



Traffic related PM and mortality
- exposure-response functions
and impact calculations for TESS

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Preface

This is a report from the research project TESS – *Traffic Emissions, Socio-economic valuation and Socio-economic measures*, which is funded by EMFO. In 2002 an agreement about the EMFO program was reached between the partners from the Swedish Vehicle Research Council, PFF. EMFO is a sector-wide research competence to develop vehicles and vehicle components with emission levels that are sustainable in the long term. The aim of EMFO is to offer academia, industry and authorities access to necessary knowledge and pioneering solutions that are necessary if vehicle technology is to develop in the desired direction. One important task is to coordinate activities within the program with both national and international research in the field.

EMFO comprises subsidiary programs and two of these were: “Socio-economic evaluation of the health and environmental impact of different emissions” and “Optimal range of socio-economic measures”. TESS undertakes research in these two areas but it is also related to the subsidiary program: “Health and Environmental Impact”. The application was approved in 2005 and the project runs during 2005-2008.

The basis for the research in TESS is the valuation methods developed in the EU funded ExternE projects where the external cost of emissions is calculated by tracing the effects that the emissions have on human health and then valuing these effects. The aim is to calculate the external costs related to particles that local emissions (from traffic and other sources) generate on a local and regional scale using Stockholm as a case study. Based on this information an analysis will be made on what reductions measures are likely to be efficient from an economic point of view.

The analysis undertaken in TESS requires collaboration between researchers from different research disciplines. There are four parties involved in this project; VTI, SLB analys (Environment and Health Administration, Stockholm), Umeå University and SMHI.

This report is focused on the health impact assessment. The first aim is to overview and discuss the epidemiological basis for selection of an exposure-response function, ERF, (or several different) for traffic related particles and mortality. The second aim is to use the exposure calculations by SLB and SMHI presented in TESS to make impact calculations using the selected exposure-response assumptions for particles of three types; exhaust particles, road dust particles and secondary particles (especially nitrates).

General background

It has long been recognized that emissions from traffic have a negative impact on human health. In latter years there has been emerging consensus that the main influence is due to particulate matter (WHO, 2005). From an economic point of view these negative effects are external costs caused by traffic that, if not accounted for in decision making regarding transport, will result in a non-optimal allocation of resources leading to welfare losses. There are however various measures in place aimed at reducing the negative health impact (i.e. the external costs) of the emissions from traffic. The measures include emission control legislation but also air quality objectives for local concentration levels in urban areas that if exceeded compels the local authorities to take action. Also road pricing measures are increasingly considered as an option since the new information technology has opened up for new technical solutions. One such example is the Stockholm trial where rush hour road pricing was implemented, resulting in reduced traffic to and within the city area and thereby reductions in emissions and concentration levels.

To be able to implement road pricing measures, but also for the evaluation of other control measures through benefit-cost analysis, information on the external cost of traffic emissions is needed. In the Impact pathway approach (IPA), that has been developed in the ExternE projects, the external cost is calculated as the product of exposure, effect and value. All these inputs are the result of ongoing empirical research and they are all related to uncertainties, hence the external cost that is calculated is not “the” cost. Regarding particles there is for example recognition among the research community that there are different types of particles and that it is likely that their impact on human health differ (WHO, 2006a; WHO, 2007). Still the current practice is to treat fine particles (which are considered to be most detrimental to health) as equally harmful irrespective of origin. Hence, there is often only one assumed exposure-function used for assessing the health impact of fine particles (so called PM_{2.5}).

In previous ExternE reports (European Commission, 1999; ExternE, 2000), the assumption was made that the long-term effect on mortality of all sulfates is equal to that of PM_{2.5} and the toxicity of particulate nitrates equal to that of PM₁₀. The assumed ratio of exposure-response slopes for PM₁₀ versus PM_{2.5} was 0.6, because this is a typical value of the ratio of concentrations of PM_{2.5} and PM₁₀. ExternE earlier treated power plant emissions as being equivalent in toxicity to PM₁₀ and vehicle emissions as equivalent to PM_{2.5}.

For the current version of ExternE (2005), the assumptions about the toxicity of the different PM types have been changed to reflect newer evidence that indicates high toxicity of combustion particles and especially of particles from internal combustion engines. ExternE now treats nitrates as equivalent to half the toxicity of PM₁₀; sulfates as equivalent to PM₁₀; primary particles from power stations as equivalent to PM₁₀; primary particles from vehicles as equivalent to 1.5 times the toxicity of PM_{2.5}. The long-term effect on mortality of PM_{2.5} has from ACS (see below *Cohort results for long term exposure and mortality*) been assumed to be 1.06 (6 %) for a 10 µg/m³

increment of average PM_{2.5}. For example was this assumption used in the Clean Air For Europe (CAFE) and calculations on effects of transboundary particle pollution (WHO, 2006b).

