SIXTH FRAMEWORK PROGRAMME

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NEEDS

New Energy Externalities Developments for Sustainability

INTEGRATED PROJECT

Priority 6.1: Sustainable Energy Systems and, more specifically,
Sub-priority 6.1.3.2.5: Socio-economic tools and concepts for energy strategy.

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"A set of concentration-response functions"

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Abstract

This report provides the scientific basis for a set of concentration-response functions (CRFs) to quantify health impacts from ambient air pollution in Europe (i.e. in the EU-27). Starting from a comprehensive review of quantification issues, as a joint exercise of the cost-benefit analysis (CBA) in the preparation of the thematic strategy 'clean air for Europe' (CAFE) and of NEEDS, this report describes work carried out on a range of issues, especially aspects of indoor air pollution. Where necessary we have repeated and extended our views on issues that keep being raised, like the transferability of epidemiological results from the US to a European HIA.

Quantification of the classical air pollutant mixtures is restricted to CRFs expressing the effects of PM and of ozone. This means that effects from NO_2, SO_2 and CO are not considered independent from PM or ozone, or that evidence of the associations between these gases and some health effects is indecisive.

Main outcomes of this work package are:

1. A set of CRF;
2. A discussion of the component specific toxicity of PM;
3. An overview of transferability issues, exposure issues (including indoor exposure) and uncertainty;

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1 SUMMARY

The purpose of the present report is to provide the scientific basis for a set of concentration-response functions (CRFs) to quantify health impacts from ambient air pollution in Europe (i.e. in the EU-27). The set of CRFs and associated background rates are needed to update previous work in the field of external cost assessments. The set of CRFs is the basic outcome of this work package in stream 1B of the IP NEEDS. The NEEDS project extends work previously carried out for the European Commission under the various projects of the ExternE programme. Central in the ExternE methodology to estimate quantitatively the effects of energy systems, including transport and electricity generation from fossil fuels, are issues of air pollution and health. Work on the present project can be seen as consisting of two parts. First, there was a comprehensive review of quantification issues, as a joint exercise of the cost-benefit analysis (CBA) in the preparation of the thematic strategy 'clean air for Europe' (CAFÉ) and of NEEDS. Secondly, within NEEDS we carried out further work on a range of issues. This present report includes a short account of the joint work between CAFE CBA and NEEDS (chapter 2 and 3); and follows this with chapters of further work since the CAFE-NEEDS literature review. Work since the CAFE-NEEDS HIA literature review has focussed on three broad sorts of issues:

1. We have re-visited some of the framework issues of CAFE CBA and the CRFs on particulate matter ((PM) and ozone used there. Our recommendation with respect to these methodological issues are:
    - Quantification of the classical air pollutant mixtures is restricted to CRFs expressing the effects of PM and of ozone. This means that effects from NO2, SO_2 and CO are not considered independent from PM or ozone, or that evidence of the associations between these gases and some health effects is indecisive.
    - Quantification of PM is based on anthropogenic PM, without threshold. In a modelling approach, like the ExternE methodology, this is also practical, because data on natural emissions of PM is mostly lacking.
    - The WHO framework decision to apply the same coefficient to all PM in the $PM_{2.5}$ (or PM_{10}) size range, regardless of source, composition or other characteristics that might be relevant to toxicity, has been carefully reconsidered. Components of the $PM_{2.5}$ ambient urban mixture are quantified (per $\mu g/m^3$) the same as the overall mixture itself; i.e. that the known or suspected differences in toxicity between different components of $PM_{2.5}$ be ignored for quantification purposes. This methodological issue has an effect on the

quantification of nitrates and sulphates, and on especially on the quantification of chronic mortality effects of $PM_{2.5}$. This is discussed separately (in chapter 6)

- Quantification of ozone is also without threshold, but that ozone effects be estimated only above 35 $\mu g/m^{-3}$ (8-hr daily max), on days when this level is exceeded.

2. Secondly, we have considered some other pollutants, notably gases other than ozone, and metals. (paragraph 4.1 and 4.2)
3. Thirdly, we have examined some issues not considered by CAFE, especially aspects of indoor air pollution (chapter 9). Where necessary we have repeated and extended our views on issues that keep being raised, like the transferability of epidemiological results from the US to a European HIA (chapter 7).

