Quantification of population exposure to NO$_2$, PM$_{2.5}$ and PM$_{10}$ and estimated health impacts

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SWEDISH ENVIRONMENTAL PROTECTION AGENCY

NATIONAL ENVIRONMENTAL MONITORING COMMISSIONED BY THE SWEDISH EPA

UMEÅ UNIVERSITET

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Report title and subtitle
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Keywords for location (specify in Swedish)
Sweden (Sverige)

Keywords for subject (specify in Swedish)
NO₂, PM₂.₅, PM₁₀, particles, air quality, population exposure, health impact assessment, risk assessment, socio-economic valuation (NO₂, PM₂.₅, PM₁₀, partiklar, luftkvalitet, befolkningsexponering, hälsoeffekter, samhällsekonomiska konsekvenser)

Period in which underlying data were collected
2015

Summary
In this study population exposure to annual mean concentrations of NO₂, PM₁₀ and PM₂.₅ in ambient air has been quantified, and the health and associated economic consequences have been calculated based on these results. To allow application of known exposure-response functions for assessment of health effects this study exclusively focus on regional and urban background concentrations. Nearly the entire Swedish population was exposed to concentrations below the environmental standards, and 97 %, 78 % and 77 % was exposed to concentrations below the respective specifications of the environmental objective for NO₂, PM₁₀ and PM₂.₅. The highest concentrations of NO₂ and PM were found in the most polluted central parts of our largest cities.

Excess mortality was used as the main health indicator. The total number of excess deaths due to air pollution exposure was estimated to be 7600 in 2015. Of these, we estimated that approximately 3600 deaths per year were associated with exposure to regional background, 900 from local wood burning, 215 due to road dust and approximately 2850 deaths per year from vehicle exhaust.

Based on these results the health impacts from exposure to NO₂ and PM₂.₅ were conservatively estimated to cause socio-economic costs of ~56 billion Krona in 2015. Just absence from work and studies was estimated to cause socio-economic costs of ~0.4% of GDP in Sweden.

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Summary

Air pollution concentrations in Swedish cities are among the lowest in Europe. Despite this, health impacts due to exposure to ambient air pollution is still an important issue and the concentration levels, especially of nitrogen dioxide (NO_2) and particles (PM_{10} and $PM_{2.5}$), occasionally exceed the air quality standards at street level in many urban areas.

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 2015. The population exposure to annual mean concentrations of NO_2, 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.

To allow application of known exposure-response functions for assessment of health effects this study exclusively focus on regional and urban background concentrations. Roadside concentrations are not addressed here. The results from this study show that background concentrations of the examined pollutants in 2015 were overall low, well below the environmental standards in most parts of the country. The background concentrations were also below the environmental objective for all examined pollutants, with the exception of a small stretch along the Swedish west coast and Skåne, where the particle concentrations were of the same magnitude as the environmental objective. It should be noted that a slight over-estimation of $PM_{2.5}$ may occur in coastal regions due to the presence of sea salt which may affect the $PM_{2.5}$/PM_{10} ratio used to calculate $PM_{2.5}$ in this study.

Nearly the entire Swedish population was exposed to concentrations below the environmental standards, and 97%, 78% and 77% was exposed to concentrations below the respective specifications of the environmental objective for NO_2, PM_{10} and $PM_{2.5}$. Exposure to the highest concentrations was found in the most polluted central parts of our largest cities.

Comparing the results from this study to the 2010 assessment shows a slight increase in mean population exposure to NO_2 and PM. For NO_2, we also find a slight increase in the percentage of the population exposed to concentrations above the environmental objective. For PM, exposure to concentrations above the environmental objective was instead found to have decreased with up to 5%. Particle concentrations show a decreasing trend in Sweden, resulting in reduced exposure to the highest PM concentrations and an increased exposure to concentrations just below the environmental objectives. The slight increase in mean population exposure to PM can be explained by a growing population and ongoing urbanization, resulting in more people exposed to relatively high PM concentrations in the urban centres. While the contribution of local sources is minor for the smallest PM, it makes up the major part of NO_2 concentrations in urban areas. The slight increase indicated for NO_2 exposure is thus primarily connected to increased local emissions of NO_2, due to, for example, increasing traffic and use of diesel vehicles. This, in combination with the ongoing urbanization, results in a growing number of people living in areas with higher concentrations.

Excess mortality is usually the main health indicator. We estimate approximately 3600 deaths per year associated with exposure to regional background (long-distance transported) concentrations of $PM_{2.5}$. On average each premature death represents over 11 years of life lost. The total exposure to $PM_{2.5}$ was recently in an EU report estimated to cause just over 3700 deaths per year in Sweden when no differences between sources and no threshold for effects were assumed. We assume that

locally emitted particles (road dust, wood smoke and exhaust particles) have different effects on mortality, but face problems to find specific exposure-response functions. This is even more striking regarding effects on morbidity. Acknowledging the uncertainty, we estimate particles from local wood burning to cause more than 900 deaths per year, but here the exposure estimate is very uncertain. For road dust we calculate 215 deaths per year based on the exposure-response function from a Swedish study. We believe that the impact on mortality from locally emitted vehicle exhaust including particles is best indicated by exposure-response functions for within city gradients in NO_2, which also could include effects of NO_2 itself. We estimate approximately 2850 deaths per year from vehicle exhaust, but using alternative risk functions would result in 15-30% reduced estimates.

The total number of excess deaths due to air pollution exposure was estimated up to 7600 in 2015. The increase in comparison to the 2010 estimate is not due to changes in estimated exposure, but resulting from a revision of assumed exposure-response relations. If we for 2010 had assumed the urban NO_2 contribution to increase mortality without any cutoff, we would have estimated almost the same impact on mortality associated with NO_2 as in 2015.

Finally, the health impacts from exposure to NO_2 and $PM_{2.5}$ can be conservatively estimated to cause socio-economic costs of ~56 billion Krona in 2015. Just absence from work and studies can be estimated to cause socio-economic costs of ~0.4% of GDP in Sweden.

Sammanfattning

Halterna av luftföroreningar i svenska städer är bland de lägsta i Europa. Trots detta överskrider föroreningshalterna i gaturum, särskilt kvävedioxid (NO_2) och partiklar (PM_{10} och $PM_{2.5}$), i vissa fall de miljökvalitetsnormer (MKN) för människors hälsa som gäller för utomhusluft.

På uppdrag av Naturvårdsverket har IVL Svenska Miljöinstitutet och Yrkes- och miljömedicin vid Umeå universitet kvantifierat den svenska befolkningens exponering för halter i luft av NO_2, $PM_{2.5}$ och PM_{10} för år 2015, beräknat som årsmedelkoncentrationer. Även de samhällsekonomiska konsekvenserna av de uppskattade hälsoeffekterna har beräknats.

För att kunna applicera kända dos-responsfunktioner för bedömning av hälsoeffekter från exponering för luftföroreningar har vi i den här studien fokuserat på halter i urban och regional bakgrundsmiljö. Halter i gaturum inkluderas inte. Resultaten visar att halter av de undersökta föroreningarna i bakgrundsluft år 2015 generellt var låga, med halter långt under respektive MKN i större delen av landet. Föroreningskoncentrationerna i bakgrundsluft låg också långt under preciseringarna i miljökvalitetsmålet *Frisk Luft* för alla undersökta föroreningar, med undantag för en liten sträckning längs den svenska västkusten och Skåne, där partikelkoncentrationerna låg på samma nivå som miljökvalitetsmålet. Det bör noteras att $PM_{2.5}$-halterna kan vara något överskattade i kustområdena på grund av havssalt, vilket kan påverka den $PM_{2.5}/PM_{10}$-kvot som används för att beräkna $PM_{2.5}$ i denna studie.

Nästan hela den svenska befolkningen exponerades för koncentrationer under MKN, med 97 %, 78 % och 77 % utsatta för koncentrationer även under miljökvalitetsmålets preciseringar för NO_2, PM_{10} och $PM_{2.5}$. Exponeringen för de högst koncentrationerna sker i de mest centrala delarna av våra största städer.

Jämförelse med bedömningen 2010 visar en svag ökning i medelexponeringen för NO_2 och PM för Sveriges befolkning. För NO_2 fann vi även en svag ökning av andelen av befolkningen som exponerades för halter över miljökvalitetsmålets preciseringar. För PM noterade vi istället en minskning på upp till 5 % av andelen av befolkningen som exponerades för halter över miljökvalitetsmålets preciseringar. Partiklar visar en trend mot lägre halter, vilket innebär en minskning i exponering för de högsta halterna, samtidigt som exponeringen för halter strax under miljömålets precisering ökar. Den något ökande medelexponeringen för PM kan förklaras med att befolkningen växer och urbaniseringstrenden medför att fler utsätts för de relativt höga halterna i städernas centrum. Medan lokala källor har begränsat inflytande på de minsta partklarna, bidrar de med huvuddelen av NO_2, speciellt i städer. Den något högre exponeringen för NO_2 är därmed främst kopplad till en ökning av lokala källor, som till exempel mer trafikarbete och fler dieselfordon. Detta, i kombination med urbaniseringen, medför en ökning i antal människor exponerade för de högre halterna i städernas centrala delar.

Förhöjd dödlighet är oftast det viktigaste ohälsomåttet. Vi uppskattar att omkring 3600 dödsfall per år kan tillskrivas exponeringen för den regionala bakgrundshalten (långdistanstransport) av $PM_{2.5}$. I genomsnitt motsvarar varje dödsfall en förlust av drygt 11 levnadsår. Den totala exponeringen för $PM_{2.5}$ i Sverige beräknades nyligen i en EU-rapport leda till strax över 3700 dödsfall per år om riskökningen är lika för alla källor och haltbidrag. Vi antar att lokalt genererade partiklar (vägdamm, vedrök och avgaspartiklar) har olika effekt per haltökning på dödligheten, men har problem att finna specifika samband som publicerats. Avsaknaden är ännu mer tydlig beträffande effekterna på sjuklighet. Medvetna om osäkerhetsfaktorerna uppskattar vi att exponeringen för partiklar från vedeldning ger upphov till över 900 dödsfall per år, men i detta fall är exponeringsuppskattningen särskilt osäker. Utifrån exponerings-responssambandet i en svensk studie beräknas vägdamm ligga bakom 215 dödsfall per år. Vi tror att effekten på dödligheten till följd av lokalt genererade fordonsavgaser bäst beräknas med exponerings-responsfunktionen för inomstadsvariationen i kvävedioxid, vilken också kan inkludera effekter av kvävedioxid i sig. Vi uppskattar att bilavgaserna leder till cirka 2850 dödsfall per år, men alternativa riskfunktioner skulle resultera i 15-30% lägre skattningar.

Det totalt beräknade årliga antalet dödsfall till följd av luftföroreningarna uppskattas till 7600 för 2015. Den betydande ökningen jämfört med beräkningen för 2010 förklaras inte främst av ökad exponering, utan beror på att antaganden om relationerna mellan exponering och ökad dödlighet har reviderats. Ifall vi i tidigare rapport för 2010 hade antagit att hela det lokala tillskottet av NO_2 påverkar mortaliteten utan någon tröskel, så hade antalet beräknade dödsfall relaterade till NO_2 blivit nästa lika högt som för 2015.

Hälsoeffekter från förhöjda halter av NO_2 och $PM_{2.5}$ kan med konservativa bedömningar skattas orsaka samhällsekonomiska kostnader på ca 56 miljarder svenska kronor år 2015. Enbart produktivitetsförluster från sjukfrånvaro kan uppskattas orsaka samhällsekonomiska kostnader på ca 0,4 % av BNP i Sverige.

1 Introduction

Despite the successful work to improve the outdoor air quality situation in Sweden (SOU 2016:47; Naturvårdsverket, 2018a) by reducing emissions from both stationary and mobile sources, the health impacts of exposure to ambient air pollution is still an important issue. As shown in many studies during recent years, the concentration levels, especially of nitrogen dioxide (NO_2) and particles (PM_{10} and $PM_{2.5}$), in many areas exceed the air quality standards and the impact on human health, due to exposure to these pollutants, is still significant (Grennfelt et al., 2017; Fredricsson et al., 2017; WHO, 2015; WHO, 2016a).

Within the framework of the health-related environmental monitoring programme, conducted by the Swedish Environmental Protection Agency (Swedish EPA), a number of different activities are performed to monitor health effects that may be related to environmental factors. As a part of this programme 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 NO_2, PM_{10} and $PM_{2.5}$ in ambient air in Sweden for the year 2015. Also the health and associated economic consequences have been calculated based on these results.

2 Background

Emission reductions regarding both NO_2 and particles have been on the agenda for the past few decades and progress have been made, but urban areas are growing and more people are moving to cities where the air pollution load in general is higher than in rural areas.

Environmental conditions and trends have been monitored for a long time in Sweden. Already in 1990/91 (winter half year, October-March) a study was performed, within the Swedish EPA´s investigation of the environmental status in the country, concerning the number of people exposed to ambient air concentrations of nitrogen dioxide (NO_2) in excess of the ambient air quality guidelines valid at that time (Steen and Cooper, 1992). Similar calculations were later made for the conditions during the winter half years 1995/96 and 1999/2000 using the same technique (Steen and Svanberg, 1997; Persson et al., 2001), and the results indicated a slight decrease in the excess exposure.

In 2007 a study of NO_2 exposure in Sweden for the year 2005 was conducted using a statistical model for air quality assessment, the so-called URBAN model, which can be used to estimate urban air pollution levels in Sweden and quantify population exposure to ambient air pollutants (Persson et al., 1999; Persson and Haeger-Eugensson, 2001; Haeger-Eugensson et al., 2002; Sjöberg et al., 2004; Sjöberg et al., 2007). Later the method was further developed to include the population exposure to PM_{10} and $PM_{2.5}$ (Sjöberg et al., 2009). Using the calculated population exposure to NO_2, PM_{10} and $PM_{2.5}$ the health consequences and socio-economic costs were calculated for 2005 (Sjöberg et al., 2007; Sjöberg et al., 2009).

The same method, using the URBAN-model, was used to calculate the exposure, health impact and socio-economic costs of NO_2, PM_{10} and $PM_{2.5}$ concentrations in Sweden for 2010 (Gustafsson et al., 2014). In Table 1 the main results from the 2005 and 2010 studies are presented.

Table 1 Main results from the 2005 and 2010 exposure studies (Sjöberg et al., 2007, Sjöberg et al., 2009, Gustafsson et al., 2014)

		2005	2010
Total population (no. of inhabitants)		8 899 724	9 546 546
Mean population weighted exposure ($\mu g/m^3$)	NO_2	6.3	6.2
	PM_{10}	13	12
	$PM_{2.5}$	9.8	8.6
Percentage of the total population exposed to concentrations above the environmental objective	NO_2 (20 $\mu g/m^3$)	2.3%	2.7%
	PM_{10} (15 $\mu g/m^3$)	38%	25%
	$PM_{2.5}$ (10 $\mu g/m^3$)	49%	28%
Percentage of the total population exposed to concentrations above the environmental quality standard	NO_2 (40 $\mu g/m^3$)	0%	0%
	PM_{10} (40 $\mu g/m^3$)	0.4%	0.3%
	$PM_{2.5}$ (25 $\mu g/m^3$)	0%	0.6%

The results from the previously presented urban modelling showed that most of the country had concentrations of NO_2, PM_{10} and $PM_{2.5}$ in ambient air well below the environmental standards for annual means (Sjöberg et al., 2007; Sjöberg et al., 2009; Gustafsson et al., 2014). Only in the larger urban centers, concentrations were reaching the same magnitude as the environmental standards. In parts along the west coast, concentrations approaching the long-term environmental objective were noted, especially for PM. The calculations showed that more than 99% of the population were exposed to concentrations below the environmental standards. A clear positive development towards a larger proportion exposed to concentrations also below the environmental objectives was presented in the reports. Population weighted mean concentrations were found to remain relatively stable with a slight decrease in PM. Sjöberg et al (2007) also presented a trend analysis between 1990 and 2010 showing a continuous reduction in NO_2 exposure. During the same period the annual mean of NO_2 decreased by almost 40%, which was attributed to a reduction of the total NO_X emissions in Sweden (Naturvårdsverket, 2017).

2.1 Aim of this study

The aim of this study is to update the calculated exposure to yearly mean concentrations of NO_2, PM_{10} and $PM_{2.5}$ on a national scale for 2015, and to assess the associated long-term health impact as well as the related economic consequences. The results are also compared to earlier studies to assess trends. In order to enable comparison with previously calculated numbers, the same calculation methods as in the latest studies are applied where possible.

3 Methods

The method applied for calculation of ambient air concentrations and exposure to air pollutants has been described earlier (Sjöberg et al., 2007; Sjöberg et al., 2009). 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 in the urban background. To calculate the exposure across Sweden, regional background concentration of the NO_2, PM_{10} and $PM_{2.5}$, as well as population distribution, are needed in addition to the calculated urban background air concentrations. The concentration

patterns of NO_2, PM_{10} and $PM_{2.5}$ over Sweden were calculated with a 1x1 km grid resolution (section 3.1, 3.2 and 3.3). PM_{10} and $PM_{2.5}$ were calculated both as total annual means and separated for different source contributions (section 3.4).

The quantification of the annual means of population exposure to NO_2, PM_{10} and $PM_{2.5}$ was based on comparisons between the pollution concentrations and the population density. Like the calculated air pollutant concentrations the population density data had a grid resolution of 1x1 km (section 3.5). By over-laying the population grid to the air pollution grid the population exposure to a specific pollutant was estimated for each grid cell (section 3.6).

To estimate the health consequences, exposure-response functions for the long-term health effects were used, together with the calculated NO_2 and PM exposure (section 3.7). For calculation of socio-economic costs, results from economic valuation studies and other cost calculations were used (section 3.8). These cost estimates were combined with the estimated quantity of health consequences performed in this study to give the related total socio-economic costs of NO_2 and PM concentrations in ambient air during 2015.

3.1 NO_2 concentration calculations

The NO_2 concentration was calculated based on i) regional background levels, and ii) local source contributions to the urban background concentrations. For each urban area the contribution from regional background NO_2 concentration was calculated from the background grid, and subtracted from the urban NO_2 concentration to avoid double counting. Hence, only the additional local NO_2 concentration (on top of the background levels) in urban areas was distributed.