However, in most urban areas it is the concentration of PM₁₀ that is measured since the current air quality limits are based on this fraction including ultrafine soot particles, fine secondary particles as well as more coarse particles of mainly crustal origin. The most important local source of PM₁₀ in many urban areas in Sweden is road dust, including wear particles, sand etc. In spring, when the roadways are dry, the contribution from road wear particles may be 30 times the direct emissions from the exhaust pipe. These mechanically generated road dust particles are not considered in calculations of the external cost that is based on the original ExternE-methodology (see for example ExternE methodology update and HEATCO-results).

It is rather unsophisticated to lump together all kind of particles in PM₁₀. In reality the measured concentrations of both PM_{2.5} and PM₁₀ in an urban area are composed of several different kinds of particles such as combustion particles from different sources, of non-exhaust particles from road wear and of secondary particles from sources outside the city. In order to undertake analysis of the influence of traffic emissions on exposure, dispersion models are used to model the contribution of exhaust and non-exhaust and secondary particles. One problem is that epidemiological studies usually build on measured mass concentrations of PM₁₀ or PM_{2.5}, both influenced by several sources but not so correlated to exhaust particles. Instead nitrogen oxide concentrations are highly correlated with the number of exhaust particles, why NO_x or NO₂ can be good indicators for the exposure to exhaust particles.

Mortality effects from different types of particles

The far most important effect of particles on health in previous assessments, and the largest health cost, is related to the long term effect on mortality and the so called excess deaths or life years lost. However, there is very little information on long term exposure to various types of particles in ambient air and mortality, why the short-term effects on mortality of different types of particles also must be considered as useful indications of the differences in harmfulness. It is likely that there is a good correlation between the potency to influence daily mortality and the long term effect on mortality, since the deaths in cardiovascular disease are many more than the deaths in lung cancer, that may be one exception. Thus, this paper will review first the studies of short-term exposure and daily mortality and then the cohort studies of long term exposure and excess mortality.

Exhaust particles and daily mortality

A number of European time-series studies indicate stronger short-term effects on daily number of deaths when road traffic involving a large fraction of diesel cars is a major source. This is suggested by the positive relation to levels of NO₂ in 29 European cities

(Katsouyanni et al, 2001; Samoli et al, 2005) and the stronger effects among those living along busy streets (Roemer and Wijnen, 2001). Elemental carbon (EC), as an indicator of combustion particles, especially diesel exhaust, was significantly associated with cardiovascular mortality in a time-series study from Phoenix (Mar et al, 2000).

Several studies from USA, where most cars are gasoline cars, have also found larger effects of particles originating from road traffic. In a reanalysis of the large project NMMAPS Zeka and Schwartz (2004) used hierarchical modeling to deal with potential bias related to differences in the size of measurement errors for different pollutants. The greater effect for CO on daily mortality then seen may according to the authors reflect an effect of CO itself, or that CO here was a surrogate for traffic particles.

A meta-analysis of the daily number of deaths in “The Harvard Six Cities” using the elemental composition of size-fractionated particles to identify source-related fractions of fine particles, found in the combined analysis PM(2.5) from mobile sources to produce the strongest effect on daily mortality (Laden et al, 2000).

In three New Jersey cities daily mortality was investigated after a factor-analysis had resolved source-related factors (Tsai et al, 2000). Statistically significant associations were found between mortality and the sources oil burning, industry (Zn/Cd), sulfate and motor vehicles, while PM from geological sources was not significant.

In a time-series analysis of daily mortality in Phoenix, Mar et al (2000) used gases and detailed PM composition data. In this study total mortality was significantly associated with indicators for traffic exhaust (CO and NO₂) and cardiovascular mortality with combustion-related pollutants in general and secondary aerosols (sulfates). In a later analysis of the apportioned anthropogenic PM_{2.5} source categories the sources with the largest cardiovascular mortality effect size were secondary sulfate and traffic (Mar et al, 2006). For total mortality, the associations were weaker.

Ito and coworkers (2006) concluded after an examination of reported associations between daily mortality and source investigators' estimated source-apportioned PM_{2.5} for Washington, DC for 1988-1997 that risk estimates for traffic-related PM_{2.5} were significant in some cases but more variable than the estimates for secondary sulfate and oil-burning (Ito et al, 2006).