We have been cautious in changing the CAFE CBA recommendations in the light of these updates. This is because we recognise that CAFE CBA now has a particular status within air pollution HIA in Europe. It is consistent with recommendations of WHO and within the UNECE Convention on the Long-Range Transport of Air Pollutants (CLRTAP). Its methodology has been open to consultation and comment, including peer-review by a top US team. And it has been used by DG Environment on behalf of the Commission to develop proposals for the control of ambient $PM_{2.5}$ within the EU. We recognise also, however, that the evidence on air pollution and health, and the understanding of it, continue to evolve and that there is an advantage in also maintaining consistency with earlier ExternE-related projects, but of course, where CAFE CBA and ExternE are different, it is not possible to be consistent with both at the same time. The most important change with respect to previous ExternE methodology reports is the application of the same coefficient to all PM in the $PM_{2.5}$ (or PM_{10}) size range.

Main outcomes of this work package are:

1. A set of CRF;
2. A discussion of the component specific toxicity of PM;
3. An overview of transferability issues, exposure issues and uncertainty;

Finally we recommend that this methodology be used within the context of its intended use: to assess health impacts of air pollutants in Europe. Extension to other regions must be seen as a first order approximation of the impact of air pollution, and within the uncertainty ranges of the methodology this approximation is plausible. We also regard further research essential in improving these results, especially:

1. a European based assessment of long-term effects of air pollution;

2. a harmonised European approach for the definition and registration of disease categories, including mild symptoms, their incidence and prevalence;

1.1 A set of CRF

A detailed literature review, and the associated recommendations for CRFs and for background rates, that underpin the quantification of health in CAFE CBA is addressed in Hurley et al. (2005a). The report also includes discussion of a wide range of relevant methodological issues. A summary of the literature review is given in annex 1. CRFs for PM and ozone are summarized in Table 1.1. Additional background data is given in Table 1.2. For carcinogenic pollutants, lead and mercury a summary of the exposure response functions, currently used in NEEDS, and other ExternE related projects (like Espreme), is given in Table 1.3 and Table 1.5. Finally we analysed whether there was a need to update risk coefficients for radioactive pollutants. Although a new ICRP recommendation is in its final stage, it is advised to use the ICRP 60 values Table 1.4. New proposed risk factors will be revised downwards, but for practical purposes and within the uncertainties of the risk assessment of low-dose ionizing radiation effects, the ICRP 60 values are still valid.

We prefer a mortality impact assessment of air pollution to be based on a life expectancy approach, and not on the number of attributable cases. We recognize that this is not always possible due to the lack of quantitative information for acute mortality effects. The contribution of acute mortality to the total life expectancy loss of chronic exposure is equal to the relative risk increase times the time constant of the repair processes that are significant immediately after a pollution peak. But the exact time constants involved in the repair processes after a pollution peak are still uncertain. However the life expectancy loss due to acute mortality is much smaller than that of mortality due to chronic exposure.

1.2 Component specific toxicity of PM

A theoretical model that is practicable and able to address different components of PM or the mixture of ambient air pollution is described, but the range of application of the model at this time is limited by two important factors. One of these are the limitations in the modelling of particulate matter dispersion from emissions of energy sources (or any other source of air pollution). The second is the view of many individual air pollution researchers, and of established working groups e.g. of the World Health Organisation, that although the toxicity of different kinds of particles may well vary, per unit mass, it is not possible, based on current evidence, to quantify reliably any

differences in toxicity. Consequently, in its quantitative estimates, CAFE CBA distinguished between PM_{10} and $PM_{2.5}$, but did not otherwise differentiate in toxicity between different components of PM. Nevertheless, we consider that the model described here remains important as a framework for quantifying differential effects of different kinds of particles. It is useful to evaluate alternative assumptions about the components and mixture that is being modelled. It can also be used to demonstrate the importance of including other fractions not modelled, like metals, or organic components. The extensive formulation of the model is given in chapter 6.3. For PM_{10} (and similarly for $PM_{2.5}$) the formula can be simplified to

$$\Delta I = s_{PM10}\ \Delta c_{PM10amb}\ (\%_{PMpower}\ f_{PMpower} + \%_{PMtrans}\ f_{PMtrans} + \%_{sulf}\ f_{sulf} + \%_{nitr}\ f_{nitr,P} + \%_{HNO3}\ f_{HNO3,P} + \%_{PMother}\ f_{PMother})$$