3.1.1   Regional background

A national grid (1 x 1 km) representing the regional background concentration of NO_2 was calculated by interpolating measurement data from regional background sites. For 2015, 34 sites with monthly regional background data were used. 18 of these sites were monitored by the national air quality monitoring network within the Swedish environmental monitoring programme (Naturvårdsverket, 2018b), while the remaining 14 were monitored within The Swedish Throughfall Monitoring Network (http://krondroppsnatet.ivl.se).

The background grid was calculated for two-month periods during the year to account for seasonal variations in the NO_2 concentration. Dividing the year in two-month periods was deemed an appropriate time resolution as it gave a representation of the seasons without increasing the computational time for the calculations too much. At the end, an annual background map was compiled based on the results calculated from the 6 interpolated bimonthly maps, see Figure 1.
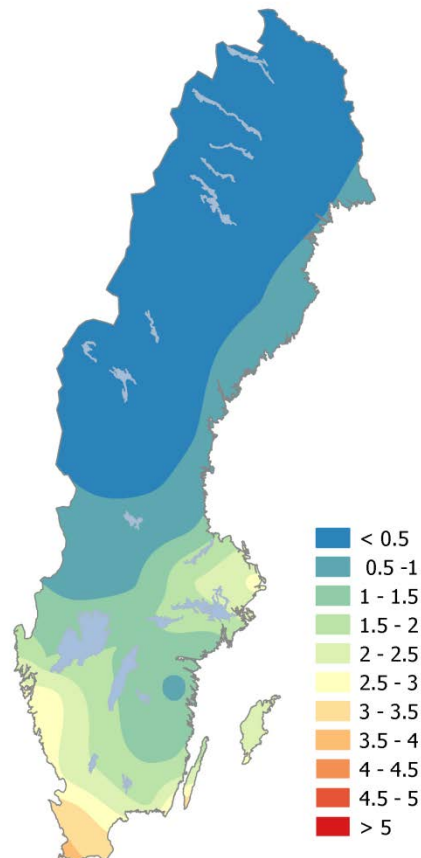
Figure 1   2015 annual mean regional background concentrations of NO_2 in Sweden ($\mu g/m^3$).

3.1.2   Urban background

The urban (local) contribution to NO_2 was calculated using the URBAN model, as described by Sjöberg et al. (2007). The distribution of the locally produced NO_2 in urban background air within cities was estimated based on the area of the city, where the grid cell within this area with the highest number of inhabitants was assigned the highest concentration of NO_2. Each grid cell within the city boundaries was then given a NO_2 concentration proportional to the number of inhabitants in each respective grid cell. The calculated concentrations of air pollutants are valid for the similar height above ground level as the input data (4-8 m) in order to describe the relevant concentrations for human exposure.

In the previous population exposure assessment for 2010 (Gustafsson et al., 2014), the method for distributing the urban background concentrations differed as information of the spatial extent was not available for the majority of the urban areas. Urban background was then distributed in a bell shaped pattern, assuming a decreasing gradient from the town center towards the regional background areas. The current method increases the accuracy of the spatial distribution of the urban background pollutant concentrations, but in order to ensure that the change of method does

not prevent comparison between this and the previous studies, a comparison between the current method and the previous was carried out based on the 2010 dataset. The results indicated that the new method slightly increased the exposure, but that the effect fell within the uncertainty limits of the data, and the change in method is thus not likely to influence the exposure assessment.

The total NO_2 concentrations were then calculated by adding the urban contribution to the regional background NO_2 concentrations for each grid cell.

3.2 PM$_{10}$ concentration calculations

3.2.1 Regional background

Monitoring of particles (PM$_{10}$ and PM$_{2.5}$) in regional background air is carried out at four sites in Sweden, within the national environmental monitoring programme financed by the Swedish Environmental Protection Agency (data from 2015 hosted by www.smhi.se). Possibilities to produce a realistic geographical distribution of PM$_{10}$ and PM$_{2.5}$ concentrations over Sweden based only on results from these stations are thus limited. Therefore, calculated distribution patterns by the mesoscale dispersion model EMEP (2012) were used, in combination with the existing monitoring data from the EMEP monitoring network. The calculated regional background concentrations used in this study are assumed to be long-distance transported particles and in coastal areas with a contribution of sea salt.

In order to separate the regional and urban/local PM$_{10}$ contributions, it was necessary to divide the regional background concentrations into two-month periods. This was done by using data for the four monitoring sites and applying similar conditions between the annual and monthly distribution of the calculated PM$_{10}$ concentrations from the EMEP model. The annual background map of PM$_{10}$ was compiled based on the results calculated from the 6 bimonthly interpolated maps, see Figure 2. The area with elevated concentrations of PM$_{10}$ in the northwest part of Sweden is caused by the results from the EMEP model indicating a strong increase in this area, primarily during July and August. The origin and accuracy of this irregularity has not been determined. It cannot be connected to any larger volcanic event and there are no indications that other potential sources, such as unusual shipping activity or wind patterns causing high air borne sea salt content, are the source. However, as this mountainous area is very sparsely inhabited (no inhabitants in the yellow area, 37 in the light green, and less than 300 in the darker green), the effect in the exposure assessment is negligible.

■	< 2
■	2 - 4
■	4 - 6
■	6 - 8
■	8 - 10
■	10 - 12
■	12 - 14
■	14 - 16
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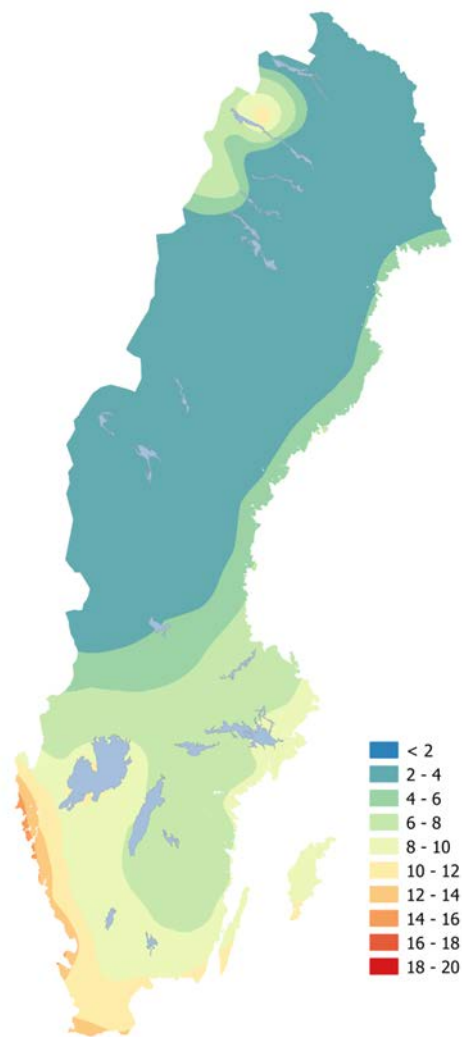
Figure 2 Annual mean regional background concentrations of PM_{10} in Sweden in 2015 (the EMEP model in combination with monitoring data), unit $\mu g/m^3$.

3.2.2   Urban background

The urban background concentration of PM_{10} was calculated by using the relationship NO_2/PM_{10} in urban background air for the year 2015 (see further Sjöberg et al., 2009; Chapter 3.1.2). To reflect the seasonal variation in the particle load the calculated yearly means were based on concentrations calculated with a bimonthly resolution.

In order to derive urban background concentrations of PM_{10}, the PM_{10}/NO_2 ratio for the stations providing data of both PM_{10} and NO_2 for the years 2005-2015 was used. For data from these stations, regional estimated background concentrations of NO_2 and PM_{10} were subtracted, and ratios of PM_{10}/NO_2 for the remaining local contribution were derived and analysed with respect to

the latitude. In previous reports, this has been done based on bimonthly means, but due to data limitations caused by a reduced number of urban background stations providing data for both PM_{10} and NO_2, a yearly mean latitude dependent ratio was used instead this time, see Figure 3. As the exposure assessment is based on yearly means it will not be affected by this change of method. It may, however, partly affect the seasonal source apportionment of the PM_{10} compared to the previous exposure assessments. Compared to the bimonthly differences calculated in the previous report (Gustafsson et al., 2014), using a yearly mean would slightly increase the wintertime PM_{10} and reduce the summertime PM_{10} concentrations. This effect would likely be more pronounced in the south compared to the north. It was not statistically relevant to calculate a standard deviation of the ratios due to the low data coverage.
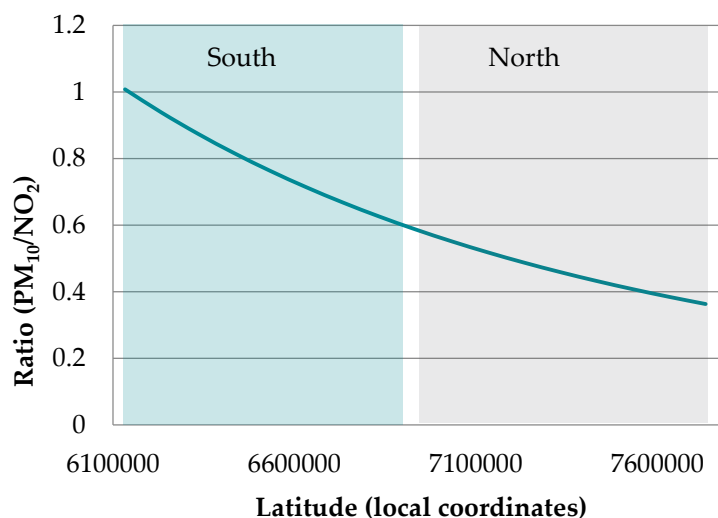
Figure 3   Latitudinal variation of the function PM_{10}/NO_2 based on the locally developed contribution to the concentrations in urban background air.

3.3 $PM_{2.5}$ concentration calculations

Based on the calculated PM_{10} concentrations, $PM_{2.5}$ in regional background and local source contributions to the urban background concentrations were calculated. 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.

3.3.1   Regional and urban background

The estimation of the $PM_{2.5}$ concentrations in Sweden was performed using a ratio relation between monitored $PM_{2.5}/PM_{10}$ since 2000 (data from www.smhi.se). The ratio varies with type of site location, from lower values in city centers 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 regional background, central urban background and suburban background (a mean between the two others) conditions (Table 2). This is a rough estimate as the ratio is likely to vary between years and with season, and for regional background the available monitoring data was very limited for 2015 with only two stations, Bredkälen and Råö, within the national environmental monitoring programme and one site, Asa, with intermittent measurements,

measuring both PM_{10} and $PM_{2.5}$ for the entire year. The station Råö is located on the sea front only a few meters away from the water, and is thus influenced by sea salt. As sea salt contribute more to the PM_{10} fraction than to the $PM_{2.5}$ fraction the $PM_{2.5}/PM_{10}$ ratio at Råö were deemed not to be representative for the rest of the country. With only two stations left, with calculated $PM_{2.5}/PM_{10}$ ratios of 0.65 (Bredkälen) to 0.75 (Asa), the decision was made to use the same ratio (0.8) as used in the 2005 and 2010 assessments, this to make the studies comparable. It should be noted that a slight over-estimation of $PM_{2.5}$ may occur in coastal regions due to the effect of sea salt and the subsequent low $PM_{2.5}/PM_{10}$ ratio discussed above.

Table 2   Calculated ratios applied for different types of surroundings, based on monitoring data.

Type of area	Ratio ($PM_{2.5}/PM_{10}$)
Central urban background	0.6
Suburban background	0.7
Regional background	0.8

The ratios in Table 2 were allocated to the urban areas based on the population distribution pattern. For the three major cities (Malmö, Göteborg and Stockholm) 60% of the population was estimated to live in central urban areas and 40% in suburban areas. For the smaller cities, 45% of the population was estimated to live in central urban areas and 55% in suburban areas. These population distribution relations are based on information from cities in the eastern part of USA (Figure 4), as no similar studies of distribution patterns was found for European conditions.
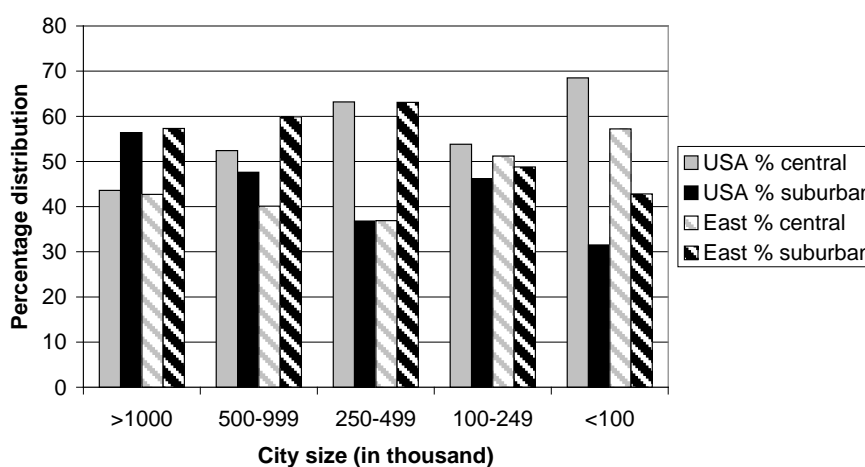
Figure 4   Relations between distribution of population in central parts and suburban parts of cities, both for all cities in the USA and for cities located in the eastern part of the USA (developed in USA by Demographia, 2000, www.demographia.com/).

The GIS-methodology applied to allocate the grid cells within each city into the different classes in Figure 4 consists of several steps: At first, the population size estimated to the central areas [pop_central] was identified (60 or 45% 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 area, 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 40 or 55% 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 2 was applied to the PM$_{10}$ map to calculate the PM$_{2.5}$ map.

3.4 Separation of particle source contributions

Since it is assumed that the relative risk factors for health impact varies depending on the source of particles (WHO, 2013b) the total PM$_{10}$ concentration was separated into different source contributions by using a multivariate method (see further Chapter 3.4.4). In the following sections calculations of different contributions of particles are described.

3.4.1 Small scale domestic heating

Small scale domestic wood fuel burning is an important contributor to particle emission in Sweden (Naturvårdsverket, 2018a). Specific information on the use of wood fuel on municipality level was not available for 2015. Therefore, in order to evaluate the proportion of PM$_{10}$ from small scale domestic wood fuel burning, a relationship was established between total biofuel (of which wood fuel makes up a significant part) and wood fuel consumption on municipality level using data from 2003 (SCB, 2007). This relationship was then applied to the biofuel consumption data from 2015 to derive the wood fuel consumption (www.scb.se). Figure 5 and Figure 6 present the distribution of energy consumption on a county level. The proportion is governed by the air temperature and the supply of wood, as well as traditions in household fuel use in the area.

The energy consumption from wood burning for each of the densely built-up areas in Sweden was drawn from the information presented in Figure 7.
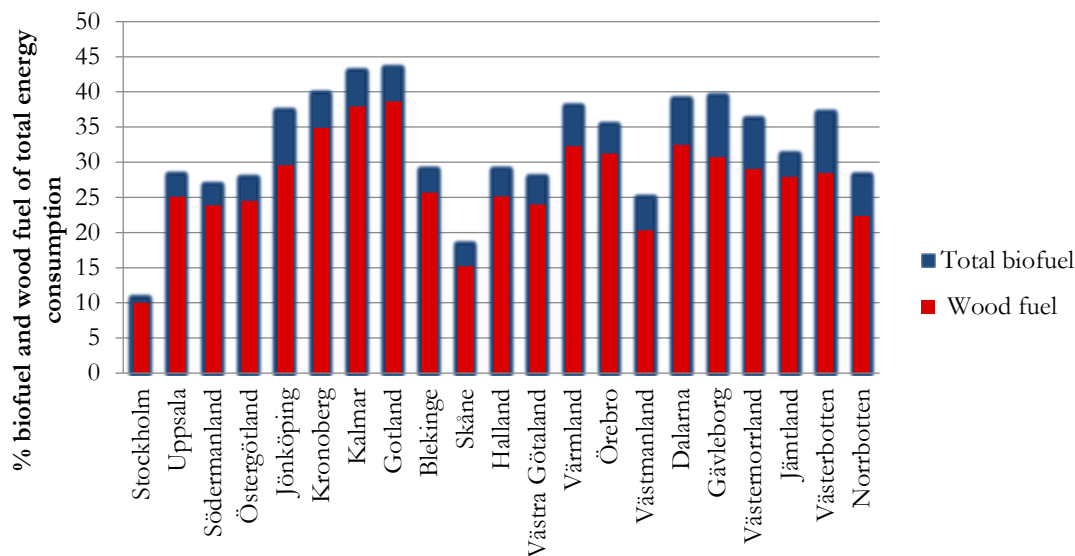
Figure 5 Percentage of total energy consumption from biofuels including wood fuel (blue bars), the percentage from wood fuel (red bars) and per county in 2015.
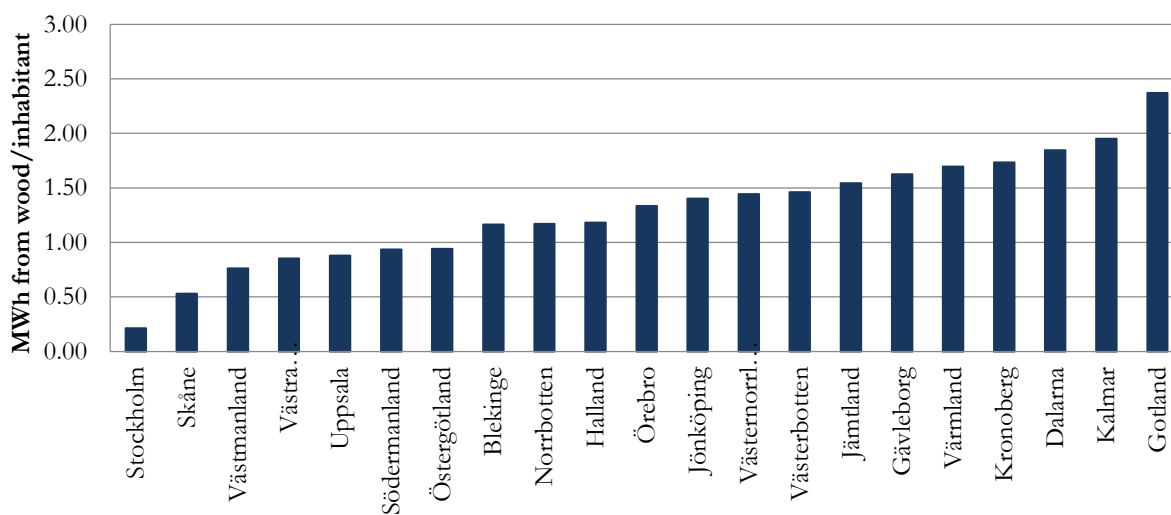
Figure 6 Yearly energy consumption from wood burning (MWh) per inhabitant in each county in 2015.
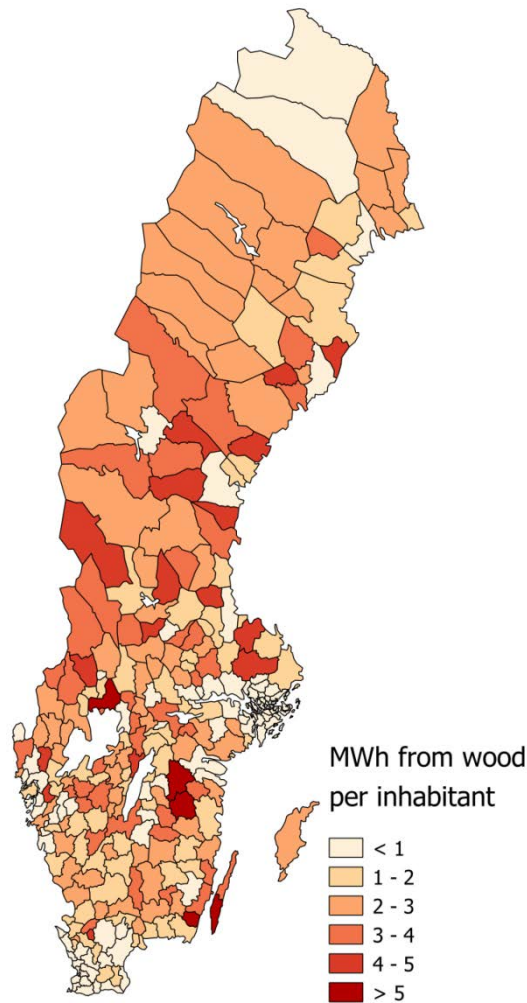
Figure 7   Energy consumption from wood burning (MWh)/inhabitant in each municipality in Sweden in 2015.

