondents to discount the values themselves.

For policy purposes, our central estimate of the annual socio-economic costs related to high PM levels in Sweden seems to be fairly robust. Even if discounted with a 4 % discount rate, the socio-economic cost would still stay well above 10 billion SEK<sub>2005</sub> annually.

# 4.5 Consequence analysis of reduced PM<sub>2.5</sub> concentrations

According to the revised EU directive (EU, 2007) a limit value of 20  $\mu$ g PM<sub>2.5</sub>/m<sup>3</sup> as a yearly mean is to be met in urban background air by 1 January 2020. In order to evaluate the positive health and socio-economic consequences of reaching this PM<sub>2.5</sub> level everywhere, calculations have been performed based on the relation between air concentrations in urban background and street level. Also the consequences of applying limit values of 10 and 15  $\mu$ g PM<sub>2.5</sub>/m<sup>3</sup> in a similar way have been investigated.

The calculated maximum  $PM_{2.5}$  concentration in urban background in 2005 was identified, and the corresponding street level concentration was estimated by using a factor of 1.4 based on monthly monitoring data from three towns in Sweden (www.ivl.se). The decrease in percent necessary to reach the certain limit value at street level was then applied to the urban background as well as regional background concentrations all over Sweden. The resulting difference in exposure levels, compared to the actual situation in 2005, are presented in Table 29. The same population data as used for 2005 has been applied.

PM <sub>2.5</sub> concentration	Difference in number of people exposed to PM <sub>2.5</sub> in 2005 and with a maximum limit value of			
[µg m <sup>-3</sup> ]	20	15	10	
0-5	563 060	2 954 300	8 596 040	
5-10	1 934 250	1 379 110	-4 238 720	
10-15	-2 369 000	-4 205 020	-4 228 930	
15-20	-128 280	-128 370	-128 370	

**Table 29**The difference between the number of people in Sweden exposed levels of total  $PM_{2.5}$  in<br/>2005 and by applying a limit value of 20, 15 and 10 µg/m³ respectively.

Figure 23 shows the number of excess deaths according to different levels of PM<sub>2.5</sub> concentrations.

The concentrations shown in the figure refer to the situation in street levels, while the exposure calculation is based on urban background levels. As can be seen there will be a 30% decrease in the number of excess deaths due to  $PM_{2.5}$  concentrations compared to the current situation if the limit value of 20 µg/m<sup>3</sup> is reached, corresponding to a general decrease of about 15 % in the  $PM_{2.5}$  concentrations.



**Figure 23** The number of excess deaths in Sweden due to different limit values of total PM<sub>2.5</sub>. The concentrations refer to the situation in street level.

The socio-economic benefits associated with a shift from the current situation of 2005 to these maximum limit values are given in Table 30 below. The results show that reaching our analysed limit values for PM would constitute substantial benefits for society via reduced socio-economic costs related to high levels of PM. Furthermore, the benefits continue to increase in an almost linear fashion down to maximum concentrations of  $10 \ \mu g/m^3$ . Moving from a max  $20 \ \mu g/m^3$  situation to max  $15 \ \mu g/m^3$  is only slightly more beneficial than moving from a max  $15 \ \mu g/m^3$  to a max  $10 \ \mu g/m^3$  situation. At the lowest concentrations analysed, there would only be some ~2 billions worth of PM<sub>2.5</sub>-related health effects left in Sweden, including some 261 fatalities.

	Socio- economic benefit	Avoided fatalities	Avoided Chronic Bronchitis (27+)	Avoided CHA	Avoided RHA	Avoided RAD (age 15 - 64)	Avoided RAD (other ages)
[µg/m³]	[million SEK <sub>2005</sub> ]	[cases / year]	[cases / year]	[CHA / year]	[RHA / year]	[RAD / year]	[RAD / year]
In 2005	0	0	0	0	0	0	0
Max 20	7 026	945	282	121	158	473 459	961 264
Max 15	15 143	2037	606	261	345	1 020 828	2 072 590
Max 10	21 401	2879	856	370	488	1 442 341	2 928 390

Table 30	Socio-economic	benefit from reaching	different PM <sub>2</sub> c limit values
1 abic 50		benefit from reaching	uniterent i my similit values.