Road dust, crustal particles and daily mortality

There are in the literature only a few studies of road dust or the coarse fraction when studded tires are used and road dust has its greatest contribution to PM₁₀, and these studies do not focus on mortality. However, there are some studies of coarse and crustal particles and mortality. In the Six Cities Study the mass of crustal particles (using Silicon as a tracer) was not associated with daily mortality (Laden et al, 2000). In that study the elemental profile of the crustal factor was qualitatively similar to published chemical analysis of road dust. Also a study of daily mortality in Phoenix found no increase in

mortality with the identified soil factor, but the coarse fraction was positively associated with mortality (Mar et al, 2000). Also in a later analysis fine particle soil was not associated with increased risks (Mar et al, 2006).

Sulfates and daily mortality

A recent review found that sulfate generally is less frequently associated with health endpoints than is PM_{2.5} (Reiss et al, 2005). However, there are not so many studies of sulfate versus other fractions of PM_{2.5} and their results are not very consistent. There are altogether a lot of studies reporting associations with sulfate.

A time-series analysis of daily mortality and size-fractionated particulate mass and gaseous pollutants included eight large cities in Canada from 1986 to 1996, and found that sulfate, iron, nickel, and zinc from the fine fraction were most strongly associated with mortality (Burnett et al, 2000).

In Phoenix, the secondary aerosol factor was significantly associated with cardiovascular mortality but not total mortality in a similar time-series study (Mar et al, 2000). These results were confirmed by a later analysis of the apportioned anthropogenic PM_{2.5} source categories, where secondary sulfate, traffic, and copper smelter-derived particles were most consistently associated with cardiovascular mortality (Mar et al, 2006).

In the Six Cities study the sulfate-related factor had a significant effect on daily mortality in the combined analysis (Laden et al, 2000) and close to significant in the Health Effects Institute coordinated reanalysis (HEI, 2003).

In three New Jersey cities daily mortality was analysed in relation to PM from different sources according to factor-analysis, and statistically significant associations were found between mortality and several sources including sulfate (Tsai et al, 2000).

In an examination of reported associations between daily mortality and source, the investigators' estimated source-apportioned PM_{2.5} for Washington, DC for 1988-1997, and found that patterns were similar across investigators/methods, with the largest and most significant percent excess deaths per 5-95th percentile increment for secondary sulfate (Ito et al, 2006). When results from seven research institutions that studied effects of source-apportioned PM_{2.5} in Washington, DC, and Phoenix were evaluated, the sulfate-related PM_{2.5} component was most consistently significant across analyses in these cities (Thurston et al, 2005).

Nitrates and daily mortality

There are almost no epidemiological studies of particles and mortality using nitrate levels. One published study of daily mortality found no significant association between nitrate and mortality (Klemm et al, 2004). In a time-series of six California counties,

ambient concentrations of several constituents of fine particles, including nitrate, demonstrated stronger effects on daily mortality than did PM_{2.5} mass (Ostro et al, 2007).

Cohort results for long term exposure and mortality

Health impact assessments including mortality effects of long-term exposure to air pollution are mostly using the American Cancer Society (ACS) cohort results (Pope et al, 1995) to assess the impact. As it is used both in the US and by WHO and EU in the CAFE program the ACS study has become the most influential and widely cited study of particles and mortality. The original report, a reanalysis initiated by HEI (Krewski et al, 2000) and more recent analyses with longer follow up and improved exposure data (Pope et al, 2002) have all demonstrated associations between PM and all-cause and cause-specific mortality. As a consequence this robust relation and the few other studies of long-term effects, the ACS study together with the older Six Cities study (Dockery et al, 1993) have been important also for setting limit values. Two other small studies, the Southern California study (Abbey et al, 1999) and the Veterans cohort study (Lipfert et al, 2000) found in principle no statistically significant associations with PM.