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_e). The index is based on the principle that the indoor heating system should heat up the building to +17 °C, while the remaining part is generated by radiation from the sun and passive heating from people and electrical equipment. The calculation of I_e is thus the difference between +17 °C and the outdoor air temperature. For example, if the outdoor temperature is -5 °C the I_e will be 22. During spring, summer and autumn the requirement of indoor heating is less than during wintertime (November – March). Thus, during those months, the outdoor temperature is calculated with a baseline specified in Table 3. The energy index calculations are based on monitored outdoor temperature as means for 30 years at 535 sites distributed over Sweden (www.smhi.se) and result in monthly national distribution of the energy indices, see Figure 8.

Table 3 The base line for the outdoor temperature for calculation of I_e during April - October.

Months	Baseline outdoor temperature (°C)
April	+ 12
May-July	+ 10
August	+ 11
September	+ 12
October	+ 13
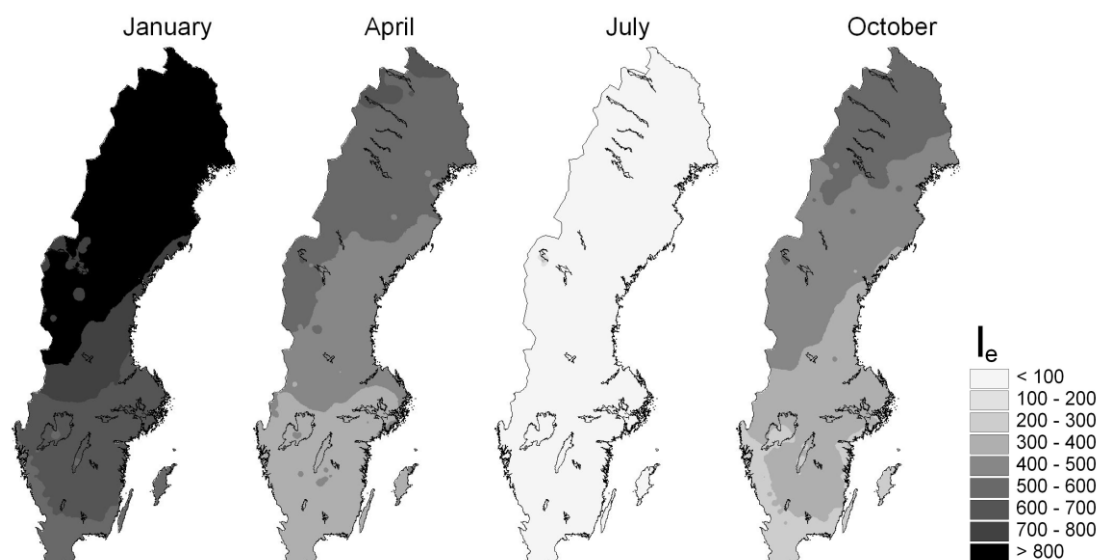
Figure 8 The calculated energy index (I_e) for Sweden i January, April, July, October.

Based on these interpolated maps, bimonthly means of I_e were extracted for each of the 1979 towns in Sweden, and used for calculation of a seasonal variation in the wood fuel consumption.

3.4.2 Traffic induced particles

Traffic contributes to the total concentration of PM_{10} both directly through exhaust emissions from vehicles and secondarily through re-suspension of dust from roads. Traffic related particle concentrations are associated with the NO_2 concentration in urban areas (Sjöberg et al., 2007). Therefore, the previously calculated NO_2 concentrations for all densely built-up areas in Sweden were used to include the direct emissions from traffic in the multivariate analysis to determine the contribution from this source.

Road dust arises mainly from wear of the road surface, brakes, and tyres, and in particular the use of studded tyres. It has been shown that the number of cars using studded tyres is a parameter that regulates the amount of road dust (Gustafsson et al., 2005). Therefore, the use of studded tyres was also included as a parameter in the multivariate analysis.

Re-suspension of road dust occurs mainly during late winter and spring, as a result of the drying 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 the vegetation season starts, mainly in the southern part of Sweden.

The use of studded tyres in January through March 2015 in six different road administration regions (Figure 9 and Figure 10) was obtained from The Swedish Transport Administration (Trafikverket, 2016). Unfortunately, there is no such information available with a monthly resolution throughout the year. A monthly based usage of studded tyres in the road administration regions was established using the distribution pattern derived by Sjöberg et al. (2009).

From this information bimonthly means of the percentage use of studded tyres were calculated for each densely built-up area in Sweden to be further used in the multivariate analysis.
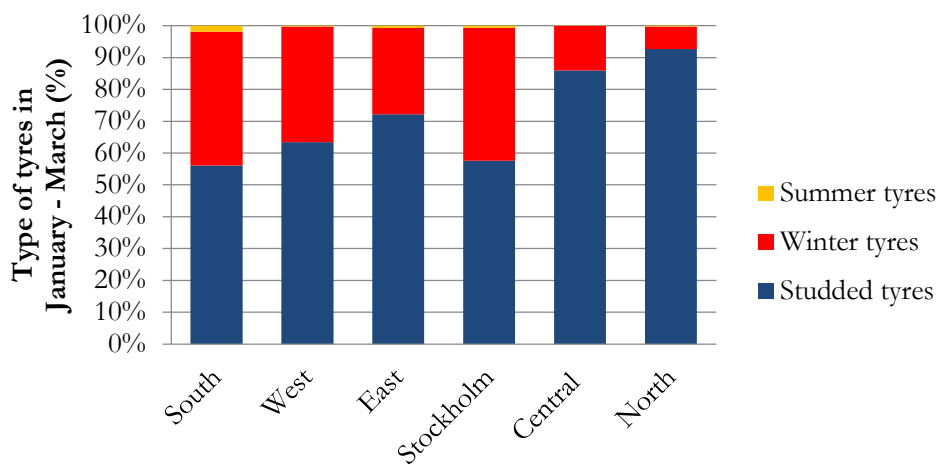
Figure 9   The usage of different types of tyres in January/February within the seven road administration regions in Sweden (visualized in Figure 10).

Swedish road administration regions:

1. South
2. West
3. East
4. Stockholm and Gotland
5. Central north
6. North
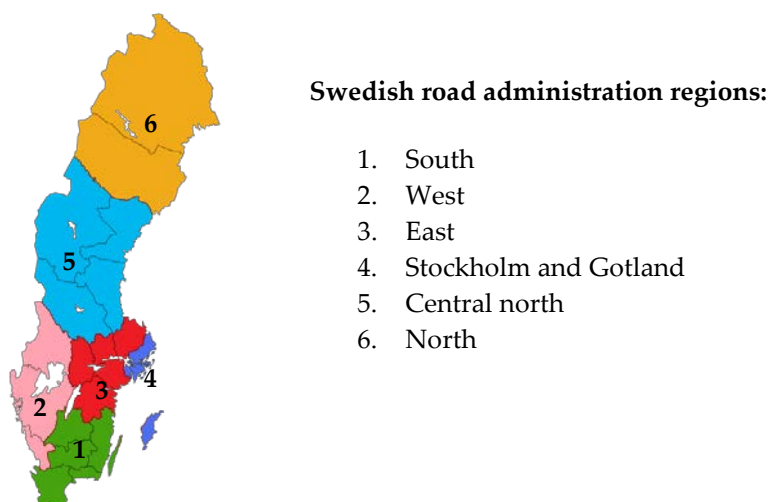
Figure 10   The six road administration regions of Sweden.

3.4.3 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). The mean values of V_i presented in Figure 11 have been calculated in groups of every 1000 steps of the local coordinates.

Figure 11 Bimonthly means (0102 indicates January and February etc. for each monthly pair of a full year) of V_i 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 (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 1500 in the south to about 7000 at the level of about Gävle (between 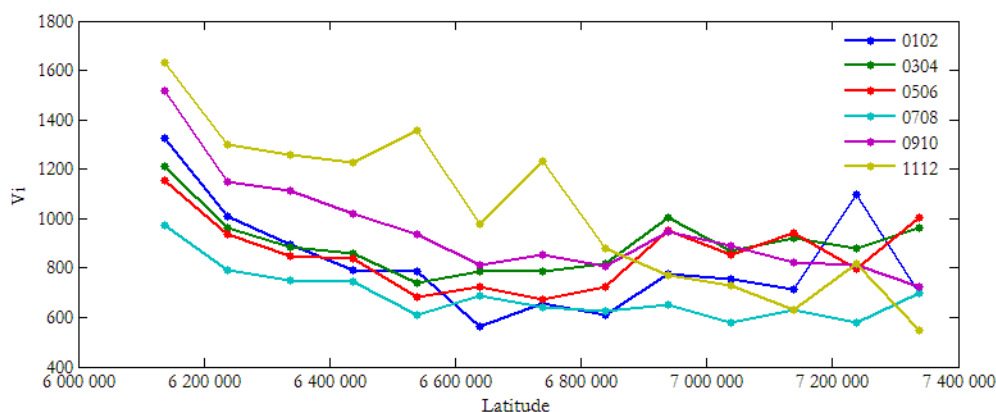6838000 and 6938000 in Figure 11), indicating better dispersion facilities in the south. In Sweden different weather systems are dominant in the northern and southern parts during winter, influencing the V_i, and thus the dispersion of air pollutants, differently. However, this latitudinal pattern is reduced during spring and summer, when other local differences, such as topographical effects, become more important to the dispersion pattern (see Sjöberg et al., 2007).

3.4.4 Multivariate data analysis

In this project Multivariate data analysis (MVDA) has been used to separate different contributions to the total PM_{10} concentration based on six parameters which represent different sources as presented in the previous chapters. The data has been evaluated for 1 979 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). For further description of MVDA and evaluation of model performance see Sjöberg et al. (2009).

In this project, the data was divided into six different bimonthly time periods, 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. Therefore, 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, one for each bimonthly period, predicting the PM_{10} content based on:

- urban background NO_2 concentration;
- usage of studded tyres;
- wood fuel burning;
- energy index;
- mixing index ;
- latitude for each community.

Three of the 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 was therefore excluded in these three models.

All six models gave good predictions of the PM_{10} content. 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 cannot be explained, the air does not behave exactly the same at all times. The model performance was here assessed by cross-validation[1], see Sjöberg et al. (2009).

The result presented in Table 4 shows the performance (Q^2)[2] of the models for each time period.

Table 4   The performance of the models, measured as cross validated explained variance for PM_{10}.

Model	Performance (%)
Month 1-2	99.3
Month 3-4	99.3
Month 5-6	98.2
Month 7-8	99.5
Month 9-10	99.0
Month 11-12	97.6

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 5 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 4.2.2).

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[1] **Cross validation:** Parameters are estimated on one part of a data matrix (observations) and the suitability of the parameters tested in terms of its success in the prediction of the rest of the data matrix (observations)

[2] **Q^2 :** Performance of model prediction of PM_{10} levels, describes the fraction of the total variation of the different parameters that can be predicted by the model according to cross validation (max 1) (in this case Q^2 = performance)

Table 5

Table 5 Average contribution (%) to the PM$_{10}$ content for each variable and time period normalised to sum up to 100. Other variables, not included in this analysis, are also affecting the PM$_{10}$ content.

Time period /Variable	Wood fuel burning	Energy index	Studded tyres	Traffic content	Meteorological index	Latitude
Month 1-2	18	18	18	22	18	7
Month 3-4	11	11	42	26	10	0
Month 5-6	18	19	0	32	24	6
Month 7-8	1	1	0	51	38	9
Month 9-10	6	26	0	32	26	10
Month 11-12	5	21	21	25	20	8

3.5 Population distribution

The current population data applied for exposure calculations in this study were supplied by Statistics Sweden (www.scb.se). The population dataset was based on 2015 census, and in total, 9 851 017 inhabitants were recorded. For 9 839 105 persons it was possible to have the geocoded place of residence. The population data used in the exposure assessment had a resolution of 1 x 1 km.

3.6 Exposure calculation

The distribution of the NO$_2$, PM$_{10}$ and PM$_{2.5}$ concentrations in the urban areas was added to the maps of the background concentration levels to arrive at the final concentration maps. The number of people exposed to different levels of NO$_2$, PM$_{10}$ and PM$_{2.5}$ concentrations were then calculated. By over-laying the population grid to the air pollution grid the population exposure to a specific pollutant was estimated for each grid cell.

3.7 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; β is the exposure-response function (natural logarithm of relative risk per change in concentration), and x is the estimated (excess) exposure.

The calculations were facilitated by a WHO Centre for Environment and Health developed software AirQ+ (Air Quality Health Impact Assessment Tool, WHO, 2016b).

3.7.1 Exposure-response functions (ERFs) 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).

The WHO Review of evidence on health aspects of air pollution, REVIHAAP, (WHO, 2013a), concludes that recent long-term studies are showing associations between PM and mortality at levels well below the current annual WHO air quality guideline level for $PM_{2.5}$ (10 µg/m³). The WHO expert panel thus concluded that for Europe it is reasonable to use linear exposure-response functions, at least for particles and all-cause mortality, and to assume that any reduction in exposure will have benefits. The findings from REVIHAAP are used as a basis for the WHO Project Health risks of air pollution in Europe – HRAPIE (WHO, 2013b).

The REVIHAAP report also concludes that more studies have now been published showing associations between long-term exposure to NO_2 and mortality (WHO, 2013a). This observation makes the situation a bit more complicated when it comes to impact assessments for vehicle exhaust particles, where the close correlation between long-term concentrations of NO_2 and exhaust particles may be confounding (both pollutants cause similar disease and overestimation might appear) in epidemiological studies.

For long-term exposure to NO_2 and mortality the WHO HRAPIE impact assessment report (WHO, 2013b) recommended a risk ratio (RR) of 1.055 (95% CI 1.031-1.08) from the meta-analysis of 11 studies by Hoek et al. (2013). Because of the potential confounding and double counting of mortality effects from $PM_{2.5}$, the HRAPIE report stressed more uncertainty about quantification of NO_2 effects from single-pollutant models. The HRAPIE report also recommended to use the RR from Hoek et al only above the annual mean 20 µg/m³, a recommendation later seen as too conservative by the same group of experts (Heroux et al., 2015).

The potential confounding problem in studies of effects from NO_2 and $PM_{2.5}$ on mortality was dealt with in a recent review paper focusing on 19 epidemiological long-term studies of mortality using both pollutants as exposure variables (Faustini et al., 2014). In their analysis, studies with bi-pollutant analyses ($PM_{2.5}$ and NO_2) in the same model showed decrease in the effect estimates of NO_2, but still suggesting partly independent effects. The greatest effect on natural and total mortality was observed in Europe for both NO_2 and $PM_{2.5}$. In Europe, there was a 7% increase in total mortality for both NO_2 and fine particles, the relative risk (RR) for NO_2 was 1.066 (95% CI 1.029-1.104) per 10 µg/m³ and RR for $PM_{2.5}$ was 1.071 (95% CI 1.021-1.124) per 10 µg/m³.

One relevant study of NO_2 and mortality not included in the meta-analysis followed up 52 061 participants in a Danish cohort for mortality from enrolment in 1993–1997 through 2009, traced their residential addresses from 1971 onwards and used dispersion-modelled concentration of nitrogen dioxide (NO_2) since 1971 to estimate mortality rate ratios with adjustment for potential confounders (Raaschou-Nielsen et al., 2012). The mean NO_2 concentration at the residences of all participants after 1971 was 16.9 µg/m³ (median 15.1 µg/m³). The modelled NO_2 concentration at home was associated with a RR of 1.08 (95% CI 1.01–1.14%), corresponding to 8% higher all-cause mortality per 10 µg/m³.