### 4.6 Model evaluation

The model calculations have been evaluated in different scales. On the national level the output from the URBAN model, as concentrations for  $PM_{10}$  and  $PM_{2.5}$  respectively, has been compared to observed data. In order to evaluate the model on a local level and to be able to estimate the uncertainty of the results achieved, as regards both concentrations and exposure, comparisons have been made with calculations in the Greater Stockholm area.

A comparison was made between calculated and monitored  $PM_{10}$  concentrations in urban background (Figure 24). In general the calculated concentrations are higher, but the agreement is still reasonable. In a few towns the calculated concentrations are rather different from the monitored concentrations, but most of them lay within a limit of  $\pm 20\%$  (Figure 25). According to the result there is an overestimation of the calculated means in southern Sweden. In Malmö the  $PM_{10}$  concentration is of the same magnitude, or even lower, than in smaller towns in the south. Thus, the Urban model calculates a larger local increment for Malmö due to a larger population. However, this should not be the case according to measurements.



**Figure 24** Comparison between calculated and monitored annual means of PM<sub>10</sub> concentration in urban background from southern to northern Sweden.



Figure 25 Comparison between the calculated and monitored  $PM_{10}$  concentrations in percent in the towns shown in Figure 24. The dotted lines are  $\pm 20\%$  limitation.

Since the calculation of  $PM_{2.5}$  is based on the calculated  $PM_{10}$  concentrations these are also overestimated in southern Sweden. In Figure 26 a comparison between monitored, modelled and

calculated PM<sub>2.5</sub> concentrations in regional as well as in urban background air is shown. Except for the overestimation in the south the results achieved with the different methods agree quite well. However, the MATCH model somewhat underestimates the regional background levels (Andersson et al., 2008).



**Figure 26** Comparison of annual PM<sub>2.5</sub> means between monitored and calculated (1 RB =Interpolated EMEP model/measurements and 2 RB =MATCH model, concentrations in regional background (RB) as well as in urban background (UB) in Malmö, Göteborg and Stockholm.

An investigation of population exposure has been performed for the Stockholm area (SLB, 2007). According to the results presented in Figure 27 the percentage distribution of the population is in good agreement between the two studies. The result calculated with the URBAN model, compared to the Stockholm study, performs an underestimation of the exposure in the lowest exposure class (10-14  $\mu$ g/m<sup>3</sup>) with about 5 % of the total population exposure within the area. In the group 18-26  $\mu$ g/m<sup>3</sup> the two calculations agree well, while the URBAN model did not predict any exposure in the highest class (>26  $\mu$ g/m<sup>3</sup>). However, only 0.2 % of the people in Stockholm is exposed to these concentrations.



Figure 27 Percentage distribution of the population exposure of PM<sub>10</sub> concentrations in Stockholm.

A further comparison has been made between calculations of the local contribution to  $PM_{10}$  from the different sources based on the results achieved in this study and similar calculations for Stockholm (Johansson & Eneroth , 2007), see Figure 28.



Figure 28 Calculated local contributions to  $PM_{10}$  in Stockholm, comparison between this study and Johansson & Eneroth (2007).

In general this study is somewhat underestimating the different shares compared to the Stockholm study. Some of the calculated shares are not based on the same conditions why they are not totally analogous. In the Stockholm study the share "residential heating" includes all heating using wood, while in this study that share solely contains  $PM_{10}$  from wood burning used for domestic heating. The other part of the residential heating is possibly included in the share "rest", where also  $PM_{10}$  from power plants and all other sources are included. The traffic combustion shares are, however, in good agreement between the two studies, but the resuspended share in the Stockholm study is higher.

The local contribution to the total  $PM_{2.5}$  concentrations has also been calculated by IIASA (Amann et al., 2007), see Figure 29. A comparison between their results, the results achieved with the URBAN model and monitored data shows a similar pattern as above.



**Figure 29** Local contribution to the  $PM_{2.5}$  concentrations in urban background, monitored data ( $\Delta$  urban – regional background) and data from the URBAN and IIASA models.