Where s_i indicates the slope of the CRF, f_i a modifying factor and % with subscripts the fraction of $\Delta c_{PM10amb}$ that is of the type indicated by the subscript ("other" refers to other PM constituents such as organic matter, metals and soil particles). f_i factors can be based on evidence, which is limited, or it can be based on expert opinion. Unfortunately at the present time not enough experts are willing to commit themselves to making such a choice, or, more precisely, to making a choice other than the one implicit in the CAFÉ methodology. Once we have established the CRF slopes s_i and the modifying f_i factors, we can evaluate the impact of these choices.

Our recommendation in NEEDS is in line with the CAFÉ approach. In core analyses we will use particle mass within size ranges, i.e. $PM_{2.5}$ or PM_{10}, and we will not differentiate further than that, quantitatively (f_i equal to 1). In sensitivity analyses the impact of this choice of weighting factors can be explored, through application of different weighting factors to different constituents of $PM_{2.5}$ and/or PM_{10}. Especially, for sulphate and nitrate particles we will treat these according to the general principle, above; i.e. their effects will be quantified insofar as they contribute to $PM_{2.5}$ and/or PM_{10} (in $\mu g/m^3$). Organic constituents are treated similarly, but it is possible that limitations on the air quality modelling implies some under-estimation of health effects. Overall this is a change from the most recent ExternE (2005) position. The position above is the position of CAFE CBA, WHO, US EPA, COMEAP…. We see a benefit in consistency with these other expert groups, unless we have a (very) strong basis for an alternative. As noted (e.g in annex, and in the ACS study (Pope et al., 2002)), there is some basis for an alternative but no critical mass of support for it – hence no differentiation in the core analyses. In addition, we think that a position which simultaneously played down the toxicity of sulphates and nitrates and did not quantify direct effects of SO_2 and/or of NO_2 would imply the unlikely situation that SO_2 and/or NO_2 are to be ignored. The

proposed differential quantification of ExternE (2005) might be somewhat extreme; as a sensitivity analysis we propose a possible intermediate position, to treat:

- Primary particles at 1.3 times the toxicity of the $PM_{2.5}$ mixture;
- Secondary particles are at 0.7 times the toxicity of $PM_{2.5}$

We do not support a quantification that regards either nitrates or sulphates as inert, i.e. with zero toxicity. Other sensitivity analyses are possible but any use of a differential quantification needs to emphasise that this is for illustrative / exploratory purposes only; and that there is no consensus about the exact values chosen. Because there is such a strong swell of opinion against differential quantification, we propose that we do not focus strongly on the quantified results of the sensitivity analyses, but rather on whether the sensitivity analyses lead to the same policy conclusions as the core analyses.

1.3 An overview of transferability issues, exposure issues and uncertainty

When applying epidemiological concentration response functions (CRF) in a different context, the transferability of the original study results to the new situation is a matter that needs some attention. Inevitably there are differences between locations and populations. Of primary interest here, however, is to make a judgement – based on evidence – about whether these differences really matter. The concern that is most often raised is that by transferring results from one location to another, the effects of air pollution in the target population may be over-estimated. It is important to consider that transferring CRFs (or background rates) between locations may also lead to under-estimation of effects – though an under-estimation is less severe than if CRFs had not been transferred at all, leaving the particular pollutant-endpoint combination unquantified and so in effect estimated as zero. For time series results the simple way to treat this issue of transferability is to look at the similarities between results of time series studies on daily mortality and hospitalization world-wide, given the completely different background rates and population characteristics. The percent change in daily mortality per unit exposure remains remarkably invariant to changes in population, location and population mixtures. The results of time series studies on acute mortality worldwide compare sufficiently close in view of the uncertainties with the value of 0.6% per 10 $\mu g/m^3$ PM_{10} that we have chosen. This observation of similarities between risk estimates around the world is a good argument in favour of the validity of using ambient PM_{10} concentrations as a measure of exposure tot outdoor air pollution, and of transferring results of epidemiological studies of acute effects of air pollution to target areas, when no original information for this area is

available, and irrespective of where ambient monitors are located or of how measurements are performed.