A smaller Swedish cohort study of men only (n=6557) in Gothenburg studied modelled NOx exposure and mortality during the period 1973-2007. In the group least old at enrolment, aged 48-52 yrs, the RR was 1.06 (95% CI 1.03–1.09) per 10 $\mu g/m^3$ NOx.

The UK expert committee COMEAP has published several reports on long-term effects from NO_2 on mortality. COMEAP has recommended a coefficient of 1.025 (1.01–1.04) with no cutoff (COMEAP, 2015). COMEAP also concludes that reduction of this coefficient may be needed to avoid double counting of effects associated with PM.

US EPA (2016) has revised their conclusion on NO_2 long-term exposure and total mortality from "inadequate to infer the presence or absence of a causal relationship"(2008) into "suggestive of, but not sufficient to infer a causal relationship" (2016), arguing that "potential confounding by $PM_{2.5}$ and traffic-related co-pollutants remains largely unresolved". In contrast, Faustini et al (2014) concluded "the magnitude of the long-term effects of NO_2 on mortality is at least as important as that of $PM_{2.5}$. These results hold when using either 10 $\mu g/m^3$ or the interquartile range, IQR, as the metric of choice. The results of the multipollutant models suggest that the role of NO_2 is independent of that of particles."

From the WHO HRAPIE impact assessment report (WHO, 2013b) it was for long-term exposure to $PM_{2.5}$ and all cause (natural) mortality in ages 30+ recommended to use the exposure-response function from a meta-analysis of 13 cohort studies (Hoek et al., 2013). The RR for $PM_{2.5}$ from this meta-analysis was 1.062 (95% CI 1.040-1.083) per 10 $\mu g/m^3$. This is a coefficient very close to the long-term effect on mortality of $PM_{2.5}$ from the American Cancer Society CPS II cohort (Pope et al., 1995) reported to be 1.06 per 10 $\mu g/m^3$ increment of the annual average $PM_{2.5}$. This assumption, 6% per 10 $\mu g/m^3$, has been used in many health impact assessments, especially for total and long-range transported $PM_{2.5}$, including in our previous national reports (Sjöberg et al, 2007; Gustafsson et al, 2014) and for long-ranged transported $PM_{2.5}$ in the Swedish Clean Air and Climate Research Program (Segersson et al., 2017).

Since many years the research community has meant that it is likely that particles of different types have different effects on mortality and other health outcomes (WHO, 2007; WHO, 2013a). However, a common view is that limited evidence 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).

However, for example ExternE[3] (2005) included assumptions about the toxicity of other different types of PM, which reflect results that indicate 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_{10}; sulfates as equivalent to PM_{10}; primary particles from power stations as equivalent to PM_{10}; primary particles from vehicles as equivalent to 1.5 times the toxicity of $PM_{2.5}$.

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[3] 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.

The effects of combustion-related particles have also been studied using black smoke, black carbon or elemental carbon as the exposure variable. The WHO Project REVIHAAP (WHO, 2013a) recommended that black carbon should be used as exposure variable in more studies, but did not recommend it to be used for the HRAPIE impact calculations (WHO, 2013b).

Information was collected in a review on studies of mortality and long-term exposure to the combustion-related particle indicators (Hoek et al., 2013). The included studies used different methods, and their relation and conversion factors have been described before (Janssen et al., 2011) All-cause mortality was significantly associated with elemental carbon (EC), the meta-analysis resulted in a RR of 1.061 per 1 $\mu g/m^3$ EC (95% CI 1.049-1.073), with highly non-significant heterogeneity of effect estimates. Most of the included studies assessed EC exposure without accounting for small-scale variations related to proximity to major roads.

The conversion from $PM_{exhaust}$ to EC is complicated. The vehicle emission model HBEFA gives the emissions of NO_X and $PM_{exhaust}$ from the vehicle fleet. Measurements performed 2013 by Stockholm City Environment Administration in the tunnel Söderledstunneln suggest that EC represents 30% of exhaust PM_{avgas} (Krecl et al, 2011). Other studies have indicated similar results, and confirm that the RR for background $PM_{2.5}$ becomes too low for $PM_{exhaust}$. With the RR for EC (1.061 per 1 $\mu g/m^3$) and the assumption that 30% of $PM_{exhaust}$ is EC, the RR for $PM_{exhaust}$ would become 1.183 per 10 $\mu g/m^3$.

The calculated RR for $PM_{exhaust}$ of 1.183 per 10 $\mu g/m^3$ comes very close to a RR found for a subset of the American Cancer Society (ACS) subjects, all from Los Angeles County (Jerrett et al., 2005). The authors extracted health data from the ACS survey for metropolitan LA on a zip code-area scale. Using kriging and multiquadric models and data from 23 state and local district monitoring stations in the LA basin they then assigned exposure estimates to 267 zip code areas with a total of 22 905 subjects. For all-cause mortality with adjustments for 44 individual confounders the RR was 1.17 (95% CI = 1.05–1.30) per 10 $\mu g/m^3$. These results suggest that the chronic health effects associated with $PM_{2.5}$ from local sources, mainly traffic and heating, is much larger than reported for metropolitan areas. The direct comparison with the ACS main results show effects that are nearly 3 times larger than in models relying on inter-community exposure contrasts.

More recently 669 000 participants in the ACS CPS II cohort were included in an analysis using a land use regression hybrid model which in a multi-pollutant model separated the effect of regional $PM_{2.5}$ and the effect of near source $PM_{2.5}$ (Turner et al., 2016). For total mortality the RR per 10 $\mu g/m^3$ regional $PM_{2.5}$ was 1.04 (95% CI 1.02–1.06), close to the 6% from the between city analyses often cited (e.g. Pope et al., 1995). However, for near source $PM_{2.5}$ the RR was more than 6 times bigger, 1.26 (95% CI 1.19–1.34) per 10 $\mu g/m^3$. The estimates were also adjusted for NO_2 and ozone. These results indicate that the difference between local PM sources (mainly traffic and heating) and the regional background in RR per mass concentration could be even larger than indicated by Jerrett et al. (2005).

Coarse ($PM_{10-2.5}$) and crustal particles have not been associated with long-term mortality in the cohort studies, and have often shown less evident short-term effects on mortality (Brunekreef & Forsberg, 2005; WHO, 2006b; WHO, 2013a).

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 directly calculated from the long-term (cohort) studies. However, because of the different sources it is likely that there in addition to the effects of background $PM_{2.5}$

is a short-term effect on mortality of road dust and coarse wear particles measured as PM_{10} (Meister et al., 2012). In this study from Stockholm, the estimated short-term (lag01) RR was 1.017 per 10 μg/m³ increase (95% CI 1.002-1.032), with a somewhat smaller effect for $PM_{2.5}$, RR was 1.015 (95% CI 1.007-1,028) per 10 μg/m³ increase in $PM_{2.5}$.

3.7.1.1 Selected exposure-response functions

Despite the fact that usually, as in HRAPIE (WHO, 2013b), all PM regardless of source had to be considered as having the same effect per mass concentration, we have in this study for $PM_{2.5}$ 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 bulk of particles in the cohort studies, largely built up by secondary particles. We also assume that primary combustion PM has a larger effect than the typical, total mix of particles.

For $PM_{2.5}$ in general we have adopted the exposure-response coefficient from HRAPIE (WHO, 2013b) coming from the meta-analysis by Hoek et al. (2013), assuming the RR to be 1.062 (95% CI 1.040-1.083) per 10 μg/m³.

Primary combustion particles from motor vehicles and domestic heating are found in the fine fraction ($PM_{2.5}$). Acknowledging the indications of a stronger effect of such particles, we have in this study, as before, applied the exposure-response coefficient 17% per 10 μg/m³ in a subset of ACS subjects all from Los Angeles County (Jerrett et al., 2005). An alternative would have been 26% (95% CI 19–34) per 10 μg/m³ from the larger study also using cohort data from ACS CPS II (Turner et al., 2016). A high RR for local $PM_{2.5}$ is also supported by the much bigger RRs for EC and BC.

More than half of road dust PM is in the coarse fraction. Since there is in principle no evidence from the cohort studies for an effect of coarse particles ($PM_{2.5-10}$) 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 the study of coarse PM and road dust from Stockholm (Meister et al., 2012) indicated very similar effects for the coarse and fine fraction, we will for road dust (PM_{10}) use the RR for the coarse fraction, 1.017 per 10 μg/m³ increase (95% CI 1.002-1.032).

The issue concerning NO_2 and mortality has become very controversial during the last few years. Local, national and European assessments have used very different approaches. It is not clear if the association between long-term concentrations of NO_2 and mortality are driven by NO_2 itself, or partly related to correlated exposure to exhaust particles. In the multi-pollutant model for mortality in ACS CPS II (Turner et al., 2016), producing a very high RR for near-source $PM_{2.5}$, the RR was very small for NO_2. When we estimate the effects on mortality from NO_2 exposure, we select the results from Denmark, with similar conditions as in Sweden, presented by Raaschou-Nielsen et al. (2012) with a RR of 1.08 per 10 μg/m³ (95% CI 1.01–1.14%) for all-cause mortality.

3.7.2 Exposure-response functions (ERFs) for morbidity

In the recent work on the Thematic Strategy on Air Pollution of European Union about 50 different strategy options are compared, each requiring substantial modelling effort (WHO, 2013b). For practical reasons the number of recommended exposure-response functions must be kept to a minimum. For a national health impact assessment, as for cost-benefit analyses, the set of

exposure-response functions selected should be more complete. For morbidity we have in this study included only some of the potentially available health endpoints to be selected. Previous impact assessments have often used short-term exposures and hospital admissions as an important indicator of health impacts. This approach has been questioned as underestimating the effects (Künzli et al., 2008). Since more results now exists on induction of disease (incidence studies), we have decided for this report to shift our focus from hospital admissions towards incidence. We have decided to include important endpoints that allow comparisons with other health impact assessments and health cost studies.

3.7.2.1 Exposure-response functions for myocardial infarction and stroke

In the European Study of Cohorts for Air Pollution Effects (ESCAPE) long term effects of exposure to air pollution were studied in prospective cohorts. 11 European cohorts from five countries had information about incident cases of cardiovascular events and the most important potential confounders. Individual air pollution exposures were predicted from land-use regression models developed within ESCAPE. One ESCAPE study focussed on the incidence of stroke (Stafoggia et al., 2014) and another similar on incidence of myocardial infarction or other acute coronary events (Cesaroni et al., 2014). The identification of first events during follow-up was accomplished by interviews, inspection of medical records and death certificates, or by record linkage with mortality registries and hospital discharge databases. Prevalent cases at baseline were excluded, however, methods to define and ascertain prevalent cases differed between the 11 cohorts.

For stroke most associations were positive but not fully significant (Stafoggia et al., 2014). The association between $PM_{2.5} < 20$ μg/m^3 and incident stroke was high and borderline significant (Relative risk = 1.29; 95% CI: 1.00, 1.68) for the nine cohorts with individuals below such concentrations. For the seven cohorts with $PM_{2.5}$ concentrations below 15, the hazard ratio was 1.33 (33% increased risk) of incident stroke (95% CI: 1.01, 1.77) for each 5 μg/m^3 increase in $PM_{2.5}$.

In the fully adjusted analysis PM_{10} showed a significant association with the risk of first coronary events, hazard ratio 1.12 (95% CI 1.01 to 1.25) for each 10 μg/m^3 increase (Cesaroni et al., 2014). There was also an almost statistically significant association for $PM_{2.5}$, 1.13 (95% CI 0.98 to 1.30) for each 5 μg/m^3 increase.

3.7.2.2 Exposure-response function 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 et al., 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 met the criteria at the follow up in 1987-88 but not when included in 1977. Assuming the RR from Abbey et 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) for the CAFE programme 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 μg/m^3 PM_{10} per year per 100 000 adults, or 0.0000265 new cases for a change of 1 μg/m^3*person and year. Airport visibility data was used to estimate $PM_{2.5}$ (Abbey et al., 1995) resulting in 14% (95% CI = −0.45–26.2%) change in new cases per 10 μg/m^3 of $PM_{2.5}$. The HRAPIE (WHO, 2013b) preferred this second calculation, finding it much more uncertain to estimate PM_{10} in multiple cities from total suspended particles than in directly estimating $PM_{2.5}$ from airport visibility in each city where good model fits were obtained.

A Swiss study examined relationships between chronic bronchitis and the change in modelled concentrations of PM_{10} in the residence in about 7000 adults aged 16–60 years, at first survey residing in eight communities in Switzerland (Schindler et al., 2009). This study estimated an odds ratio of 0.78 (95% CI = 0.62–0.98), equivalent to a decrease of risk of new reports of chronic bronchitis by 22% (95% CI = 2–38%) per 10 $\mu g/m^3$ decrease in PM_{10}.

In HRAPIE (WHO, 2013b) the results of the Abbey et al. (1995) study was converted to PM_{10} units (assuming $PM_{2.5}/PM_{10}$ = 0.65), and using an inverse-variance weighted average of that study with the results of the Schindler et al. (2009) study resulted in an RR for chronic bronchitis of 1.117 (95% CI = 1.040, 1.189) per 10 $\mu g/m^3$ PM_{10}.

3.7.2.3 Exposure-response function 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 RADs was linked to estimated background rates of, on average, 19 RADs per person per year. From this study came an exposure-response function of 902 RADs (95% CI 792, 1013) per 10 $\mu g/m^3$ $PM_{2.5}$ per 1,000 adults at age 15–64, or 0.092 RADs for a change of 1 $\mu g/m^3$*person and year. In this age group we may see this as work loss days.

In HRAPIE (WHO, 2013b) this RR is expressed as 1.046 per 10 $\mu g/m^3$ $PM_{2.5}$, giving almost the same number of RADs for a change of 1 $\mu g/m^3$*person and year. According to the experts in HRAPIE similar or greater effects of PM should be expected in older and younger persons (WHO, 2013b).

3.7.3   Selected base-line rates for mortality and morbidity

In order to estimate how many deaths and incident cases of myocardial infarction and stroke that depend on elevated air pollution exposure we need to use a base-line rate. We collected the base-line rates 2015 for Sweden from the Statistic database at Swedish National Board of Health and Welfare (www.socialstyrelsen.se).

For mortality we calculated the following rates: total mortality (all causes) all ages: 929 per 100 000 persons and total mortality in age group 30+: 1438; of which with external causes: 72, giving the total natural mortality rate 1366 per 100 000 persons aged 30 years old or older.

Since the first time incident cases of myocardial infarction and stroke include also those who died, and we calculate also effects on total (natural) mortality in age group 30+, we decided to adjust the annual incidence rate by subtracting deaths and those who died within one year. For the age group 30+ this left us with an incidence of 327 per 100 000 persons in myocardial infarction and 253 per 100 000 in stroke.

Without country-specific baseline rates for chronic bronchitis, HRAPIE experts (WHO, 2013b) recommended estimates based on the studies AHSMOG and SAPALDIA (WHO, 2013b). However, a new Swedish study found an incidence of 90 per 100 000 person years (Holm et al., 2014), much

lower than the 390 per 100 000 recommended and earlier used in our reports. We have now applied the Swedish baseline.

For RADs we have applied the commonly used 0.092 RADs for a change of 1 µg/m³*person and year for PM$_{2.5}$ exposure recommended by HRAPIE (WHO, 2013b), which fits quite good with the 7.5 days per year of sick leave reported by Statistics Sweden for year 2013[4].

3.7.4   Health impact calculations

<u>1. Impact of local NO$_2$ and vehicle exhaust on mortality</u>

We calculate the local (urban) NO$_2$ (an indicator also of other locally generated pollutants, e.g. soot and ultrafine particles in motor vehicle exhaust) long-term impact on natural mortality in ages 30+. We apply the Risk Ratio (RR) from Denmark (Raaschou-Nielsen et al., 2012) of 1.08 per 10 µg/m³ (95% CI 1.01–1.14%) for all-cause mortality. Since the smooth risk slope is steep at low concentrations, and we consider *only* the local contribution, we use no cutoff.

<u>2. Impact of local road dust and wood smoke on mortality</u>

For a more detailed assessment of PM$_{10}$, including the fine fraction (PM$_{2.5}$), we assume that the effects on all-cause mortality are different for different types of particles.

For combustion generated primary particles, we assume the total mass to be in the fine fraction, and apply the RR 1.17 (95% CI = 1.05–1.30) per 10 µg/m³ from the intraurban Los Angeles analysis of ACS data (Jerrett et al., 2005) in the age group 30+ years. This RR is also supported by results for EC. We assume no threshold. For PM$_{2.5}$ from vehicle exhaust it may be double counting to add estimates for mortality associated with PM$_{2.5}$ from exhaust and from urban NO$_2$. Thus, we consider the results for urban NO$_2$ to represent the effect on natural mortality from local vehicle exhaust, including exhaust particles.

For road dust PM$_{10}$, dominated by the coarse fraction, we assume a short-term effect on daily total number of deaths in all ages, and apply the RR for the coarse fraction 1.017 per 10 µg/m³ increase (95% CI 1.002-1.032) observed in Stockholm (Meister et al., 2012). We assume no threshold, and as the study included all ages we assume the association to occur for all ages.

<u>3. Impact of regional PM$_{2.5}$ on mortality</u>

We estimate the regional (long-distance transported) PM$_{2.5}$ long-term impact on natural mortality in ages 30+. We assume the same effect (same RR) for all sources without any cutoff. We apply the RR from HRAPIE (WHO, 2013b) 1.062 (95% CI 1.040-1.083) per 10 µg/m3.

<u>4. Impact of total and local PM on morbidity</u>

We assume that total PM$_{2.5}$ has an impact on RADs both in the age group 15-64 years, and in the rest of the population, but have to assume the same effect from all sources. The effect is assumed to

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be 902 RADs (95% CI 792-1013) per 10 $\mu g/m^3$ PM$_{2.5}$ per 1,000 adults at age 15-64, or 0.0902 RADs for a change of 1 $\mu g/m^3$*person and year.

We assume total PM$_{10}$ to have an impact on the incidence of Chronic Bronchitis in the age group 30+ with a RR of 1.117 (95% CI = 1.040, 1.189) per 10 $\mu g/m^3$ PM$_{10}$ and no cutoff.