# 5 Discussion

The general performance of the URBAN model has been discussed earlier (Sjöberg et al., 2007), and it has been shown that the uncertainty increases to a different extent depending on both latitude and longitude. This is due to the large variations in dispersion facilities over the country as well as between years.

The methodology of using an empirical model on a national basis, combined with advanced meteorological parameters, but still generating the results in a good geographical resolution, have not been used before Sjöberg et. al. (2004). However, a similar approach has been used focusing on greater city areas (Amann, 2007). The great advantage with the URBAN model, compared to ordinary dispersion models, is that an emission database is not needed. This eliminates the uncertainties associated with an emission database and the limited possibilities to capture unknown changes. The URBAN model reflects different large and local scale concentrations, through monitoring data and local scale meteorology (via the TAPM model).

The method used to estimate PM<sub>10</sub> concentrations in urban areas, based on the relation to the levels of NO<sub>2</sub>, has earlier been applied by i.e. UK (Muri 1998). The relationship was adjusted to Swedish conditions, reflecting both latitudinal and seasonal variations, see Figure 2. Comparison between the calculated PM<sub>10</sub> concentrations and monitoring data in urban background show a good agreement. Long range transport is the dominating source of the particles observed in Sweden. Since it is difficult to estimate this contribution it generally leads to a large uncertainty in particle modelling.

The assumption that the PM concentration is proportional to the number of people in a grid cell fails to capture the spatial patterns of roads, where PM emissions are significant. The comparison between this approach and modelling with a higher spatial resolution also shows similar population exposure results, see Figure 27 (SLB, 2007). The reason is possibly that not many people live next to roads. Thus, the assumption is therefore considered appropriate when calculating the PM exposure at a national level and in the resolution of 1\*1 km grid cells. Future development of the modelling methodology would be possible by incorporating an improved spatial pattern of emissions. It might also be possible to use concentration maps available in larger cities, and apply the dispersion pattern to the URBAN model.

The attempt to separate between different sources of  $PM_{10}$  is also connected with some uncertainties. When comparing with the local study for Stockholm (Johansson and Eneroth, 2007) the contribution from traffic combustion and residential heating coincides very well. However, the road dust part is underestimated by almost 1 µg/m<sup>3</sup>, while the so called "remaining" part is overestimated, probably caused by difficulties to allocate the road dust contribution properly. In the multivariate analysis some of the parameters used for separation of sources are possibly interacting, and therefore the use of this method needs to be further developed. Possible future improvements could be to apply weighting factors and/or to include more parameters governing these processes. Nevertheless, since it is assumed that the smallest fractions, and thus the combustion part, contribute largely to the health effects the method can be assumed to give a reasonably good result.

Environmental standards as well as environmental objectives are to be met everywhere, even at the most exposed kerb sites. However, for exposure calculations it is more relevant to used urban background data, on which available exposure-response functions are also based. The results from the urban modelling show that in 2005 most of the country had rather low  $PM_{10}$  urban background concentrations, compared to the environmental standard for the annual mean (40  $\mu$ g/m<sup>3</sup>). However, in some parts, mainly in southern Sweden, the concentrations were of the same

magnitude as the environmental objective (20  $\mu$ g/m<sup>3</sup> as an annual mean) for the year 2010. The majority of people, 90%, were exposed to annual mean concentrations of PM<sub>10</sub> less than 20  $\mu$ g/m<sup>3</sup>. Less than 1% of Swedish inhabitants experienced exposure levels of PM<sub>10</sub> above 25  $\mu$ g/m<sup>3</sup>. We have estimated that almost 3 400 deaths per year are brought forward due to exposure to local air pollution concentration at home, indicated by PM<sub>10</sub> levels above a cut off at 5  $\mu$ g/m<sup>3</sup> as an annual mean.

For calculation of  $PM_{2.5}$  concentrations the relation to levels of  $PM_{10}$  on a yearly basis has been used. In spite of this rather rough method the agreement was rather good when comparing calculated and monitored  $PM_{2.5}$  concentrations in Stockholm. However, in southern Sweden (Malmö, Göteborg) the accordance was quite bad. A similar pattern is also seen for the local  $PM_{2.5}$ contribution calculated by IIASA (2007). To achieve a more reliable estimation of the  $PM_{2.5}$  levels additional monitoring data is needed.