It is obvious that in a health impact assessment care has to be given to the way exposure is measured or modelled. In general, exposure is approximated by the concentration of a certain pollutant. Transferring the CRF (expressed as the % change per unit of exposure) from the US to a European situation, and applying a measured or modelled concentration can induce errors, but is strictly speaking no transferability issue. In the ACS study PM2.5 was measured in very different metropolitan areas across the US, with varying PM2.5 concentrations and composition. It is therefore unlikely that a different composition of PM2.5 in Europe will affect the CRF derived from the ACS study. But it remains important to have as many components as possible included in the modelled PM2.5 concentrations. The same considerations apply for air pollution mixture as a whole. In general we consider the modelling as a source of additional uncertainty, but not as a factor that limits the transferability of CRF from the US. In this respect it is important to note that the incomplete modelling of organic matter (OM) as fraction of $PM_{2.5}$ may result in an under-estimation of the benefits of an emission reduction policy. Similarly, if the toxicity of OM is comparable to that of primary combustion-derived particles, then an impact assessment using modelled concentrations without the OM fraction will underestimate the effect of $PM_{2.5}$ on a mass concentration basis. Note that this under-estimation is not *per se* an issue of transferability. Rather, it is a consequence of a mismatch between modelled PM concentrations and the measured values used in the underlying epidemiological studies.

Another concern in the application of CRF from epidemiological studies in the US in Europe, is the question whether the indoor-outdoor relationship for $PM_{2.5}$ and PM_{10} is comparable in the US and in the EU? Substantial research has been carried out on personal exposures to particulate matter and the contribution of particles from outdoor sources to total personal exposure, an area of investigation corresponding to Topic 1 of the US National Research Council's Committee. Studies have been carried out in the United States and in Europe that have involved personal monitoring of various population groups of interest in order to characterize the contribution of outdoor particles to personal exposure, and particularly to variation in personal exposure over time. In general our conclusion is that ambient concentrations of $PM_{2.5}$ can serve as the adequate index of exposure in epidemiological time-series studies, intra-personal differences being explained in the original exposure studies through the indoor penetration of $PM_{2.5}$ and the indoor sources of $PM_{2.5}$.

Detailed analyses of the ACS study have shown that the relative risks of mortality from long-term exposure to ambient PM2.5 are robust to a range of population characteristics. Educational attainment is the one population characteristic which has been clearly shown, in the ACS study, to

modify the relative risks of PM and mortality, with higher risks among people with lower educational attainment. It is understood that this may well not be an effect of education per se, but rather that educational status is here acting as a surrogate measure for socio-economic factors more generally. The ACS study is adjusted for a multitude of (44) potential individual confounders, which in practice have little or no effect on the estimated coefficient linking longer-term exposure to PM and mortality. This gives reassurance in transferring the ACS coefficient for use in Europe. It may further be a reassurance that overall mortality rates and most important causes of death are quite similar, in the US and Europe. The socio-economic status of the ACS study population is higher than average in the US, and so use of the ACS study in Europe might imply some under-estimation of mortality effects associated with socio-economic differences implied by differences in educational status.

1.4 A case study on indoor exposure from wood combustion

The fact that people spent more than 80% of their time indoors keeps raising questions about the relevancy of studying outdoor air pollution only. Does indoor exposure confound with outdoor exposure? How does indoor exposure correlate with exposure to ambient concentrations or with personal exposure? This issue has been highlighted in chapter 7, on the use of ambient concentrations as a surrogate for exposure. Apart from this there are numerous sources of indoor air pollution, that interact or act independently from the outdoor air pollution. In the context of NEEDS a case study is developed to illustrate the relative importance of energy-related sources of indoor air pollution. Wood stove[1] combustion is selected as a typical case study, for which the exposure to particles indoors is elaborated in terms of exposure. It is clear that indoor air pollution is far more complex, involving many sources, among which the most important are cigarettes, consumer products and building materials, and involving a wide spectrum of pollutants. Pollutants like VOCs, formaldehydes and other aldehydes, NO2, CO, benzene, PAHs, toluene, xylenes are among the most important. A limitation to particles from woodstoves is thus automatically an underestimation of the full indoor exposure to air pollution.