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

Study	Indicator (Increment)	Relative Risk (95% CI)	Study Concentrations ($\mu\text{g}/\text{m}^3$)
Increased Total Mortality in Adults			
Six City ^A	PM _{15/10} (20 $\mu\text{g}/\text{m}^3$)	1.18 (1.06, 1.32)	NR (18, 47)
	PM _{2.5} (10 $\mu\text{g}/\text{m}^3$)	1.13 (1.04, 1.23)	NR (11, 30)
	SO ₄ ⁻² (15 $\mu\text{g}/\text{m}^3$)	1.54 (1.15, 2.07)	NR (5, 13)
Six City ^B	PM _{15-2.5} (10 $\mu\text{g}/\text{m}^3$)	1.43 (0.83, 2.48)	
ACS Study ^C (151 U.S. SMSA)	PM _{2.5} (10 $\mu\text{g}/\text{m}^3$)	1.07 (1.04, 1.10)	18 ^U (9, 34)
	SO ₄ ⁻² (15 $\mu\text{g}/\text{m}^3$)	1.11 (1.06, 1.16)	11 ^U (4, 24)
Six City Reanalysis ^D	PM _{15/10} (20 $\mu\text{g}/\text{m}^3$)	1.19 (1.06, 1.34)	NR (18, 47)
	PM _{2.5} (10 $\mu\text{g}/\text{m}^3$)	1.14 (1.05, 1.23)	NR (11, 30)
ACS Study Reanalysis ^D	PM _{15/10} (20 $\mu\text{g}/\text{m}^3$) (dichot)	1.04 (1.01, 1.07)	59 (34, 101)
	PM _{2.5} (10 $\mu\text{g}/\text{m}^3$)	1.07 (1.04, 1.10)	20 (10, 38)
	PM _{15-2.5} (10 $\mu\text{g}/\text{m}^3$)	1.00 (0.99, 1.02)	7.1 (9, 42)
ACS Study Extended Analyses ^E	PM _{2.5} (10 $\mu\text{g}/\text{m}^3$) (1979-83)	1.04 (1.01, 1.08)	21 (9, 34)
	PM _{2.5} (10 $\mu\text{g}/\text{m}^3$) (1999-00)	1.06 (1.02, 1.10)	14 (5, 20)
	PM _{2.5} (10 $\mu\text{g}/\text{m}^3$) (average)	1.06 (1.02, 1.11)	18 (7.5, 30)
Southern California ^F	PM ₁₀ (20 $\mu\text{g}/\text{m}^3$)	1.09 (0.99, 1.21) (males)	51 (0, 84)
	PM ₁₀ (30 days/year > 100 $\mu\text{g}/\text{m}^3$)	1.08 (1.01, 1.16) (males)	
	PM ₁₀ (20 $\mu\text{g}/\text{m}^3$)	0.95 (0.87, 1.03) (females)	51 (0, 84)
	PM ₁₀ (30 days/year > 100 $\mu\text{g}/\text{m}^3$)	0.96 (0.90, 1.02) (females)	
Southern California ^H	PM _{2.5} (10 $\mu\text{g}/\text{m}^3$)	1.09 (0.98, 1.21) (males)	32 (17, 45)
	PM _{10-2.5} (10 $\mu\text{g}/\text{m}^3$)	1.05 (0.92, 1.21) (males)	27 (4, 44)
Veterans Cohort ^G	PM _{2.5} (10 $\mu\text{g}/\text{m}^3$) (1979-81)	0.90 (0.85, 0.95) (males)	24 (6, 42)

^A Dockery et al. (1993)

^B EPA (1996)

^C Pope et al. (1995)

^D Krewski et al. (2000)

^E Pope et al. (2002)

^F Abbey et al. (1999)

^G Lipfert et al. (2000)

In the longer ACS cohort follow up Pope et al (2002) found a relative risk of 1.06 (6 %) due to a 10 $\mu\text{g}/\text{m}^3$ increment of average PM_{2.5}. This RR is assumed in most assessments, without adjustments, for all anthropogenic sources (as in APHEIS, the RAINS modelling for CAFE and the WHO report on LRTAP) or with various modifications (as in ExternE). In ACS there was also close correlations of the relative risk RR with the concentration of sulfates. The good association with sulfates indicates that differences in mortality may be driven mainly by secondary particles, in particular sulfates. A major shortcoming shared by ACS and the Six Cities study is the assessment of air pollution exposure using only community average concentrations from central monitors. Such monitors often reflect mainly the regional background. The approach used in these

studies “community exposure contrasts” at the scale of a metropolitan area, gives all subjects from a city the same exposure concentrations. This focus on the urban background likely underestimates the effects of more locally elevated concentrations in the vicinity of sources such as traffic. The single monitor effect of exposure misclassification has been discussed in an article by Mallick et al. (2002) that performed a hypothetical analysis that attempted to correct for such misclassification in the Six Cities Study. This analysis found two- to three-fold higher effect estimates than the originally reported.

In the long-term studies the Harvard Six Cities cohort (Dockery et al, 2003) and the American Cancer Society (ACS) II cohort study (Pope et al, 1995; Pope et al, 2002) there were significant associations between mortality and both PM_{2.5} and sulfate. In The Six Cities study the correlation between PM_{2.5} and sulfate was so high ($r=0.98$) that they reflect the same exposure contrast. In a separate study ACS data were analyzed at the county scale instead of using larger metropolitan areas (Willis et al, 2003). At the county scale, long-term exposure to sulfates was more strongly associated with increased risk of all-cause and cardiopulmonary mortality than previously indicated. In this case there was for all-cause mortality in the restricted cohort a doubling of the sulfate coefficient compared to including all individuals in the metropolitan-area. The authors of this paper stated that it provided evidence that better spatial resolution reduces exposure misclassification and leads to increased effect estimates.