In quite a large part of the country the modelling results regarding  $PM_{2.5}$  show that the urban background concentrations in 2005 were in the same order of magnitude as the environmental objective (12 µg/m<sup>3</sup> as an annual mean for the year 2010) in a quite large part of the country. About 50% of the population was exposed to PM<sub>2.5</sub> annual mean concentrations less than 10 µg/m<sup>3</sup> while less than 2% experienced levels above 15 µg/m<sup>3</sup>. The number of excess deaths due to PM<sub>2.5</sub> exposure levels, using a cut off at 4 µg/m<sup>3</sup>, was estimated to about 3 100.

Assessment of health impacts of particle pollution is difficult since PM is a complex mixture where different components are very likely have different toxicity. However, due to the lack of enough evidence for differential quantification (Forsberg et al., 2005) we still have to assume the same relative risk per particle mass concentration regardless of source and composition. This may be a too conservative approach and unwise with respect to the implications for actions.

The cut off levels used in this study for  $PM_{10}$  and  $PM_{2.5}$  are rather arbitrary, since we do not exactly know the natural background levels nor the shape of the exposure-response association in different concentration intervals. There is no evidence of a specific toxicological threshold level shown to support a specific cut off level. The cut off levels used in the present report are lower than in most studies.

The conversion of exposure-response functions between  $PM_{10}$  and  $PM_{2.5}$  is quite common for mortality effects, but not very scientific. Usually the ratio 0.6-0.8 between  $PM_{2.5}$  and  $PM_{10}$  is used as the factor. If the effect is mainly related to  $PM_{2.5}$  this conversion factor may be relevant. If coarse particles are as important as fine, this down-scaling of effects is not motivated.

According to the literature we can assume that the impact on mortality of anthropogenic  $PM_{10}$  and  $PM_{2.5}$  respectively would be almost of similar size, while for respiratory morbidity the contribution of the coarse fraction may be greater. However, our actual estimates are a product of selected cut off levels and ER-functions, and do not fully reflect statements on impacts related to comparisons of  $PM_{10}$  and  $PM_{2.5}$ .

The assessment of health impacts using  $PM_{10}$  or  $PM_{2.5}$  as exposure indicators is most valid for the regional background particle pollution. At first, urban background PM is largely built up by secondary particles, where a large part originates from remote sources. Secondly, the most commonly applied exposure-response relations for long-term effects on mortality come from studies where such particles were important for the contrasts in exposure. Recent research has shown that within-city gradients in air pollution seem to be very important for health effects (Jerret et al, 2005; WHO, 2006a). However, particle mass concentration (as  $PM_{10}$  or  $PM_{2.5}$ ) is not a good indicator of vehicle exhaust levels. Street levels of  $PM_{10}$  may be a good indicator for traffic when there is a lot of road dust, in particular during winter and spring where studded tyres are used.

Nitrogen dioxide is on the other hand in most areas a good indicator of air pollution from the transport sector (cars, trucks, shipping). This does not mean that  $NO_2$  is very important as a causal agent behind the health effects related to air pollution. Even if the exhaust particles contribute most to the health impacts, the health effects from local-regional gradients in vehicle exhaust are likely to be much better studied using  $NO_2$  or  $NO_X$  as indicators, rather than using particle mass as  $PM_{10}$ . Thus, the assessment using  $NO_2$  (Sjöberg et al, 2007) is therefore a better indication of the magnitude of the mortality effects from traffic in Sweden, than the estimates for exhaust PM and road dust PM in this assessment.

The previous report (Sjöberg et al, 2007) estimated that more than 3 200 deaths per year are brought forward due to exposure to the local air pollution contribution, indicated by modelled nitrogen dioxide levels at home above a cut off at  $10 \,\mu\text{g/m}^3$  as an annual mean. In order to determine the total air pollution impact, it is probably justified to sum up almost all the 3 240 excess deaths per year attributed to PM<sub>10</sub> exposure in this study due to the regional background, wood smoke and the non-specified other sources with the deaths per year estimated in the previous report. Likewise, the effects of ozone could be added.