Health impacts due to combustion of wood have been reported in literature, but are not coherent or comparable across studies to derive concentration response functions, exposure response functions or source response functions. This inhibits a quantification of impacts from these sources. Apart from this obvious lack of scientific data there is a question whether these impact are external, i.e.

[1] the term 'woodstove' here refers to both indoor (closed) window woodstoves and indoor open fireplaces

generating a cost to society that is not borne by those who enjoy the benefits of that good or service. It is defendable to assume that the impact on children's health due to indoor cooking or heating is an external cost, since they do not have part in the decision process. Moreover it is in general even for adults a decision that is not based on complete information, nor on a rational balance of costs and benefits, let alone that there is a possibility for consumers to choose an better (healthier) alternative.

Using a simple steady state model to describe PM concentrations indoor, it is possible to develop scenarios. Using this model and taking into account (i) stove PM concentrations modelled; (ii) average time activity patterns of adults and (iii) some assumption on average outdoor concentration of PM enables an assessment of the relative importance of this indoor source to the overall, population average exposure. In annex 5 the time window for woodstove users in the EU is discussed. From the EU energy report (2006), we estimate that on average an equivalent of 14 % inhabitants of the former EU-15 is exposed to indoor wood smoke during 14 % of their time. 86% of people in this part of Europe is hence not exposed to wood stove PM, whereas 100% of people is exposed to ambient PM.

To answer the question whether wood stove use might interfere with the overall exposure to PM_{10}, a scenario analysis was made, taking into account an ambient average PM_{10} concentration of 40 $\mu g/m^3$, for a given emission strength and in a particular case of a 125 m^3 inhabited volume. For example, if on average (across all stoves in Europe) the wood stove PM_{10} concentration indoor would be 50 $\mu g/m^3$, this would then contribute 4% to the total PM_{10} exposure, if ambient average concentrations would be 40 $\mu g/m^3$ and given the stove use pattern and time use. At the individual level, the PM woodstove exposure can amount to >10 % of the personal PM exposure. At average population level, the relative toxicity of wood stove PM must be very high to be of any significant concern when assessing population based health impacts from ambient air pollution. There is not sufficient evidence available on the differential toxicity of wood stove PM versus ambient (fossil fuel) PM. There is no good reason to believe that wood stove PM would be more toxic, given the fact wood contains less sulphur and less metals compared to fossil fuels. Note that exposure to some individuals in Europe might still be very high and of concern. Indeed, the negative health impact of woodstove use in a number of studies performed at present times in the EU confirms this. But from this population averaged contribution of PM to personal exposure example, we can safely assume that the use of ambient PM concentrations –either modelled or measured- are valid in an overall health impact assessment of air pollution, because it dominates the total exposure. Of course in cases where multiple sources are involved, and especially for ETS, the indoor contribution to total exposure increases.

Table 1.1: Overview of the concentration response functions for PM and ozone. Functions for sensitivity analysis in italics.