A recent paper from the Veterans Administration Cohort included nitrate and found a statistically significant effect on mortality (Lipfert et al, 2006a). However, traffic density was a more robust predictor of mortality than was PM_{2.5} or sulfate (Lipfert et al, 2006b).

Recent studies using within-city gradients of PM

In a study of ACS subjects from Los Angeles County only (Jerrett et al, 2005), the authors extracted health data from the ACS Cancer Prevention II survey for metropolitan LA at the zip code-area scale (zip codes are used for U.S. mail delivery; average population per zip code in LA is approximately 35,000, with an average area of approximately 22.5 km²). They then constructed distribution-weighted centroids using spatial boundary files based on 1980 and 1990 definitions, and were able to assign exposure to 267 zip code areas with a total of 22,905 subjects. Among these subjects enrolled in 1982, 5856 had died during the follow up to 2000. The authors interpolated PM_{2.5} data from 23 state and local district monitoring stations in the LA basin for the year 2000 to derive exposure assessments. They initially used five interpolation methods and after validation they ended up using a combination of universal kriging and multiquadric models.

For all-cause mortality they found for PM_{2.5} alone and control for age, sex, and race, the relative risk per 10 µg/m³ to be 1.24 (95% CI = 1.11–1.37), whereas the RR with adjustments for 44 individual confounder was 1.17 (95% CI = 1.05–1.30). These results suggest that the chronic health effects associated with intraurban gradients in

exposure to PM_{2.5} may be even larger than previously reported associations across metropolitan areas. A direct comparison with the previous ACS results show effects that are nearly 3 times larger than in models relying on between-community exposure contrasts. It appears likely that the within-city contrast are more dependent on traffic emissions than the previously used between-community gradients to a large extent driven by sulfates related to SO₂ emissions from coal and oil burning.

The findings from this study from Los Angeles are in line with recent evidence suggesting that intraurban exposure gradients may be associated with even larger health effects than reported in interurban studies. For example, in the Netherlands a doubling of cardiopulmonary mortality (RR =1.95; 95% CI 1.09 –3.52) was found for persons living near major roads (Hoek et al, 2002). Using modeled levels of NO_x in the residential area as a marker, a Norwegian study of 16 000 men from Oslo of whom 25 % died during the follow up, found a strong association (Naftstad et al, 2004). This cohort, with people of between 40-49 years of age at the start of the study, was followed from 1972/73 through 1998. NO_x was estimated in a model with 1000 m grids, and a street contribution added for the largest streets. When the median concentration of NO_x for 1974-78 was used (10.7 µg/m³), the relative risk for total non-violent mortality was 8 % per 10 µg/m³ (95% CI 6-11%).

Modeled NO₂ concentrations in Auckland (Scoggins et al, 2004) as well as locally measured NO₂ concentrations within French cities (Filleul et al, 2005) have been used as exposure indicators in recent studies reporting an association between air pollution and mortality. In likeness to the Norwegian study, the study from Auckland, New Zealand used a dispersion model, but was an ecological cross-sectional study, where the data analysis was undertaken at the national census area unit level (Scoggins et al, 2004). In this study urban airshed modeling and GIS-based techniques were used to quantify long-term exposure to air pollution. The models were run on a 3 km grid that covered almost the entire Auckland region.

The studies from Auckland and France found deaths from non-external causes to increase by 13 and 14 % per 10 µg/m³, respectively, which based on NO₂ is in line with the Norwegian result, 8 % per 10 µg/m³ NO_x (Nafstad et al, 2004), and very close to the indicated 12 % per 10 µg/m³ NO₂ in the Dutch study (Hoek et al, 2002). These associations are very strong per unit increase in the concentration in relation to the results for PM in the ACS study if vehicle exhaust pollutants are assumed to be most important.

A recent study from Germany is a follow-up of a series of cross-sectional studies carried out during the 1980s and 1990s on the health of women aged 50–59 years (Gehring et al, 2006). Approximately 4800 women were followed up for vital status and migration. Exposure to air pollution was defined by distance to major roads calculated from Geographic Information System data and by 1- and 5-year average nitrogen dioxide (NO₂) and particle (PM₁₀) concentrations calculated from air monitoring station data. In this study elevated levels of NO₂ (1- and 5-year averages) were associated with an increased risk of all-cause mortality and in particular with cardiopulmonary mortality. In addition, PM₁₀ was associated with an increased all-cause mortality 13% per 7 µg/m³ as

5-year average (95% CI for RR 0.99–1.30) and mortality due to cardiopulmonary causes 59% per 7 $\mu\text{g}/\text{m}^3$ as 5-year average (95% CI for RR 1.23–2.04).