Because the risk functions are from analyses of within city contrasts in exposure, we assume only local PM to impact cardiovascular morbidity included as the incidence of myocardial infarction and incidence of stroke in the age group 30+. The applied relative risks are 1.12 (12%) per 10 $\mu g/m^3$ PM$_{10}$ for myocardial infarction, and 1.66 per 10 $\mu g/m^3$ PM$_{2.5}$ for stroke.

3.8   Socio-economic valuation

In brief, socio-economic valuation of health impacts from air pollution should include all welfare parameters of relevance for health effects related to air pollution. The valuation allows for consideration of all economic decision makers in society; individuals (households), firms and government, and should include direct and indirect use costs as well as non-use (intangible) costs of poor air quality. Ideally, all these cost parameters should be taken under consideration during the valuation of health impacts, but it is sometimes difficult to measure and calculate reliable estimates of them. It can also be that some methods of valuation aggregate the parameters, thereby making it difficult to distinguish between them.

The method and data used for socio-economic valuation of mortality impact, impact on chronic bronchitis, and impact on restricted activity days, are in this study identical to the method and data used in Sjöberg et al. (2007) and Gustafsson et al. (2014), but updated with respect to inflation between 2010 and 2015. In this study, the socio-economic central estimate cost of additional fatality associated with poor air quality is ~5.7 million Swedish Krona in year 2015 nominal value (SEK$_{2015}$) per case, and the central estimate of additional chronic bronchitis 2.2 million SEK$_{2015}$/case. Further, the central estimate of restricted activity days are for working age population ~1480 SEK$_{2015}$/day, and for the non-working age population ~530 SEK$_{2015}$/day. These will not be further discussed here, but method description and literature review are available in Sjöberg et al. (2007) and Gustafsson et al. (2014).

In this study, relevant updates on health end points are presented for incidences of stroke and myocardial infarction. Correspondingly, there are changes to the valuation of morbidity presented in Gustafsson et al. (2014). Instead of valuating respiratory and cardiovascular hospital admissions, we value incidences of myocardial infarction and stroke, as well as an estimate of the value of post-incidence illness. To separate health impacts associated with anthropogenic emissions from health impacts associated with natural emissions we considered concentrations above 2 $\mu g/m^3$ as anthropogenic for long-range transport of PM$_{2.5}$, and above 5 $\mu g/m^3$ as anthropogenic for NO$_2$. We did not use any cut-off for PM concentrations due to domestic heating and transport.

3.8.1   Socio-economic costs of myocardial infarction

There are some recent studies that provide useful input to a reasonable estimate of socio-economic costs of myocardial infarction, but unfortunately no Swedish studies are available. Kern et al.

(2016) studied the long term healthcare costs of increased health care efforts to survivors of acute myocardial infarction in the United States. The healthcare costs were estimated separately for groups with or without other risk factors. Using data from insurance claim records, the authors could see that healthcare costs related to cardiovascular health increased from ~US$1 650 per year prior to the acute myocardial infarction incidence to ~US$48 000 for the year of the incidence and ~US$7 000 – 8 000 for the three years following the incidence for the group without additional risk factors. Costs were higher for the group with additional risk factors, but returned to pre-MI levels directly after the incidence year. Seo et al. (2015) studied Korean insurance claims data and found that direct health care costs and indirect costs associated with acute myocardial infarction during in 2012 were ~US$1 200 billion, a decrease with 18% from 2007, the start year of the analysis. 52% of this cost was due to direct health care costs. However, these costs included incidences leading to fatality. Chang et al. (2012) used a similar approach on Korean data and found that non-lethal acute myocardial infarction in 2005 had a direct and indirect socio-economic cost corresponding to ~$3 200/patient when considering inpatient and outpatient costs, and ~US$4 470/patient when including all costs but costs for premature death. Neither Seo et al. (2015) or Chang et al. (2012) present costs distribution over time per case, which implies that the cost estimates from Kern et al. (2016) are most suitable for analysis of socio-economic costs of myocardial infarction related to air pollution. Seo et al. (2015) and Chang et al. (2012) are used for comparison.

In order to transfer these costs to Swedish circumstances related to air pollution we extract relevant posts from the cost estimates from the literature to ensure that only costs for non-lethal myocardial infarction is included and calculate net present values using a 2% discount rate. We use the value transfer method (Boyle et al., 2013) to estimate corresponding costs in Sweden. A particular challenge when transferring the results from Kern et al. (2016) is that US healthcare costs are recognised as roughly 2 times higher than in Sweden (OECD, 2011; OECD, 2017), and using purchase power parity is not sufficient to adjust this problem. However, this US 'cost amplifier' is unevenly distributed over health procedures and some procedural costs of relevance for myocardial infarction, such as the procedures "Percutaneaous transluminal coronary angioplasty" and "Coronary artery bypass graft" have been shown to be equally expensive in the US and Sweden (OECD, 2011). Therefore, only the costs for the years following the acute incidence are adjusted, implying the assumption that costs for the first year of treatment of myocardial infarction are identical in the US and Sweden.

In total, given the above mentioned adjustments, the costs of myocardial infarction are therefore ~US$ 46 900 during the year of incidence, ~US$ 2 900 first year after, ~US$ 2 500 second year after, and ~US$ 2 700 third year after incidence. This correspond to a net present value of ~US$ 53 600 and ~428 300 SEK$_{2015}$ per survivor, given an exchange rate in 2009 of 7.6 SEK/US$ and a Consumer Price Index increase of ~4% between 2009 and 2015 (~275 800 SEK$_{2015}$ per survivor if correcting all annual costs with the 'cost amplifier'). This number is higher than in Seo et al. (2015) and Chang et al. (2012), which we estimate to ~21 500 SEK$_{2015}$/survivor and ~37 500 SEK$_{2015}$/survivor. It does however appear as if the US study includes more cost parameters than the Korean studies and the annualisation of costs in the Korean studies are problematic since it is opaque how many patients that receive multi-year treatments, and in what phase of treatment they are. As a comparison, the healthcare costs during the follow-up period were US$4 127/patient on average in Kern et al. (2016), which is quite close to the Chang et al. (2012) estimate. So the major part of the difference might be that Kern et al. (2016) clearly specifies costs of surgery/treatment per patient, whilst the Korean studies do not.

3.8.2   Socio-economic costs of stroke

Payne et al. (2002) reviewed earlier cost of stroke estimates and presented Swedish estimates of long-term costs of stroke in the range of 266 000 SEK$_{1993}$ for men and 488 000 SEK$_{1993}$ for women. Di Carlo (2009) presented a study from the UK. In the UK, costs of stroke amounted then to £8.9 billion per year for 130 000 new stroke patients per year and more than one million stroke survivors. In the EU societal costs for cerebrovascular diseases amounted to ~€19 billion per year for health care costs and ~€19.2 billion for production losses and informal care in 2009. For Sweden, the corresponding numbers are ~€0.6 and ~€0.5 billion per year (EHN, 2012). None of the cost estimates separated costs for survivors, which is needed for our purposes. In contrast to our cost estimates for myocardial infarction, there is a recent Swedish estimate of costs for stroke, which also individually presents costs for stroke survivors. Lekander et al. (2017) used a similar approach as Kern et al. (2016) and identified the same type of cost parameters and separated costs between intracerebral haemorrhage and ischemic stroke. They found that socio-economic costs of intracerebral haemorrhage are ~638 400 SEK$_{2016}$ per survivor during the first year and 242 400 SEK$_{2016}$ per survivor during the second year after a stroke event. Costs for ischemic stroke survivors are ~389 700 SEK$_{2016}$ (year 1) and ~323 600 SEK$_{2016}$ (year 2).

The cost estimates from Lekander et al. (2017) only covered year 1 and 2 costs, while it is assumed that stroke survivors will experience costs for the remainder of their lifetime. To correct for this we first calculated an estimate of remaining life years after stroke, using data from Lekander et al. (2017) and Statistics Sweden[5] on average life expectancy for the population included in Lekander et al. (2017). The mean age of stroke event was 73.3 for intracerebral haemorrhage and 76.6 years for ischemic stroke. This corresponds to a remaining life expectancy of 11 and 8 years respectively. To calculate the costs for year 3 – 11 (8) we assumed a hyperbolic decay rate of socio-economic costs over time based on year 1 and 2 data. We calculated net present values of the costs with a 2% discount rate. Correspondingly, the net present value of intracerebral haemorrhage is ~1.8 million SEK$_{2015}$ per survivor, and of ischemic stroke ~2 million SEK$_{2015}$ per survivor. To aggregate a common cost for the more generic 'stroke' we weighted the costs of intracerebral haemorrhage and ischemic stroke. 12% of the stroke patients in Lekander et al. (2017) suffered from intracerebral haemorrhage and 88% from ischemic stroke. The survivor rate after year 2 was 56% and 69% respectively. Correspondingly the weighted share of survivors was ~10% and ~90%, which gave a weighted net present value of stroke as ~2 million SEK$_{2015}$ per survivor.

3.8.3   An estimate of socio-economic costs of long-term illness after incidence

In addition to the above presented costs, which are examples of direct and indirect use costs, it is important to estimate non-use (intangible) costs of poor air quality so as to get a complete picture of the socio-economic costs of these impacts. However, literature estimates of these are scarce, which indicates a research gap. The only suitable estimate we have found is from Chilton et al. (2004), in which they studied the willingness to pay (WTP) for a longer life. One of the interesting parts with the Chilton et al. study is that they estimated WTP for a longer life in good health or poor health separately. They found that the value of an extra year in good health is worth 27 630

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[5] https://www.scb.se/hitta-statistik/statistik-efter-amne/befolkning/befolkningens-sammansattning/befolkningsstatistik/pong/tabell-och-diagram/helarsstatistik--riket/aterstaende-medellivslangd/

£$_{2003}$ and the value of a year in poor health 7 280-14 280 £$_{2003}$. By using this difference in preferences as a proxy for welfare loss of living in poor health we can estimate non-use costs of myocardial infarction and stroke. The corresponding welfare loss of a year in poor health is ~198 500-302 500 SEK$_{2015}$. Furthermore, we assume 4 years of poor health after myocardial infarction (based on years of increased health expenditures in Kern et al. (2016)), 11 years in poor health after intracerebral haemorrhage, and 8 years of poor health after ischemic stroke. Finally, we assume no discounting of these values, following the discussion on valuation of life years in Desaigues et al. (2011). Given the scarcity of earlier studies we consider the estimates for costs of long-term illness after incidence as a first approximation to hopefully be further developed in the future.
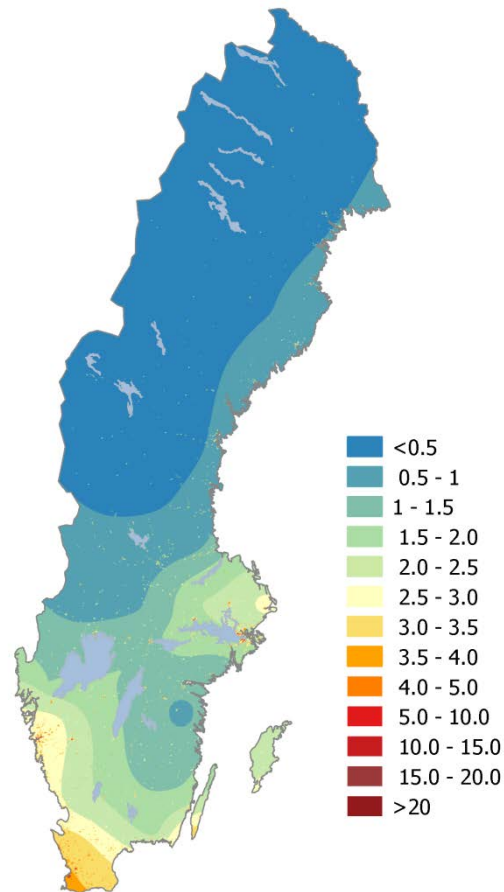
4 Results

4.1 Calculation of air pollutant concentrations

4.1.1 National distribution of NO$_2$ concentrations

The annual mean concentration of NO$_2$ for 2015, calculated with the URBAN model, is presented in Figure 12. The result is based on calculated bimonthly means in order to capture the seasonal variation, where higher concentrations usually occur during winter.

As presented in Figure 12, calculations indicate annual mean background NO$_2$ concentrations for 2015 below 5 μg/m^3 in all rural areas. Urban background concentrations in small to medium sized cities reached NO$_2$ concentrations of up to 15 μg/m^3, while concentrations exceeded 20 μg/m^3 in the central parts of the three largest cities in Sweden; Stockholm, Gothenburg and Malmö. Calculated urban background NO$_2$ concentrations indicated annual means up to 27 μg/m^3 in Malmö and Gothenburg, and up to 30 μg/m^3 in Stockholm. The calculated NO$_2$ concentrations were thus well below the environmental standard for the maximum annual mean value (40 μg/m^3). The long-term environmental objective of concentrations below 20 μg/m^3 as an annual mean for the whole country was, however, exceeded in the larger urban areas.

Based on the calculated results, no 1 x 1 km grid cell exceeded the annual air quality standard for NO$_2$ concentrations for 2015. However, the standards are also valid for road side concentrations in street canyons. A study by Persson and Haeger-Eugensson (2006) showed that road side concentrations in Swedish cities were generally around 1.5 times higher than the urban background, although in poorly ventilated urban streets with dense traffic, much higher concentrations could be found. Thus there are likely additional exceedances of the air quality standard at road-side locations, which is not considered in this study. This is also reflected in Air Quality Plans submitted by municipalities where violation of the limit values remains at some locations.
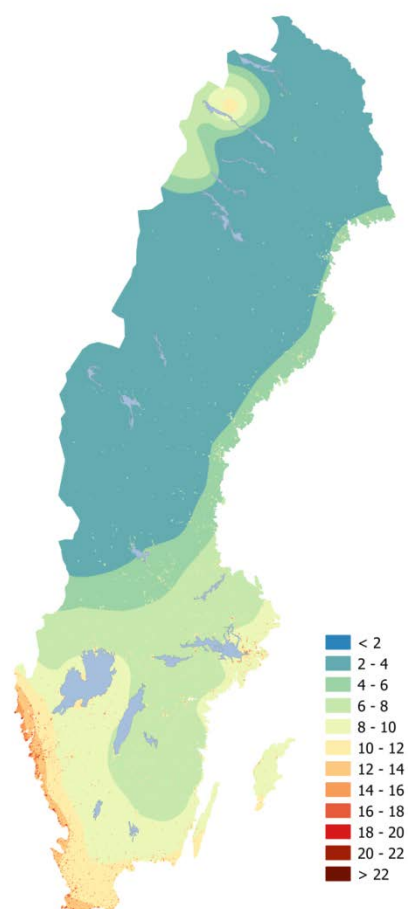
Figure 12   NO$_2$ concentrations, as annual mean, for 2015 in Sweden, unit µg/m^3.

4.1.2   National distribution of PM$_{10}$ concentrations

The annual mean concentrations of PM$_{10}$ for 2015, calculated with the URBAN model, are presented in Figure 13. The result is based on calculated bi-monthly means in order to capture the seasonal variations, where higher concentrations of PM$_{10}$ usually appear during late winter-spring depending on the location in the country.

In Figure 13 it can be seen that the PM$_{10}$ concentrations as yearly mean are primarily governed by the regional background concentrations. Due to the strong influence from the long-range transport originating from continental Europe, there is a considerable latitudinal decrease to the north in the regional background concentrations. The urban background concentrations in the larger urban areas in the southern and western parts of Sweden were calculated to be about 20 - 23 µg/m^3, while the concentration in Stockholm was estimated to approximately 19 µg/m^3. Compared to the environmental standard for the annual mean value (40 µg/m^3) there were no exceedances in urban background air in Swedish towns on the 1 x 1 km resolution in 2015. The long-term environmental objective of PM$_{10}$ annual mean concentrations below 15 µg/m^3 in the whole country was, however, exceeded in the larger urban areas as well as along the west coast.

■	< 2
■	2 - 4
■	4 - 6
■	6 - 8
■	8 - 10
■	10 - 12
■	12 - 14
■	14 - 16
■	16 - 18
■	18 - 20
■	20 - 22
■	> 22

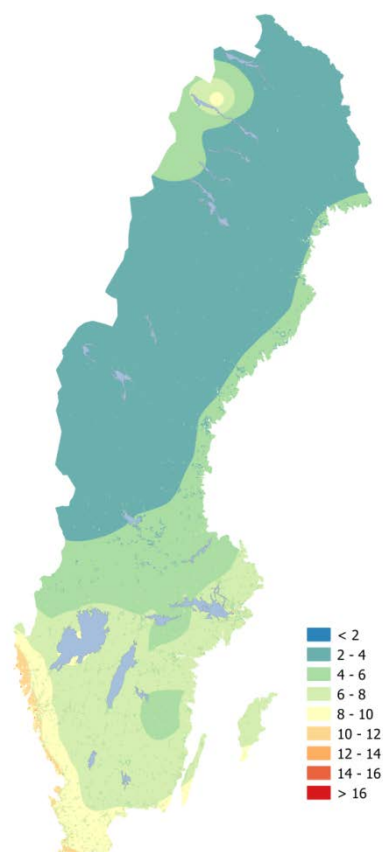**Figure 13** PM_{10} concentrations, as annual mean, for 2015 in Sweden, unit µg/m³.

4.1.3   National distribution of $PM_{2.5}$ concentrations

The annual mean concentrations of $PM_{2.5}$ for 2015 are presented in Figure 14. The result is based on the earlier calculated PM_{10} concentrations in combination with calculated ratios based on empirical relationships of $PM_{10}/PM_{2.5}$.

4.2 Population exposure

The population exposure to different NO₂ and particle concentrations has been calculated based on the calculated air concentrations.

Figure 14   PM₂.₅ concentrations, as annual mean for 2015, in Sweden, unit μg/m³.

4.2.1 Exposure to NO₂

Studies providing dose-response relationship for calculations of health impact from air pollution exposure are almost exclusively based on urban background air pollutant concentrations. In order to allow application of known relationships, this study is therefore based on urban background concentrations. As previously mentioned, higher NO₂ concentration will normally be found in roadside locations compared to urban background, due to emissions from, for example, traffic within street canyons. Consequently, a slightly higher exposure would likely have been found if roadside concentrations were used instead of background in the exposure calculations. However, very few dose-response functions are based on roadside concentrations and exposure studies such as this one can therefore not rely on roadside concentrations.