Endpoint	Pollutant (1)	Age group	Risk group fraction	CRF (95% CI)	Units
CORE CRF					
Chronic mortality					
Life expectancy reduction	PM2.5	30+	1	651 (127; 1194)	YOLL per 10 µg/m³ per 100 000 people
Infant mortality					
Increased mortality risk	PM10	0-1	0.4%	4% (2%; 7%) 18	attributable cases per 10 µg/m³ YOLL per 10 µg/m³ per 100,000 people (all ages)
Acute mortality					
Increased mortality risk	O_3/SOMO35	all	0.99%	0.30% (0.1%; 0.43%) 0.75	attributable cases per 10 µg/m³ YOLL per case
Morbidity					
New cases of chronic bronchitis	PM10	27+	0.376%	26.5 (-1.9; 54.1)	per year, per 10 µg/m³, per 100,000 adults aged 27+
Respiratory hospital admissions	PM10	all	1	7.03 (3.83;10.3)	per year, per 10 µg/m³, per 100,000 people
Cardiac hospital admissions	PM10	all	1	4.34 (2.17; 6.51)	per year, per 10 µg/m³, per 100,000 people
Medication use / bronchodilator use	PM10	5-14	PEACE criteria (15% N&E-EU) (25% W-EU) Medication use 10%	180 (-690; 1060)	per year, per 10 µg/m³ per 1000 children meeting the PEACE criteria
	PM10	20+	Asthmatics 4.5% Daily medication use probability 50%	912 (-912; 2774)	per year, per 10 µg/m³ per 1000 adults 20+
Lower respiratory symptoms	PM10	adults	symptomatic adults (30%)	1.3 (0.15; 2.43)	symptom days per year, per 10 µg/m³ per adult with chronic respiratory symptoms
	PM10	5-14		1.86 (0.92; 2.77)	symptom days per year, per 10 µg/m³ per child 5-14
Restricted activity days (RADs)	PM2.5	15-64	1	902 (792;1013)	per year, per 10 µg/m³ per 1000 adults 15-64
Work loss days (WLD)	PM2.5	15-64	1	207 (176; 208)	per year, per 10 µg/m³ per 1000 adults 15-64
Minor restricted activity days (MRAD)	PM2.5	18-64	1	577 (468; 686)	per year, per 10 µg/m³ per 1000 adults 18-64
Respiratory hospital admissions	O_3/SOMO35	65+	1	12.5 (-5; 30)	per year, per 10 µg/m³ per 100 000 people 65+
MRAD	O_3/SOMO35	18-64	1	115 (44; 186)	per year, per 10 µg/m³ per 1000 adults 18-64
Medication use / bronchodilator use	O_3/SOMO35	20+	Asthmatics 4.5%	730 (-225; 1570)	per year, per 10 µg/m³ per 1000 asthmatic adults 20+
LRS excluding cough	O_3/SOMO35	5-14	1	0.16 (-0.43; 0.81)	days of LRS per year, per 10 µg/m³ per child 5-14
Cough days	O3/SOMO35	5-14	1	0.93 (-0.19; 2.22)	cough days per days, per 10 µg/m³ per child 5-14
SENSITIVITY FUNCTIONS					

Endpoint	Pollutant (1)	Age group	Risk group fraction	CRF (95% CI)	Units
New cases of chronic bronchitis	*PM2.5*	*27+*	*0.376%*	*53.3 (-1.7; 113.4)*	*per year, per 10 µg/m³, per 100,000 adults aged 27+*
Consultations with primary care physicians					
Asthma	*PM10*	*0-14*	*1*	*1.18 (0; 2.45)*	*per year, per 10 µg/m³ per 1000 children 0-14*
	PM10	*15-64*	*1*	*0.51 (0.2; 0.82)*	*per year, per 10 µg/m³ per 1000 adults 15-64*
	PM10	*65+*	*1*	*0.95 (0.32;1.69)*	*per year, per 10 µg/m³ per 1000 adults 65+*
Upper respiratory diseases	*PM10*	*0-14*	*1*	*4 (-0.6 ; 8)*	*per year, per 10 µg/m³ per 1000 children 0-14*
	PM10	*15-64*	*1*	*3.2 (1.6-5)*	*per year, per 10 µg/m³ per 1000 adults 15-64*
	PM10	*65+*	*1*	*4.7 (2.4-7.1)*	*per year, per 10 µg/m³ per 1000 adults 65+*
Acute respiratory symptoms	*PM10*	*all*	*1*	*4650 (210; 9090)*	*symptom days per year, per 10 µg/m³ per 1000 people*
Consultations with primary care physicians for allergic rhinitis	*O_3 /SOMO35*	*0-14*	*1*	*3.03 (1.89; 429)*	*per year, per 10 µg/m³ per 1000 children 0-14*
	O_3 /SOMO35	*15-64*	*1*	*1.6 (1.22; 2.03)*	*per year, per 10 µg/m³ per 1000 adults 15-64*
Medication use / bronchodilator use Northern and Eastern Europe	*O_3 /SOMO35*	*5-14*	*1*	*124 (18; 227)*	*per year, per 10 µg/m³ per 1000 children 5-14*
Medication use / bronchodilator use Western Europe	*O_3 /SOMO35*	*5-14*	*1*	*310 (44; 569)*	*per year, per 10 µg/m³ per 1000 children 5-14*

(1) SOMO35 is the 8hr-averaged maximum O3 concentration increment above 35 ppb (70 µg/m³), aggregated over all days of one year.