Recommended exposure-response assumptions

As shown above the current literature indicates that there are differences in the harmfulness between different types of particles. A large number of studies indicate that vehicle exhaust and exhaust particles are strongly related to daily mortality, cardiovascular endpoints and excess mortality (shorter survival in cohorts). In epidemiological studies also secondary particles, and especially sulfate in the important American cohort studies, show clear associations with mortality. The often cited and used exposure-response function for the long-term effect mortality from ACS, 6% per 10 $\mu\text{g}/\text{m}^3$ of PM_{2.5} is most relevant to use for the regional background exposure, often dominated by different types of secondary particles. Since there is little information on specific results for nitrate, we do not assume any differences between sulfate and nitrate in this study.

With better precision in the exposure assessments, studies in general have found higher relative risks per concentration unit. The Los Angeles study within ACS (Jerrett et al, 2005) gives a relative risk of 17 % per 10 $\mu\text{g}/\text{m}^3$ PM_{2.5} as a realistic higher alternative to the 6 % per 10 $\mu\text{g}/\text{m}^3$ most often assumed, and the 9 % now proposed for exhaust particles by ExternE (2005). In fact, both the conducted sensitivity analyses for exposure misclassification, the Six Cities study with a finer geographical resolution and the results from Germany supports coefficients much higher than the commonly applied assumption from ACS; 6% per 10 $\mu\text{g}/\text{m}^3$. *Thus, both 6 and 17% from ACS could be used as high and low estimates for primary as well as secondary particles. For primary exhaust particles also the higher estimate may be far too low according to the relative risks obtained for NO_x (or NO₂) and the ratio between PM and NO_x in exhaust emissions* (Forsberg et al, in manuscript).

There is almost no support from the cohort studies for an effect of coarse particles (PM_{2.5-10}) on mortality, and in time-series studies less support for short-term effects of coarse than fine particles on daily mortality. In lines with these findings results from the EMFO project TRAPART indicate a non-significant short-term effect on mortality from road dust in Stockholm. *Thus road dust PM may be seen as having no effect on mortality or as the “higher estimate”, at least for a sensitivity analysis, as having the same short-term effect on mortality as PM₁₀ in general.* Studies as APHEA-2 have shown that the “short-term effect” in fact lasts over several weeks. Thus, as the higher estimate the cumulative effect of 1 % increase in all cause non-external mortality per 10 $\mu\text{g}/\text{m}^3$ could be used, based on the meta-regression in the largest European study, APHEA-2, including Stockholm (Zanobetti et al, 2002). The lower estimate for road dust (no mortality effect) is problematic when mortality is used as an indicator for all adverse health effects, since there are studies of morbidity indicating associations.

The completely different design used for studies of long-term effects of exposure on mortality within cohorts and studies of short-term fluctuations and daily number of deaths

results in different possibilities to calculate years of life lost due to the excess number of deaths. The relative risk (as % increase in mortality) per unit increase in exposure from cohort studies can simply be assumed to affect the mortality risk in every age class (or within a range) in a population with a known life table (distribution with the proportion of people dying in different ages). This way the potential years of life lost due to excess mortality from the population exposure can be given per calculated excess death or divided by the number of persons in the population as a mean value in the population. By contrast, time-series studies do not give any information how much the excess deaths are brought forward. In some studies the potential “harvesting effects” have been studied, usually without any important compensation in terms of increased mortality within 4-5 weeks after increase in exposure. Not even after the extreme increase in mortality during the “killer smog” in London 1952 was there a decrease in mortality over the next months. However, it is often assumed that people dying due to short-term exposure are very fragile and should probably have lived just a few months - years without the fatal exposure. For the short-term effects on mortality of ozone it was assumed in the CAFÉ calculations that the life shortening is 12 months per calculated excess death, but values of 2-18 months have been used at least for sensitivity analyses.

Methods for impact calculations

Health impact assessments build on epidemiological findings; exposure-response functions and population relevant rates. A typical health impact function has four components: (1) a relative risk estimate from one (or more) epidemiologic study, (2) a baseline rate for the health effect, (3) the affected number of persons and (4) the estimated “exposure” (here pollutant concentration).

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

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

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

In order to estimate how many cases that depend on elevated air pollution exposure we need to use a base-line rate. The national mean rate published (for 2002) by the register unit EpC, at The Swedish Board of Health and Welfare, was 1063 deaths per 100 000 persons. If only non-external cause of death is included, the national mean rate was approximately 1010.

There are trends in the mortality rate over time and differences between cities, regions and countries. We do however not apply different baseline rates for different regions in this project. Firstly, air pollution exposure data in this report has been aggregated in such way that it is not possible to used specific base-line rates for each country or region. Secondly, we do not want the comparison of mortality effects in different regions to be

modified by recent mortality rates. For this reason we have in our calculations used the baseline 1010 deaths per year from non-external causes per 100 000 persons for the entire studied population.