The population exposure to NO₂ annual mean concentrations in Sweden in 2015 is shown in Table 6 and Figure 15. In 2015, the annual mean population weighted exposure to NO₂ was 6.4 μg/m³ , of

which the urban contribution was 4.4 µg/m³ . The largest group in all age classes, around 45%, was exposed to annual mean concentrations of NO_2 below 5 µg/m³. Approximately 40% were exposed to NO_2 concentration levels between 5-10 µg/m³, and less than 5% to levels of NO_2 above 15 µg/m³. The population exposed to NO_2 from local urban sources are presented in Figure 15c and 15d. According to these calculations 14% of the Swedish population lives in areas without any urban NO_2 contribution.
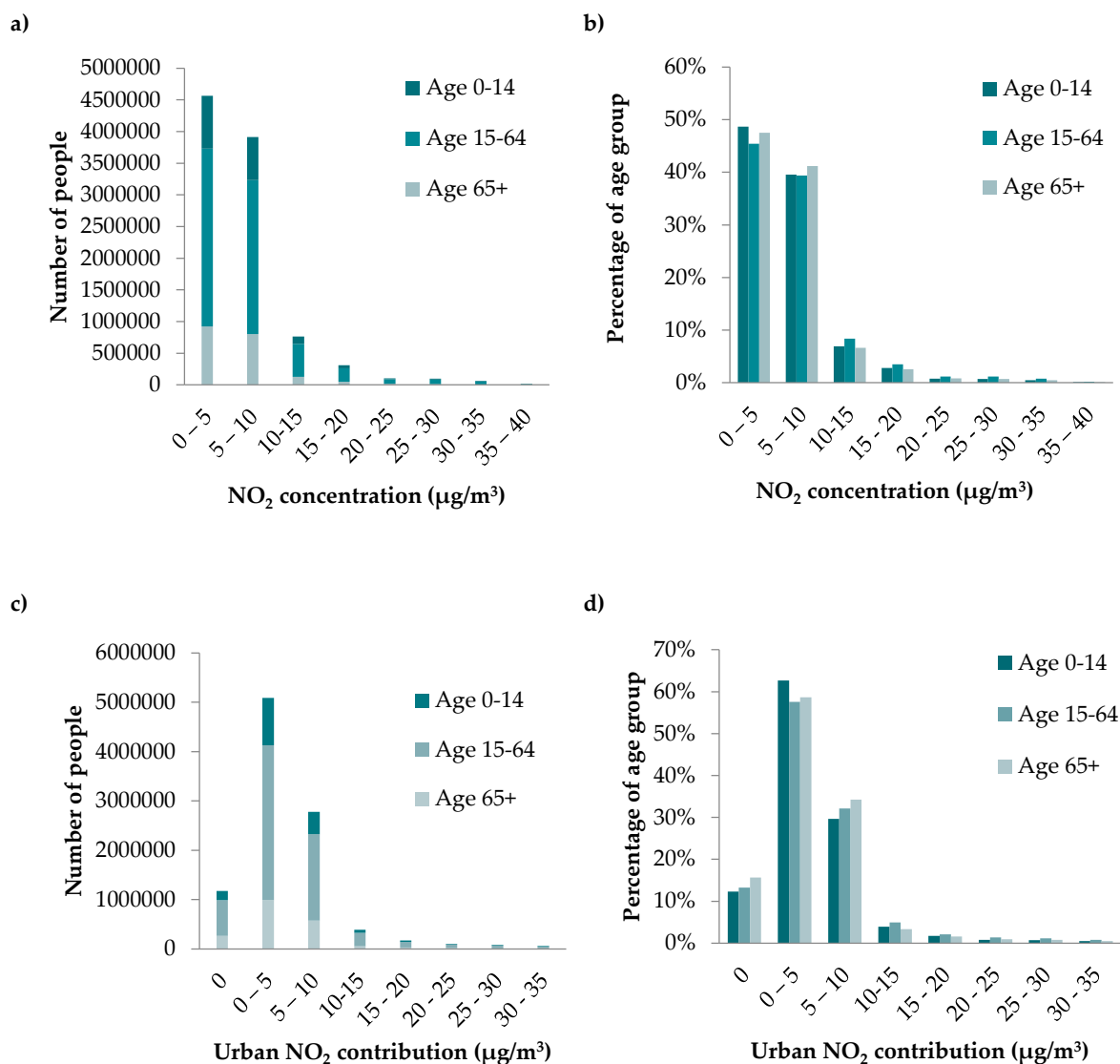
**Figure 15**   Population exposure to total NO_2 annual mean concentrations in Sweden expressed in a) number of inhabitants and b) percentage of population, divided into the age categories 0 – 14 (dark blue), 15-64 (blue), and 65+ years of age (light blue). Population exposure to mean urban NO_2 contribution in Sweden expressed in c) number of inhabitants and d) percentage of population, divided into the age categories.

Our calculations also show that compared to the population as a whole, children and elderly (age categories 0-14 and 65 +) were slightly overrepresented in the lower exposure concentration categories, and slightly underrepresented in the higher, with the opposite pattern in the age category 15-64 years of age.

## 4.2.2   Exposure to PM_{10} and $PM_{2.5}$

As for NO_2, exposure to PM_{10} and $PM_{2.5}$ are based on calculations of urban and regional background concentrations to allow application of known dose-response functions for health effects. Higher particle concentrations, especially PM_{10}, and consequently higher exposure, would likely have been found if roadside concentrations were used instead of background in the exposure calculations. However, as very few dose-response functions are based on roadside concentrations exposure studies such as this one can therefore not rely on roadside concentrations.

The exposure distribution of the Swedish population to annual mean PM_{10} concentrations in 2015 is shown in Figure 16. Less than 3% of the population was exposed to concentrations below 5 $\mu g/m^3$, with a minimum of just above 3 $\mu g/m^3$. Approximately 75% of the population was exposed to PM_{10} concentrations between 5 and 15 $\mu g/m^3$. That leaves 22% of Swedish inhabitants exposed to PM_{10} levels higher than the environmental objective for PM_{10} (15 $\mu g/m^3$). However, only 0.3% of the population was in 2015 exposed to PM_{10} concentrations above the environmental air quality standard (40 $\mu g/m^3$). As for NO_2 children and elderly (age categories 0-14 and 65 +) were slightly overrepresented in the lower exposure concentration categories, and slightly underrepresented in the higher, with the opposite pattern in the age category 15-64 years of age.
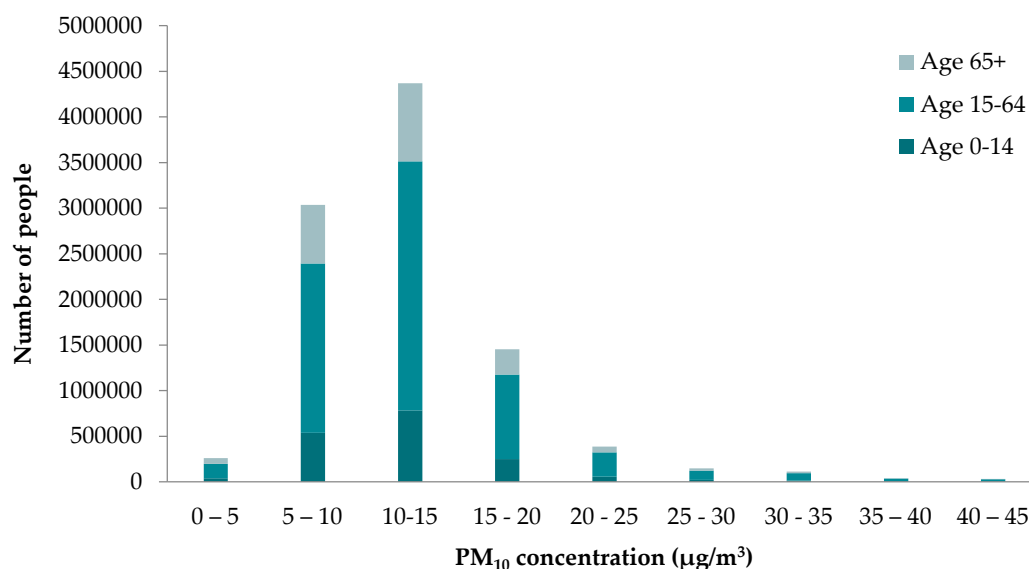
Figure 16   Number of inhabitants exposed to total PM_{10} annual mean concentrations in Sweden in 2015, divided into the age categories 0 – 14 (dark blue), 15-64 (blue), and 65+ years of age (light blue).

The estimated exposure to the total annual mean concentrations of $PM_{2.5}$ is shown in Figure 17. The majority of the population, almost 80%, was exposed to $PM_{2.5}$ annual mean concentrations below the environmental objective (10 $\mu g/m^3$), with a minimum of 2 $\mu g/m^3$. Approximately 20% of the people in Sweden were exposed to levels between 10 and 20 $\mu g/m^3$ and less than 1% was exposed to $PM_{2.5}$ concentrations above the environmental quality standard (20 $\mu g/m^3$).
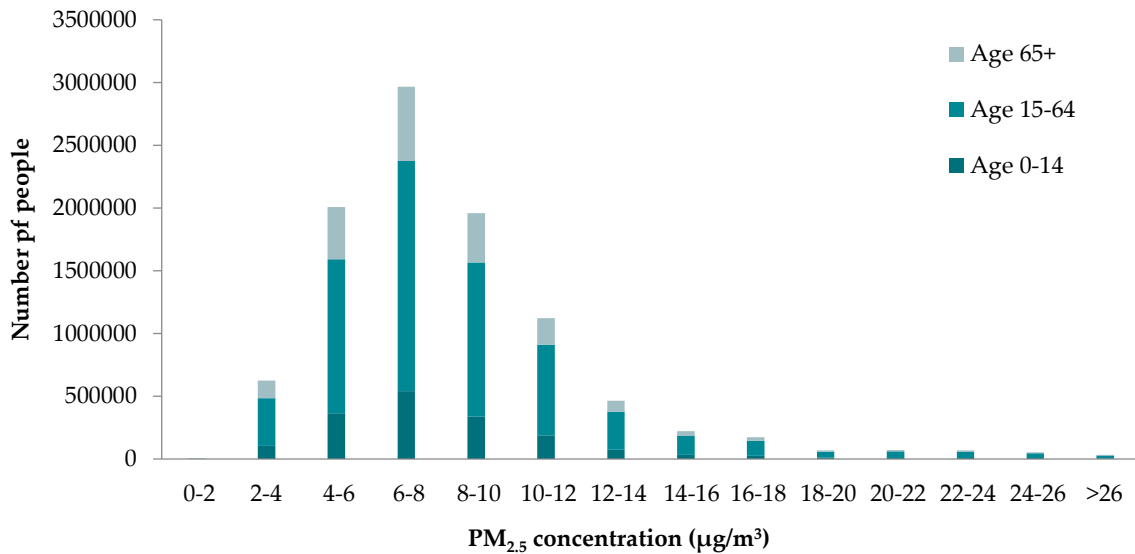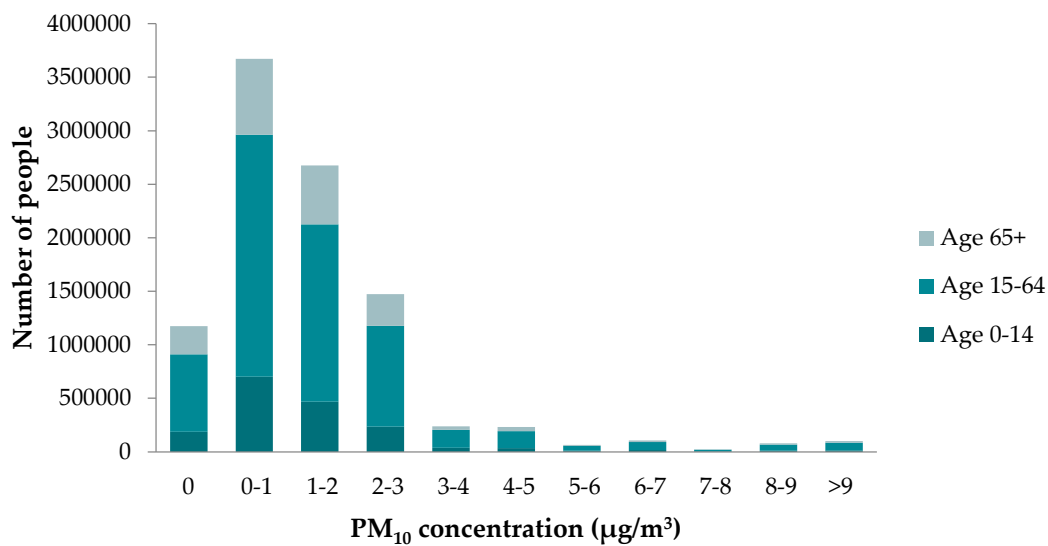
Figure 17   Number of inhabitants exposed to total PM2.5 annual mean concentrations in Sweden in 2015, divided into the age categories 0 – 14 (dark blue), 15-64 (blue), and 65+ years of age (light blue).

As mentioned earlier the particle contribution from different sources (road dust, traffic exhaust, wood burning and long-range transport) to the particle levels was calculated. The number of people exposed to different PM10 concentrations from road dust is presented in Figure 18. Particles from traffic exhaust, wood burning and long-range transport were assumed to all belong to the PM2.5 fraction and are presented in Figure 19 - 21.

Figure 18   Number of inhabitants exposed to PM10 annual mean concentrations from *road dust* in Sweden in 2015, divided into the age categories 0 – 14 (dark blue), 15-64 (blue), and 65+ years of age (light blue).

Road dust contributed on average 1.4 μg/m³ to the annual mean population weighted exposure of PM$_{10}$ in 2015. More than 90% of the population were exposed to less than 3 μg/m³ PM$_{10}$ from road dust, and almost 34% of the total population was, according to our model, exposed to negligible concentrations (less than 0.5 μg/m³) of road dust (Figure 18).

According to the calculations the contribution from traffic exhaust to the total PM$_{2.5}$ concentration was 0.1 μg/m³ (Figure 19). Over 90% of the population was exposed to less than 0.5 μg/m³ of PM$_{2.5}$ from traffic exhaust. As this does seem unrealistically low, the NO$_2$ concentration may be a better indicator of traffic exhaust pollution.

Figure 19   Number of inhabitants exposed to PM$_{2.5}$ annual mean concentrations from *traffic exhaust* in Sweden in 2015, divided into the age categories 0 – 14 (dark blue), 15-64 (blue), and 65+ years of age (light blue).
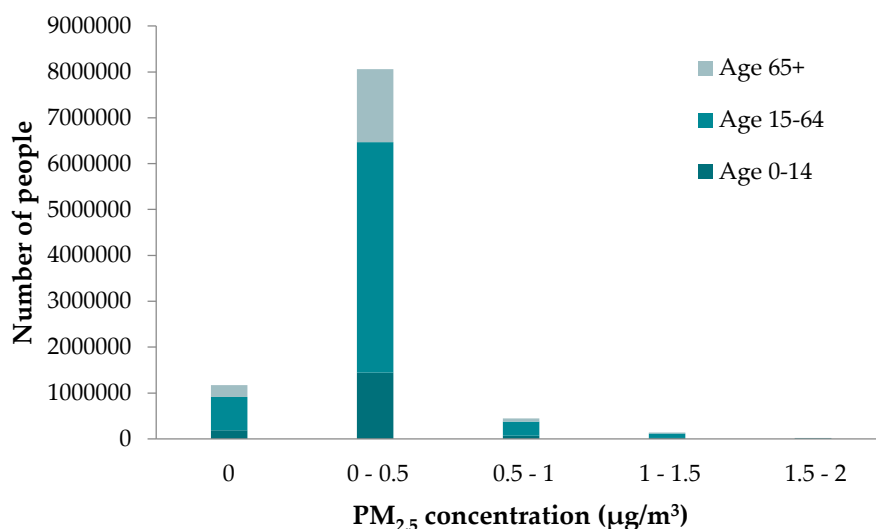
Domestic heating contributed on average 2 μg/m³ to the annual mean of PM$_{2.5}$ in 2015. Of this, 0.8 μg/m³ was attributed to wood fuel. Approximately half of the population were exposed to less than 0.5 μg/m³ PM$_{2.5}$ from *wood burning* (Figure 20).