In a recent study similar calculations for Sweden were presented using particulate matter ( $PM_{10}$  or  $PM_{2.5}$ ) as the air pollution indicator (Forsberg et al, 2005b). In that health impact assessment, the impact of long-range transported pollutants was estimated to approximately 3 500 premature deaths annually, and the local contribution to urban levels of PM was estimated to result in around 1 800 deaths per year. However, the authors suggest that it was likely that the effect of particle emissions from local traffic was underestimated with the applied risk coefficients for PM from American cohort studies across regions.

This study estimates approximately 1 340 respiratory and cardiovascular hospital admissions due to the short-term effect of  $PM_{10}$  without any other cut-off than the one used for the annual mean values. This may seem to be a low number of admission in comparison with the estimated number of deaths, new chronic bronchitis cases and restricted activity days. However, for hospital admissions only the short-term effect on admissions can be estimated, and thus not the whole effect on hospital admissions following morbidity due to PM. The total yearly number of hospital admissions in persons that developed their disease due to air pollution exposure may well be 10-20 times higher. It would be valuable to also have morbidity indicators for other long-term effects of air pollution exposure than chronic bronchitis. Most of the excess cases are related to large numbers exposed to low-moderate urban background levels, and therefore current EU targets will have a minor effect.

From an economic perspective it is important to put the socio-economic costs into perspective and to discuss solutions that would ensure cost efficient measures to abate health effects from PM.

All in all, 3 400 premature fatalities, and a number of other health effects, are related to high levels of PM in 2005. The socio-economic cost estimate from these effects sums up to ~26 billion SEK<sub>2005</sub>. Furthermore, these high levels of PM will put some 0.1% of the Swedish working force out of their daily occupation. As a comparison, the number of fatalities due to road accidents in Sweden was ~400 in 2005, the Swedish Gross Domestic Product (GDP) was 2673 billion SEK<sub>2005</sub> and the corresponding number of lost employment equals the amount of employees at the Volvo car manufacturing facility in Torslanda, Göteborg.

The distribution of these socio-economic costs are predominantly attributed to medium levels of PM air pollution and to small fractions of PM ( $PM_{2.5}$ ), suggesting that the largest potential for costefficient abatement measures will be found in a reduction of the smallest fractions. However, it has not been within the scope of our work to study specific abatement measures and their net benefit to society. Furthermore, when calculating marginal socio-economic benefits (calculated as avoided socioeconomic costs) that would be the result if maximum limit values were applied for  $PM_{2.5}$  it can be seen that reaching the limit value of 20  $\mu$ g/m<sup>3</sup> at street level would reduce welfare losses to society related to PM exposure by some 7 billion SEK<sub>2005</sub>. The socio-economic benefits continue to increase substantially when decreasing the maximum limit value even further. In a later study these results can be compared to the socio-economic costs of implementing emission abatement measures in i.e. the transport sector.

As a final remark, the importance of which ERF to use must be stressed from an economic as well as from a health effect perspective. As shown in the sensitivity analysis of the socio-economic costs, the choice of ERF corresponding to the recommended values from WHO for  $PM_{10}$  will underestimate the effects on premature fatality by ~160 cases. The underestimation of socio-economic costs equals ~1 billion SEK<sub>2005</sub>.

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www.scb.se

www.sos.se

www.vv.se

# Appendix A

The maximum and minimum spreads of the ratios  $PM_{10}/NO_2$  (local contributions in urban background air) for each season.





# Appendix B

## Counties included into the Road Administration Regions County and county code

01 Stockholm

- 24 Västerbotten
- 25 Norrbotten
- 03 Uppland
- 04 Södermanland
- 05 Östergötland
- 06 Jönköping
- 07 Kronoberg
- 08 Kalmar
- 09 Gotland
- 10 Blekinge
- 12 Skåne
- 13 Halland
- 14 Västra Götaland
- 17 Värmland
- 18 Örebro
- 19 Västmanland
- 20 Dalarna
- 21 Gävleborg
- 22 Västernorrland
- 23 Jämtland