Sometimes it is assumed that there is no effect of air pollution on mortality in younger persons, which could motivate exclusion of deaths below a certain age (often 30 years) in the calculations. However, the number of deaths in age range 0-30 years is less than 40 per 100 000 in Sweden and similar in other countries, why the impact calculation results only marginally would be changed by such an exclusion. In addition, there are studies indicating as strong effect of PM10 on infant mortality as the long-term effect seen in adults in ACS (Woodruff et al, 1997), so it would be wrong to simply exclude effects on mortality in the low age range because there are no studies.

Results from impact calculations

Here are presented the estimated impacts in terms of annual number of excess deaths using the above proposed assumptions and the exposure estimates produced by Johansson & Eneroth (2007) and Bergström (2008) within TESS project team.

Table 1. Estimated annual number of excess deaths related to combustion particles, due to emissions in Greater Stockholm.

Region\Source	Road traffic	Road traffic, ldv ^a	Road traffic, hdv ^b	Sea traffic	Power plants	Residential heating
	higher estimate (lower estimate)	higher estimate (lower estimate)	higher estimate (lower estimate)	higher estimate (lower estimate)	higher estimate (lower estimate)	higher estimate ^d (lower estimate) ^e
Greater Stockholm	32,9 (11,6) ^c	24,2 (8,5) ^c	8,7 (3,1) ^c	1,5 (0,5) ^c	12,3 (4,3) ^c	142,3 (50,2) ^c
Mälardalen, except Greater Stockholm	1,3 (0,5)	0,9 (0,3)	0,4 (0,2)	0,8 (0,3)	0,4 (0,1)	5,5 (1,9)
Europe, except Mälardalen	1,0 (0,3)	0,7 (0,2)	0,3 (0,1)	0,6 (0,2)	2,2 (0,8)	4,3 (1,5)
Europe, total population exposure	35,3 (12,4)	25,8 (9,0)	9,5 (3,4)	2,9 (1,0)	14,9 (6,2)	152,1 (53,6)

^a ldv = light duty vehicles

^b hdv = heavy duty vehicles

^c from Johansson & Eneroth (2007), assumed population within the Greater Stockholm domain 1 405 600 persons.

^{d,e} with Bergströms (2008) lower alternative, built on a down scaling of the results from Johansson & Eneroth (2007), motivated by the fact that the SMED estimated emissions are on 0,20145 times the emissions used in this study, the calculated number of excess cases would be only 20% of the estimates given in this table.

Table 2. Estimated annual number of excess deaths related to nitrate (NO_3^-), due to emissions in Greater Stockholm.

Region\Source	Road traffic higher estimate (lower estimate)	Road traffic, ldv ^a higher estimate (lower estimate)	Road traffic, hdv ^b higher estimate (lower estimate)	Sea traffic higher estimate (lower estimate)	Power plants higher estimate (lower estimate)	Residential heating higher estimate (lower estimate)
Greater Stockholm	13,4 (4,7)	13,0 (4,6)	0,4 (0,1)	-0,2 (-0,1)	0 (0)	-0,5 (-0,2)
Mälardalen, except Greater Stockholm	1,9 (0,6)	1,5 (0,5)	0,4 (0,1)	0,1 (0)	0,1 (0)	0 (0)
Europe, except Mälardalen	41,6 (14,6)	23,1 (8,1)	18,4 (6,5)	7,2 (2,5)	14,0 (4,9)	3,7 (1,3)
Europe, total population exposure	56,9 (19,9)	37,6 (13,2)	19,3 (6,7)	7,1 (2,4)	14,1 (4,9)	3,2 (1,1)

^a ldv = light duty vehicles

^b hdv = heavy duty vehicles

Table 3. Estimated annual number of excess deaths related to particulate sulphate (SO_4^{2-}), due to emissions in Greater Stockholm.

Region\Source	Road traffic higher estimate (lower estimate)	Road traffic, ldv ^a higher estimate (lower estimate)	Road traffic, hdv ^b higher estimate (lower estimate)	Sea traffic higher estimate (lower estimate)	Power plants higher estimate (lower estimate)	Residential heating higher estimate (lower estimate)
Greater Stockholm	0,4 (0,2)	0,5 (0,2)	-0,1 (0)	1,8 (0,6)	1,5 (0,5)	4,1 (1,5)
Mälardalen, except Greater Stockholm	0 (0)	0 (0)	-0 (0)	0,2 (0,1)	0,7 (0,2)	0,3 (0,1)
Europe, except Mälardalen	3,3 (1,1)	4,1 (1,4)	-0,8 (-0,3)	1,3 (0,4)	11,4 (4,0)	2,0 (0,7)
Europe, total population exposure	3,7 (1,3)	4,6 (1,6)	-0,9 (-0,3)	3,2 (1,1)	13,6 (4,7)	6,4 (2,3)

^a ldv = light duty vehicles

^b hdv = heavy duty vehicles

Table 4. Estimated annual number of excess deaths related to wear particles due to emissions in Greater Stockholm.