In order to assess the exposure and health effects from long distance transported particles, these were assumed to be represented by the regional background PM$_{2.5}$ concentrations. This was the category that contributed the most to the total PM$_{2.5}$ concentration with an average of 7.2 μg/m³ in 2015, with a population exposure between 2 and 12 μg/m³ (Figure 21).
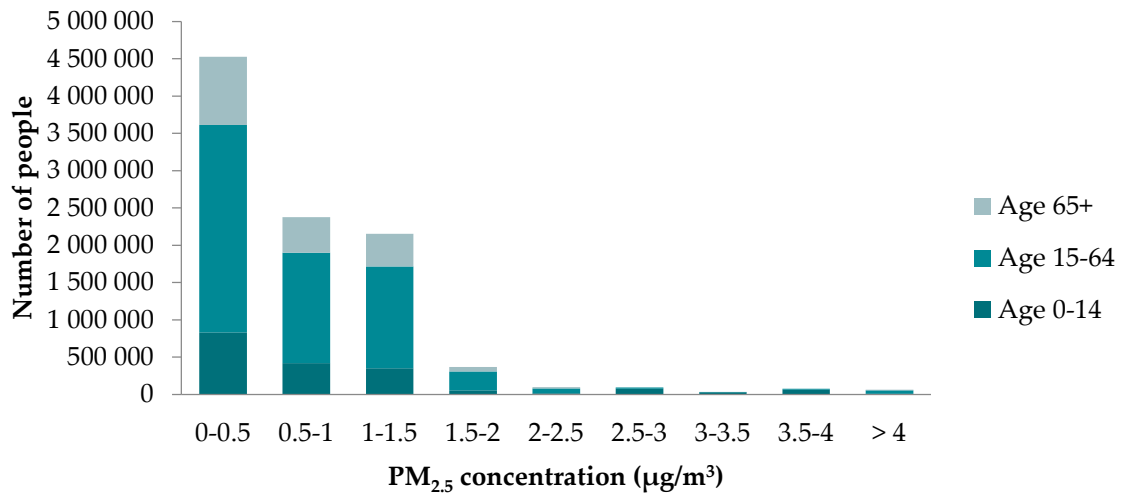
Figure 20   Distribution of exposure levels to PM2.5 annual mean concentrations from *wood burning* in the Swedish population in 2015, divided into the age categories 0 – 14 (dark blue), 15-64 (blue), and 65+ years of age (light blue).
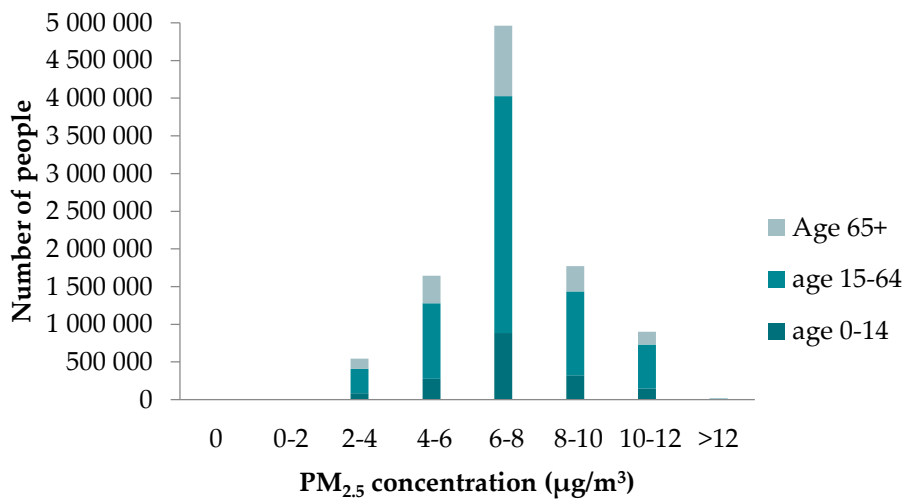
Figure 21   Distribution of exposure levels to PM2.5 annual mean concentrations from *long range transport* in the Swedish population in 2015, divided into the age categories 0 – 14 (dark blue), 15-64 (blue), and 65+ years of age (light blue).

4.3   Trends in population exposure

In Table 6 the population exposure to NO2 and particles in ambient air calculated for the years 2005, 2010 and 2015 respectively are summarized.

Table 6 Calculated population exposure to NO_2 and particles in ambient air in 2005, 2010 and 2015 respectively.

		2005	2010	2015
Total population		8 899 724	9 546 546	9 839 105
Mean population weighted exposure ($\mu g/m^3$)	NO_2	6.3	6.2	6.4
	PM_{10}	13.0	12.0	12.5
	$PM_{2.5}$	9.8	8.6	8.3
Percentage of population exposed to concentrations above the environmental objective	NO_2 (20 $\mu g/m^3$)	2.3%	2.7%	2.9%
	PM_{10} (15 $\mu g/m^3$)	38%	25%	22%
	$PM_{2.5}$ (10 $\mu g/m^3$)	49%	28%	23%
Percentage of population exposed concentrations above the environmental quality standard	NO_2 (40 $\mu g/m^3$)	0%	0%	0%
	PM_{10} (40 $\mu g/m^3$)	0.4%	0.3%	0.3%
	$PM_{2.5}$ (25 $\mu g/m^3$)	0%	0.6%	0.6%

4.3.1 NO_2

Figure 22 illustrates the percentage of the population exposed to NO_2, divided into concentration classes of 5 $\mu g/m^3$, in the six studied years. A trend towards an increasing part of the population exposed to lower concentration levels can be observed as an increased percentage of the population is exposed in the lower two categories, while exposure in the higher categories is reduced compared to exposure in the previous studies.
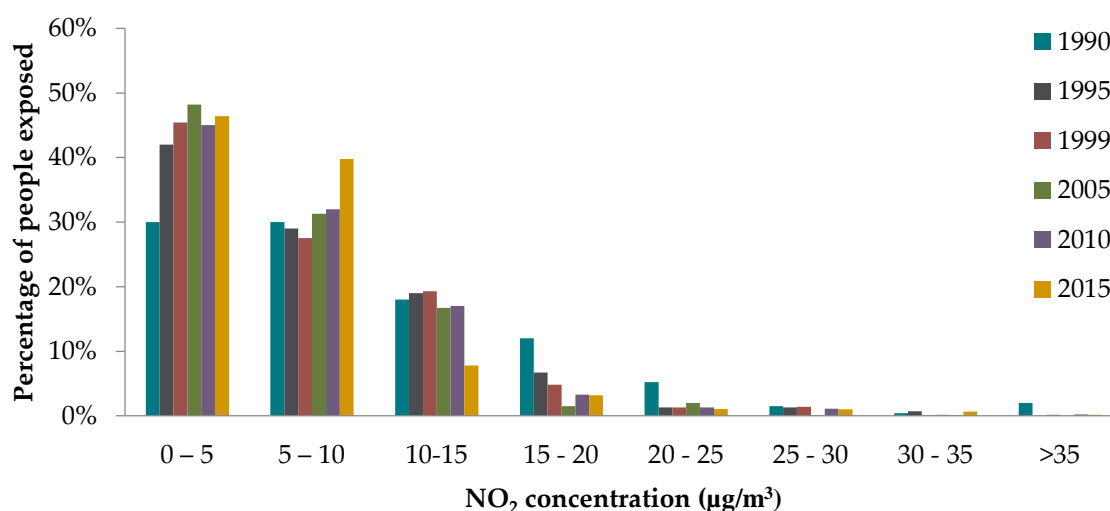
Figure 22 Percentage of the population exposed to NO_2 ($\mu g/m^3$) annual mean concentrations in 1990, 1995, 1999, 2005, 2010 and 2015.

4.3.2 Particles

Comparing the results from this study with the exposure data from the previous 2010 report (Gustafsson et al., 2014) some changes in the concentration distribution can be seen. In 2015, the exposure increased in the 10-15 μg/m³ PM_{10} concentration category while decreased in both the lower and higher categories (Figure 23). This contradicting trend can also be seen in Table 6, where a slight increase is shown in the mean population weighted exposure, while the percentage of the population exposed to concentrations above the environmental objective has decreased. This shift towards an increased population percentage in the category just below the environmental objective for PM_{10} (15 μg/m³) reduces the percentage of the population exposed to concentrations above the objective, at the same time as the mean population weighted exposure increases.

The comparison for $PM_{2.5}$ yielded similar results to PM_{10} (Figure 24), though for $PM_{2.5}$, both the mean population weighted exposure and the percentage of the population exposed to concentrations above the environmental objective decreased since the 2010 report.
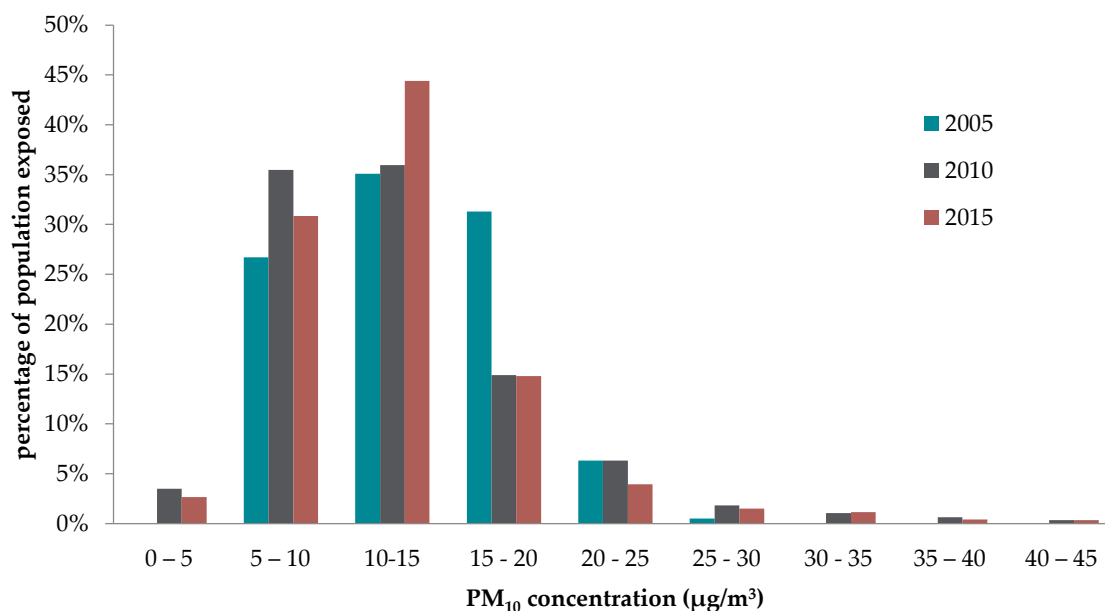
Figure 23   Percentage of the population exposed to PM_{10} (μg/m³) annual mean concentrations in 2005, 2010 and 2015.
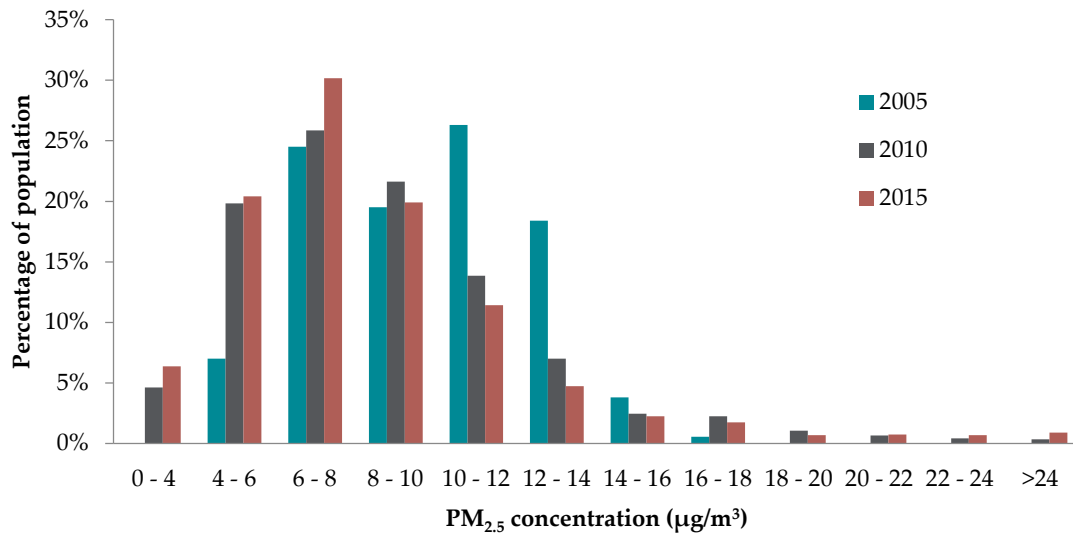
Figure 24   Percentage of the population exposed to PM₂.₅ (μg/m³) annual mean concentrations in 2005, 2010 and 2015.

4.4   Estimated health impacts

4.4.1   Mortality

4.4.1.1 Effects associated with exposure to vehicle exhaust and NO_2

We have estimated the excess mortality associated with long-term exposure to urban (local source) NO_2 without assuming any threshold below which there is no association. This urban fraction of NO_2 is not correlated with the regional (background) concentration of particles, but is dominated by local emissions from motor vehicles. We consider urban NO_2 as a good indicator of exhaust levels, why effects of co-pollutants such as ultrafine particles (e.g. soot particles) can be included in the estimated impact. However, the estimated impact on mortality is independent of the regional background levels of PM₂.₅, as well as not including the short-term effect associated with road dust (PM₁₀).

The urban (local) NO_2 contribution, with a population weighted mean of 4.4 μg/m³ in the age group 30+, is associated with 2848 deaths per year (95% CI 374 - 4792).

With the age-specific baseline mortality in Sweden 2015, the estimated number of years of life lost (YLL) due to these deaths is 31756 (95% CI 4097 - 54256), or close to 11.2 years per preterm death. This impact would also correspond to a reduction of the life expectancy of 0.3 years.

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4.4.1.2 Effects due to exposure to particle mass (PM$_{10}$ and PM$_{2.5}$)

We have estimated the excess mortality due to exposure to regional background (long-distance transported) PM$_{2.5}$, particles not emitted from the local sources such as traffic and domestic heating.

When we for regional background PM$_{2.5}$ assume the same increase in risk regardless of source (6.2% per 10 µg/m³) without any cutoff (since exposure is nowhere less than 2 µg/m³), we 2015 estimate 3616 deaths (95% CI 2375-4760) in the age group 30+.

With the age-specific baseline mortality in Sweden 2015, the estimated number of years of life lost (YLL) due to deaths attributed to regional PM$_{2.5}$ is 34424 (95% CI 22406 – 45706), or close to 11.2 years per preterm death. This impact corresponds to 0.4 years shortening of the life expectancy of the Swedish population.

Regarding the different sources, for 2015 we estimate from residential wood burning (PM$_{2.5}$) 935 deaths (95% CI 292 - 1577) in the age group 30+, and from road dust (PM$_{10}$) 215 deaths (95% CI 26 - 402) in all ages. The impact on mortality from vehicle exhaust particles we assume to a large extent included in the estimate for local NO$_2$ (see above). The estimated number of deaths associated with the regional background PM$_{2.5}$, with residential wood burning, and with road dust (PM$_{10}$), could likely be added to the estimated number of deaths associated with NO$_2$ from local sources (mainly motor vehicles) without serious double counting.

4.4.2   Morbidity effects

4.4.2.1 Effects due to exposure to particles (PM$_{10}$ and PM$_{2.5}$)

We assume that total PM$_{2.5}$ (exposure levels of at least 2 µg/m³) has an impact on RADs, and have to assume the same effect from all sources. The effect in the total population is assumed to be 7513140 RADs per year. In the age group 15-64 we estimate 4772393 RADs or work loss days per year.

We assume total PM$_{10}$ (minimum exposure levels are above 3 µg/m³) to have an impact on the incidence of Chronic Bronchitis in the age group 30+, and estimate 723 cases per year. In 2010 the mean PM$_{10}$ exposure was very similar but approximately 4 times more cases of CB per year were estimated. This large difference depends on the changed baseline incidence used for the impact calculation. We now use a Swedish baseline instead of the older incidence estimates from Switzerland and USA.

Because the risk functions are from analyses of within city contrasts in exposure, we assume only local PM to impact CVD morbidity in the age group 30+. We estimate annually 774 incident cases of myocardial infarction (95% CI 69-1496), and 874 cases of stroke (95% CI 35-1962).

4.5   Socio-economic costs

The socio-economic costs in Sweden 2015 caused by health effects linked to elevated levels of PM and NO$_2$ are estimated to ~56 billion SEK$_{2015}$, out of which 76% are due to premature fatality and 3% to long term illness after myocardial infarction and stroke (Table 7). By assuming that the natural contribution to PM$_{2.5}$ exposure is 2 µg/m³, the excess mortality due to long-range transport

of anthropogenic $PM_{2.5}$ is 94% of the total mortality from $PM_{2.5}$ exposure. Since health impacts from NO_2 exposure was calculated only on exhaust emissions, no correction for anthropogenic contribution was needed for this pollutant.

Table 7   Annual socio-economic costs of high long term air pollution levels in Sweden, 2015.

	Socio-economic cost of health Effect [million SEK$_{2015}$ / case]	Health effects from anthropogenic sources	Socio-economic cost [million SEK$_{2015}$]
Total Sweden			55 509
Out of which:			
Value of prevented fatality (VSL*/VPF) (11 years of prolonged life)	5.66	7 412	41 938
RAD (age group 0-14, 65-)	0.001	2 590 006	1 378
RAD (15-64)	0.001	4 509 911	6 678
Chronic Bronchitis	2.20	723	1 589
MI incidence	0.43	774	332
Stroke incidence	2.03	874	1 772
MI illness	1.50	774	1 163
Stroke illness	0.76	874	661

*VSL = Value of Statistical Life

5 Discussion

The exposure of Sweden's population to NO_2, PM_{10} and $PM_{2.5}$ has been calculated using the URBAN model. This is a recurring study presented every five years. In the following chapter, the resulting pollutant concentrations will be compared to previous calculations as well as other studies, followed by a discussion of the expected exposure-related health effects and the resulting costs.

5.1   Pollutant concentrations

The resulting pollutant concentrations in 2015 were overall considerably lower compared to the environmental standard for the annual mean, 40 μg/m³, for both NO_2 and PM_{10}. However, in some parts, mainly in southern Sweden, PM_{10} concentrations were of the same magnitude as the environmental objective of 15 μg/m³ as an annual mean.

Measurements of air quality have over the past decades present a trend towards reduced NO_2 and PM concentrations (Olstrup et al., 2018), however since 2004 this trend have leveled out for NO_2 due to increasing traffic and the use of diesel vehicles (Fredriksson et al., 2016, Naturvårdsverket 2017). In line with this, the population weighted exposure to NO_2 calculated for 2015 indicated very small differences in comparison to the 2010 calculation. In addition to a stagnating NO_2 concentration trend in urban areas, another factor preventing a reduction in population weighted

exposure concentrations is that the great majority of the population growth has occurred in densely populated areas (SCB, 2015). This, in combination with an ongoing densification of existing urban spaces (e.g. Boverket, 2016; SKL, 2015), result in a growing number of people being exposed to the higher NO_2 concentrations.

The mean population weighted exposure to PM for 2015 also showed similar numbers as the 2010 calculations. However, there are clear indications of improved air quality, in regards to PM, as the part of the population exposed to levels below the environmental objectives as annual means have increased with 5% and 3% for $PM_{2.5}$ and PM_{10} respectively. This pattern can be explained by the decreasing tend in the overall PM concentration in combination with an increasing population in urban areas where the highest PM concentrations are found.