Road administration regions	County included in each region				
Skåne	12				
West	14	13	17		
Southeast	10	6	8	7	5
Stockholm	1	9			
Mälardalen	4	3	18	19	
Central north	20	21	23	22	
North	24	25			
# Appendix C

# Valuation Studies expressed in Swedish Crowns [SEK<sub>2005</sub>]

Values on Mortality (VSL) given in OECD 2006

[SEK <sub>2005</sub> ]	VSL low	VSL high	
Hammit 2000	40480000	94450000	
Alberini et al. 2004	15360000	49159000	
	9217000	37894000	
Krupnick et al. 1999	2164000	4328000	
Markandya et al. 2004	11764000	27449000	
	6862000	7843000	
	8823000	18626000	
Chilton et al. 2004	2941000	14705000	

 Values on Mortality (VSL) given in ExternE (www.externe.info)

 [SEK<sub>2005</sub>]
 Low / Central
 High

 Value of Statistical Life (VSL)
 10886000
 34252000

Values on Mortality (VPF) given in Chilton et al. 2004

[SEK <sub>2005</sub> ]	Low / Central	High	
Value of Prevented Fatality (VPF) from reduced levels of air pollution*	3711000	16979000	
Value of Prevented Fatality (VPF) in road accidents* *	19204000	19204000	

\* The value is derived from the value of a one year gain in life expectancy and assumes 40 remaining life years and a 0 % discount rate.

\*\* Value depicted from a British study and quoted in Chilton et al. 2004

## Values on Mortality (VOLY) given in OECD 2006

[SEK <sub>2005</sub> ]	VOLY
Chilton et al. 2004	424000
Markandya et al. 2004	645000

Values on Mortality (VOLY) given in ExternE (www.externe.info)

[SEK <sub>2005</sub> ]	VOLY Low	VOLY Central	VOLY High	
Value of Life Year Lost	282000	517000	2328000	
(VOLY)				

#### Values on Mortality (VOLY) in Chilton et al. 2004

[SEK <sub>2005</sub> ]	VOLY Low	VOLY Central / High
Value of Life Year Lost	93000	424000
(VOLY)		

#### Values on morbidity given in OECD 2006

	Study quoted [SEK <sub>2005</sub> ]					
Type of Illness (morbidity)	Ready et al. 2004	ExternE 1998	Maddison 2000			
Hospital admission for treatment of respiratory disease	lospital admission for 5070 reatment of espiratory disease		n.a.			
3 days spent in bed with respiratory illness	1604	776	2018			

# Values on morbidity given in ExternE (www.externe.info)

Health related effect	[SEK <sub>2005</sub> ]
Hospitalisation, generic (respiration)	3342
Hospitalisation, cardiology	5592
WTP to avoid hospital admissions*	4522
Productivity loss of absence from work	849

### Values on morbidity given in Chilton et al. 2004

	[SEK <sub>2005</sub> ] low	[SEK <sub>2005</sub> ] high
Value of a one year gain in life expectancy in normal health	93000	424000
Value of avoiding a respiratory hospital admission	20000	109000

#### Summary of Morbidity valuation, values given in SEK<sub>2005</sub>

eannary er i	lion brancy i	aldation, van		021(2005			
	3 RAD	RAD [sek /	RHA [sek	CVA [sek	Chronic	WTP to	Productivity
	[sek /	day]	/day]	/day]	Bronchitis	avoid	loss of
	day]				[sek /case]	hospital	absence
						admissions	from work
						[sek / day]	[sek /day]
METHOD	Disutility	Disutility	Resource	Resource	Disutility &	Disutility	Opportunity
	-		cost	cost	resource		cost
					costs		
ExternE2005	1 428	476	3 342	5 592	1 966 143	565	849
Pearce D	1 604	525				624	
2000	1 004	555				034	
Maddison	2 0 1 8	673					
2000	2 010	073					
Ready et al.	1 604	535				634	
2004	1 004	000				004	
ExternE 1998	776	259					
Chilton et al						20 126	
2004						20 120	
Maca Scazny							2 4 5 2
2004							2 452
BAQ-Asia					3 503 897		
2006					0 000 007		
BeTa							
database			44 703	173 124	1 752 248		
2002							