Region\Source	Road wear higher estimate	Road wear (lower estimate)
Greater Stockholm	22,1 ^a	(0)
Mälardalen, except Greater Stockholm	2,0	(0)
Europe, except Mälardalen	1,3	(0)
Europe, total population exposure	25,4	(0)

^a from Johansson & Eneroth (2007) , assumed population within the Greater Stockholm domain 1 405 600 persons.

Instead of only presenting the calculated number of excess deaths, the potential years of life lost (PYLL or YoLL) due to excess mortality may be presented from calculation with life tables presenting death and survival with and without the studied exposure. We have in our YoLL calculations used the WHO software AirQ version 2.2 and Greater Stockholm 2002 life tables with exclusion of external causes of death.

Assuming the relative increase in mortality to be independent of age, we find that each estimated excess death corresponds to 11.2 potential years of life lost with the 2002 mortality rates. This means that the 32.9 excess deaths per year in Greater Stockholm due to the road traffic combustion (exhaust) particles correspond to 368.5 YOLL. The wear particle estimate of 22.1 excess deaths due to short-term exposure corresponds to as many YOLL when deaths are assumed to be brought forward 1 year.

Discussion

This report is focused on the epidemiological basis for selection of exposure-response functions for traffic related particles and mortality. A second aim was to use the exposure calculations by SLB and SMHI presented in TESS to make impact calculations using the selected exposure-response assumptions for particles of three types; exhaust particles, road dust particles and secondary particles. Emissions from domestic heating are estimated with less confidence than the exhaust particle emission from vehicles, and as reported by Bergström (2008) could the estimated emissions be 5 times too high.

There is a growing recognition among the research community that there also from a health point of view are very different types of particles and that their effect on mortality and morbidity likely differ. Still the current practice is to treat fine particles (which are considered to be most detrimental to health) as equally harmful irrespective of origin. Hence, there is often as in the European CAFE Program only one assumed exposure-function used for assessing the health impact of fine particles (PM_{2.5}); 6 % per 10 µg/m³.

The current version of ExternE (2005) has new assumptions about the toxicity of the different PM types that reflect the indicated high toxicity of combustion particles and especially from engines. ExternE now treats nitrates as equivalent to half the toxicity of PM₁₀ (~2 % per 10 µg/m³), sulphates as equivalent to PM₁₀ and primary particles from vehicles as equivalent to 1.5 times the toxicity of PM_{2.5}. The long-term effect on mortality of PM_{2.5} has been assumed to be 6 % for a 10 µg/m³ increment of average PM_{2.5}.

The studied literature indicates indeed that there are differences in the harmfulness between different types of particles. Several studies suggest that vehicle exhaust and exhaust particles are strongly related to mortality. Also the secondary particles, and especially sulfate, are important for mortality in the American cohort studies. However, when exposure estimates are from models, it is logic to use the relative risk for PM_{2.5} for all secondary components that build up the PM_{2.5} mass.

With better precision in the exposure assessments, studies in general find higher relative risks per concentration unit. A study within ACS gives a relative risk of 17 % per 10 µg/m³ PM_{2.5}. This is especially for exhaust particles a realistic alternative to the 6 % per 10 µg/m³ most often assumed.

There is from the cohort studies very little support for an effect of coarse particles (PM_{2.5-10}) on mortality, and in time-series studies less support for short-term effects of coarse than fine particles on daily mortality, and less support for an effect of crustal particles. Thus road dust PM may be assumed to have no effect on mortality or for a sensitivity analysis as having the same short-term effect on mortality as PM₁₀ in general. A cumulative effect of 1 % increase in all cause non-external mortality per 10 µg/m³, based on the meta-regression in the largest European study, APHEA-2, is proposed.

The relative increase in mortality per unit increase in exposure from cohort studies can be assumed to effect in the same way mortality in every age class in a population with a known life table. The potential years of life lost (PYLL) due to excess mortality can than be calculated per excess death or divided by the number of persons in the population to have a mean value for the whole population. We found for Stockholm that every excess death due to long-term effects of particles corresponds to 11.2 PYLL. Time-series studies do not give any information how much the excess deaths are brought forward. For the short-term effects on mortality of wear particles (road dust) the life shortening can be assumed to be 12 months per calculated excess death as in the CAFE calculations.

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