The mean population weighted exposure concentrations of NO_2 calculated for all of Sweden in this study (6.4 µg/m³) were similar to concentrations found in eastern Sweden by Östra Sveriges Luftvårdsförbund (on average 5.8 µg/m³, Lövenheim, 2017). The same comparison for PM_{10} showed up to 30% higher concentrations in this study. This is likely a result of the differences in spatial extent of the two studies, where the generally higher input of long range transported particles and sea salt on the Swedish west coast causes higher particle levels on a national level, compared to if only the eastern parts are considered.

The results from this study indicate that Sweden has a very good air quality in comparison with the average exposure situation in urban Europe presented in a report by the EEA (2017). The EEA report indicated that around 8% of the European population is exposed to both NO_2 and $PM_{2.5}$ concentrations exceeding the environmental standard for the annual mean. The same numbers calculated here for Sweden were 0, 0.3 and 0.6% for NO_2, PM_{10} and $PM_{2.5}$ respectively. However, exposure in both this and the EEA study are estimated based on background concentrations to allow application of dose-response functions for the general population. As previously mentioned, higher concentrations are often found at roadside locations due to emissions from, for example, traffic within the street canyon. It is therefore likely that calculations would return higher exposure levels if based on roadside instead of background concentrations. However, as very few dose-response functions are based on roadside concentrations it is not possible to evaluate this in exposure assessments.

The calculated contribution from different sources to the total particulate matter concentrations differ somewhat from those presented by for example Segersson et al., (2017). In their study, Segersson et al. attribute a yearly contribution from domestic wood combustion between 14 and 20% of $PM_{2.5}$ in the Swedish cities Gothenburg, Stockholm and Umeå. A European estimate is that on average 22% can be attributed to domestic wood burning (Karagulian et al., 2015). In this study, the contribution from wood combustion varied over the year but was generally considerably lower, reaching a maximum of 18% in winter but only amounting to 1% in the summer. Although numbers are not directly comparable due to differences in calculation methods and scale, the indicated differences are likely also due to uncertainties in the underlying data, as wood burning activities generally are rather poorly documented.

The part of PM_{10} attributed to traffic in this study (between 22 and 51% depending on month) is difficult to compare to other studies as, for example, scale, land use type and size fraction differs (e.g. Segersson et al., 2017; Karagulian et al., 2015). However, a coarse comparison indicates that contribution from traffic is higher in this study than in other studies.

As in all studies, the method used to determine concentrations and exposure contains uncertainties. One uncertainty in this study is that the empirical model used for calculating pollution concentrations requires a reliable and relatively dense monitoring network providing measurement data. While the Swedish monitoring network is reliable, the density of the network could be improved. The characteristics of the network have also changed significantly since 1990, impacting on the type of data available for exposure studies. During the 1990's the Swedish network was primarily made up of daily mean measurements of NO_2, sulphur dioxide (SO_2) and black smoke in urban background locations. The introduction of EU air quality directives during the 2000's led to a transition to automatic measurements, including PM, located primarily in roadside locations. This change is beneficial for monitoring the highest exposure environments, but has disadvantages for the methodology applied in this study, which is based on measurements of NO_2 concentrations in urban background locations. The NO_2 concentrations and the corresponding exposure situation have been calculated in this repeated study for the calendar years 1990, 1995, 1999, 2005, 2010 and 2015. Since 2005 this study was extended to include PM. The change in number of monitoring sites in regional and urban background with data available for NO_2 can be seen in Table 8. To compensate for this reduction in available data, measurements from the Swedish Throughfall Monitoring Network have been used in this year's study as a complement to the existing regional background stations. An extension of the existing Swedish monitoring network to include more background stations, particularly in urban areas, would be highly beneficial for verification of this and other similar exposure studies

Table 8   The number of regional and urban background monitoring sites with data available for NO_2 for the different years

Year	NO_2, Regional background sites	NO_2, Urban background sites
1990	5	62
1995	20	40
1999	73	45
2005	73	41
2010	17	41 (18)*
2015	17+17**	18

*18 stations with data for a complete year.
**17 stations were added from the Swedish Throughfall Monitoring Network

The assumption that the NO_2 and PM concentrations are proportional to the number of people in a grid cell fails to capture the spatial patterns of roads, where PM emissions are significant. However, a comparison between this approach and modelling with a higher spatial resolution showed similar population exposure results (Sjöberg et al., 2009; SLB, 2007). 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 for larger cities, and apply the dispersion pattern to the URBAN model.

The method used to estimate PM_{10} concentrations in urban areas, based on the relation to the levels of NO_2, has earlier been applied by i.a. UK (Muri, 1998). The relationship was adjusted to Swedish conditions, reflecting both latitudinal and seasonal variations. A comparison between the calculated PM_{10} concentrations, based on the PM_{10}/NO_2 ratio, and monitoring data in urban

background has shown good agreement (Sjöberg et al., 2009). However, sources of NO_2 and PM are not always the same, for instance long-range transport is the dominating source to the particle levels observed in Sweden, whereas the main sources of NO_2 are traffic and energy production. Since there are only four sites in Sweden where PM is measured in regional background it is difficult to, with certainty, estimate the contribution from long-range particle transport and sea salt. This does impose some uncertainty into the particle modelling. Additionally, sea salt influences the $PM_{2.5}/PM_{10}$ ratio in coastal areas likely resulting in an over estimation of $PM_{2.5}$ in these regions. However, the influence of sea salt decreases quickly moving inland. For example, according to the EMEP model the average sea salt concentration along the west coast of Sweden was on average 1.3 $\mu g/m^3$ whereas inland the contribution was approximately 0.1 $\mu g/m^3$.

5.2   Health effects

Time-trends in estimated health impacts of air pollution exposure are driven by many other factors than concentrations or population exposure. Population size and base-line frequency of the studied outcome are both important for the estimated numbers. The applied risk function and any assumed low threshold (below no effects are expected) are also important factors that may change as our knowledge improve.

Assessment of health impacts of particle pollution is difficult since PM is a complex mixture where different components very likely have different toxicity. However, WHO in HRAPIE (WHO, 2013b) and other assessments, lacking evidence enough for differential quantification, still choose 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. For this reason we apply different exposure-response functions for particles from residential wood burning, road dust and for the regional background of mainly secondary particles.

The recent WHO review REVIHAAP (WHO, 2013a) states that recent long-term studies show associations between $PM_{2.5}$ and mortality at levels well below 10 $\mu g/m^3$, and thus concludes that for Europe it is reasonable to use linear exposure-response functions and to assume that any reduction in exposure will have benefits. This conclusion from REVIHAAP is also incorporated as a basic assumption in HRAPIE (WHO, 2013b).

Regarding long-term exposure and mortality the REVIHAAP report also concludes that more studies have now been published showing associations between long-term exposure to NO_2 and mortality (WHO, 2013a). This observation makes the situation a bit more complicated when it comes to impact assessments for vehicle exhaust particles, where the close correlation between long-term concentrations of NO_2 and exhaust particles may result in confounding in epidemiological studies evaluating NO_2 and particles separately.

The potential confounding problem in studies of effects from NO_2 and $PM_{2.5}$ on mortality was dealt with in a recent review paper focusing on 19 epidemiological long-term studies of mortality using both pollutants as exposure variable. In the review, studies with two-pollutant models ($PM_{2.5}$ and NO_2 in the same model) showed some decrease in the effect estimates of NO_2, however still suggesting partly independent effects. One problem with such analyses is that the association between NO_2 and mortality could partly be caused by exhaust particles, which usually form only a smaller fraction of $PM_{2.5}$. In such a situation it could cause a problem of double counting if

mortality impacts of both PM$_{2.5}$ and NO$_2$ are estimated and added, and would very likely do if impacts estimated for PM$_{exhaust}$ (or diesel soot) and NO$_2$ were added.

For long-term exposure to NO$_2$ and mortality (30+) the WHO HRAPIE impact assessment report (WHO, 2013b) recommended a RR of 1.055 (95% CI 1.031-1.08) from the meta-analysis of 11 studies by Hoek et al. (2013). Because of the potential confounding and double counting of mortality effects from PM$_{2.5}$, the HRAPIE report stressed more uncertainty about quantification of NO$_2$ effects from single-pollutant models. The HRAPIE report also recommended to use the RR from Hoek et al. only above the annual mean 20 µg/m^3, a recommendation later seen as too conservative by the same group of experts after having noted the results from places with low levels (Heroux et al., 2015). In fact, the three studies in the meta-analysis with *the lowest concentrations* (mean 20 or lower) reported *higher relative risks* than the combined meta estimate (Hoek et al., 2013). The recent EEA report calculated national impacts in 2014 using counterfactual annual mean NO$_2$ concentrations of 20 and 10 µg/m^3. For Sweden this lead to 130 and 990 estimated premature deaths per year, respectively in the European report (EEA, 2017).

The UK COMEAP working group in their interim recommendation to DEFRA (COMEAP, 2015) stated that there is uncertainty in the extent to which the association between long-term average concentrations of NO$_2$ and mortality is causal: "It is likely that some of the effect is due to NO$_2$, but other co-emitted pollutants could also be responsible to some extent." Based on a meta-analysis a coefficient of 1.025 (95% CI 1.01–1.04) per 10 µg/m^3 was recommended. However, the recommendation was also in an assessment which also includes PM$_{2.5}$, to reduce this coefficient by up to 33% to take account of possible overestimation due to double counting of effects associated with PM. The recommendation also said "As there is no clear evidence for a threshold of effect at the population level, a zero cut-off for quantification is recommended for use in the main calculation. For sensitivity analysis, the working group intends to use the lowest concentration in studies in which associations were found, as a cut-off (to be determined)."

Faustini et al. (2014) in their meta-analysis found the greatest effect on natural or total mortality in studies from Europe, the relative risk for NO$_2$ was 1.066 (95% CI 1.029-1.104) per 10 µg/m^3.

We acknowledge that the local contribution of NO$_2$ is independent of the regional background concentration of PM$_{2.5}$, which is why the impact on mortality is estimated for both exposures, and then added together to avoid double counting. We believe that the effect on mortality of vehicle exhaust exposure in this national study is better described by the local contribution to NO$_2$ levels and relative risk estimates for NO$_2$, than by estimated particle exposure from motor vehicles and relative risk estimates obtained for total PM$_{2.5}$, and not specifically for exhaust particles. We have in our impact calculations used results from a large Danish cohort study of NO$_2$ and mortality, which was not included in the review by Faustini et al. (2014). The relative risk is somewhat higher than the European meta-estimate, 8 vs 7% per 10 µg/m^3, but we assume the conditions in the Danish study most relevant for Sweden (Raaschou-Nielsen et al., 2012). Our estimate for local NO$_2$, 2848 deaths per year, would become 2350 deaths with the meta coefficient from Faustini et al. (2014) and 1958 deaths annually with the risk coefficient 5.5% per 10 µg/m^3 (Hoek et al., 2013) used by EEA (2017). If the calculation for 2010 in our previous report had been for local NO$_2$, the estimated number of associated deaths per year had been 2878 with the smaller population and higher mortality baseline we had in 2010.

A study from Gothenburg of men only reported a result for NOx in the group least old at enrolment (48-52 yrs) that was close to the other findings (Stockfelt et al., 2015), and would have resulted in rather similar impact estimates.

Which, if any, cutoff level to use in a health impact assessment like this is rather arbitrary, since we do not exactly know the natural background levels nor the shape of the exposure-response association in the lowest concentration intervals. There is no evidence of a specific toxicological threshold at population level to support a specific cutoff level. For the assessment of impacts associated with the urban contribution and different local policies, this question is not critical, since it is exposure upon the regional background. Also when the total burden from PM exposure is estimated it has become quite common to use 0 or a low cutoff, because exposure-response functions down to very low levels have been shown.

The long-term impact of total $PM_{2.5}$ on mortality was estimated without any cutoff and with a cutoff at 2.5 μg/m³ in the EEA report (2017), for 2014 resulting in estimated 3710 and 2510 deaths, respectively, in Sweden. If we would use our total $PM_{2.5}$ exposure and assume impacts to start at 2.5 μg/m³ as in the EEA report, we estimate 2832 deaths. Without the cutoff we estimate 4066 deaths, approx. 350 more than EEA. However, for our main analysis we prefer not to use any cutoff for $PM_{2.5}$ or PM_{10}, because of weak epidemiological evidence of any threshold and since there are in our data no annual exposure levels lower than 2 and 3 μg/m³, respectively. We have estimated 3616 deaths per year associated with the regional background levels of the impact on mortality, using the same risk coefficient as used by EEA, but excluding the local contribution to $PM_{2.5}$ exposure because we separately estimated effects on mortality of local emissions from traffic and heating.

The assessment of health impacts using $PM_{2.5}$ as exposure indicator is most valid for the regional background particle pollution. At first, background PM is largely built up by secondary particles, where a large part originates from remote sources. Secondly, the most often 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. However, as suggested in this study 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 tires are used. NO_2 is on the other hand in most areas a good indicator of air pollution from motor vehicles. Even if the exhaust particles are thought to contribute much to the health effects in cities, the health effects from local-regional gradients in vehicle exhaust are likely better studied using NO_2 or NO_X as an indicator, rather than using levels and risk functions for total particle mass. Thus, in addition to estimates for road dust, a calculation using NO_2 is therefore a better indication of the magnitude of the mortality effects from traffic in Sweden than to use estimates for exhaust PM. This way, the estimated number of deaths associated with NO_2 from local sources could be added to the estimated number of deaths due to regional background $PM_{2.5}$, road dust and residential wood burning with minor risk of double counting.

The, in our previous reports, included estimation of respiratory and cardiovascular hospital admissions due to the short-term effects of $PM_{2.5}$ and NO_2 gave a low number of admissions 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 was estimated, and thus not the whole effect on hospital admissions following morbidity due to air pollution exposure (Künzli et al., 2008). The total yearly number of acute events in persons that developed their disease due to air pollution exposure may be 5-10 times higher (Perez et al., 2013). Thus, in this report on 2015 we have included impacts in terms of new cases (survivors) of stroke and myocardial infarction. It would be valuable to have even more morbidity indicators also for other long-term effects of air pollution exposure.

In the report for 2010 we estimated around 5500 deaths per year in Sweden associated with regional background $PM_{2.5}$, road dust PM_{10}, $PM_{2.5}$ from wood smoke and NO_2 as a marker of vehicle exhaust. In this report we estimate almost the same impact from road dust and wood smoke, but more deaths associated with the regional $PM_{2.5}$ background and the local NO_2 contribution, resulting in a total of approx. 7600 deaths per year. This increase in the estimate, by more than 2000 deaths per year, does not reflect an equal increase in exposure. Instead the most important explanation is that we have not applied any cutoff level below which background $PM_{2.5}$ and local NO_2 are assumed to have no impact on mortality. Assuming a cutoff or a lower contribution from sea salt would result in lower estimates, but there is little support for any threshold effect in the literature.

Ozone has not been included in this study, but has also an impact on preterm deaths and causes also other adverse health effects.

5.3 Socio-economic costs

Our estimated socio-economic costs from elevated levels of air pollution are higher than our estimate for 2010. This is partly due to new epidemiological knowledge which induced new approaches to economic valuation. For comparison with earlier estimates, Table 9 presents the socio-economic costs of health effects when using the method and values in 2010 (Gustafsson et al., 2014). For comparability with Gustafsson et al. (2014) we have in Table 9 excluded mortality associated with road dust (215 cases in 2015) and assumed linear relationship between mortality associated with $PM_{2.5}$ exposure and hospitalisation.

Table 9   Annual socio-economic costs of long-term air pollution levels in Sweden 2015, using the same method and economic values as in Gustafsson et al. (2014).

	Socio-economic cost of health Effect [million SEK2010 / case]	Health effects from *anthropogenic sources*	Socio-economic cost [million SEK2010]
Total Sweden			40 377
Out of which:			
Value of prevented fatality (VSL/VPF) (11 years of prolonged life)	5.48	5 887	32 258
Chronic Bronchitis	2.13	723	1 539
Hospitalisation, cardiology	0.05	1 528	71
Hospitalisation, generic (respiration)	0.03	1 565	43
RAD (age group 16-64)	0.00	4 509 911	6 467

In Gustafsson et al. (2014) we estimated total socio-economic costs to be ~42 billion SEK2010 per year. So the total socio-economic costs when calculated with the same method (Table 9) are relatively stable between 2010 and 2015.

Given that we in this study have added new values for myocardial infarction and stroke, including non-use costs of long-term illness, it is relevant to see how much our final results would vary if

other assumptions would be made. In a sensitivity analysis we therefore assumed that the US healthcare cost amplifier should apply to all cost items when valuing costs and set costs of long-term illness after myocardial infarction or stroke to zero. The results from the sensitivity analysis showed that the socio-economic costs of 2015 exposure to air pollution would be ~54 billion SEK_{2015} (~56 billion SEK_{2015} in our central estimate, Table 7). If however, one would consider all $PM_{2.5}$ exposure as caused by anthropogenic activities, the corresponding socio-economic costs would be ~57 billion SEK_{2015}. Also interesting, is that the number of RAD caused by anthropogenic air pollution in the population of working age corresponds to ~0.4% of all work days in Sweden 2015, with an approximately similar impact on GDP.

One of the most controversial aspects of valuing socio-economic effects of air pollution is the value assigned to air-pollution related fatalities. Our approach is based on estimating the number of life years lost per fatality and multiply these with values of a life year lost (VOLY) from the literature. The number of life years lost per fatality is ~11 years and the original VOLY we use is $€_{2000}$ 40 000, a much lower number than in newer estimates. One of the more interesting controversial aspects of valuing fatalities in monetary terms in general, and using VOLY to value fatalities associated with elevated levels of air pollution in particular, is that that a VOLY-based approach implies that old (or sick) persons are worth less to society than the average person (Ackerman & Heinzerling, 2005). Such an implication is hard to defend from a moral stand point, and we therefore made a sensitivity analysis in which we replaced our VOLY approach with a VSL approach. If using recent ASEK values for VSL (SRA, 2015) of 23 million SEK_{2015} the socio-economic costs of air pollution in 2015 would be ~185 billion SEK_{2015}. If using recent Danish estimates (Navrud, 2016), socio-economic costs of air pollution in 2015 would be ~294 billion SEK_{2015}. Clearly, our approach resulting in a socio-economic cost from elevated air pollution levels of 56 billion SEK_{2015} is cautious.

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