Since Work Loss Days (WLD), Minor Restricted Activity Days (MRAD and days in hospital because of cardiac hospital admissions (CHA) an Respiratory Hospital Admissions (RHA) are also RADs the net RADs are calculated as:

netRAD (per µg/m³ $PM_{2.5}$) = RAD – WLD – WRAD – (RHA and CHA due to PM_{10}) x 10 days, i.e.:

netRAD (per µg/m³ $PM_{2.5}$) = (0.0902-0.0207) x Adults_15_to_64 – 0.0577 x Adults_18_to_64 – (7.03 + 4.34) x 10^{-6} x 5/3 x Total_pop x 10 days per year , per µg/m³ PM2.5

Table 1.2: summary of the age and risk group categories used.

Age group	**[%]**
Baseline mortality	0.99
Infants 0-1 year	0.9
Adults 15 and above	83
Adults 15 to 64 years	67.2
Adults 18 to 64 years	64
Adults 20 and above	79.8
Adults 27 and above	70
Adults 30 and above	70
Children 0 to 14 years	17
Children 5 to 14 years	11.2
Elderly, i.e. 65 and above	15.8
Risk Group	**[%]**
Children_PEACE_criteria_EUaverage	20
Symptomatic adults	30
Asthmatics	4.5

Table 1.3: Exposure response functions recommended for carcinogens. Slope factor = probability of cancer per daily unit intake during 70 years. Unit risk factor = probability of cancer from lifetime exposure to a concentration of 1 μg/m^3. Risk factors for kung cancer based on www.epa.gov/iris.

	Air unit risk per μg/m^3	**Drinking water unit risk per μg/L**	**Oral slope factor per mg/kg$_{body}$/day**
PCB [a, b]	1×10^{-4}	1×10^{-5}	4×10^{-2}
Dioxins TEQ [b]			1×10^{6}
Benzene	4×10^{-6}		
Benzo[a]pyrene	1×10^{-7}		
As (inorganic)	4.3×10^{-3} [d]	5×10^{-5} [e]	1.5 [e]
Cd	1.8×10^{-3} [d]		
CrVI	1.2×10^{-2} [d]		
Ni (refinery dust)	2.4×10^{-4} [d]		

[a] haematologic cancer, cancer of the liver, gall bladder, gastrointestinal tract and biliary tract, and skin cancer.
[b] the numbers for PCB and dioxins are probably an overestimate, and Searle (2005) suggests that they may be 5 times smaller; furthermore, mortality rate for such cancers may be around 50%.
[c] this is probably an overestimate, to be used for sensitivity analyses only.
[d] lung cancer, mortality rate about 90%.
[e] lung cancer, internal organ cancers and skin cancer.

Table 1.4: Risk factors (in % per man.Sv) for cancer and hereditary effects (ICRP draft recommendations of 12/01/2007).

	Cancer		**Hereditary effects**		**Total**	
	New recom-mendations	**ICRP 60**	**New recom-mendations**	**ICRP 60**	**New recom-mendations**	**ICRP 60**
Whole population	5.5	6.0	0.2	1.3	6.0	7.3
Adults	4.1	4.8	0.1	0.8	4.0	5.6

Table 1.5: Exposure response functions recommended in the Espreme project http://espreme.ier.uni-stuttgart.de/

Effect	Pollutant	Expoure route	Population group	Exposure time [years]	Absolute risk	Exposure unit (µg/m³) or intake rate (µg/L)
Skin cancer	As	Ingestion	All	70	2×10^{-5}	µg/day
Skin cancer	As	Inhalation	All	70	4×10^{-4}	µg/m3
Lung cancer	As	Inhalation	All	70	1.5×10^{-3}	µg/m3
Bladder cancer	As	Ingestion	All	70	1×10^{-4}	µg/day
Bladder cancer	As	Inhalation	All	70	4×10^{-6}	µg/m3
Cardiovascular mortality	As	Ingestion	All	35	3×10^{-3}	µg/day
Cardiovascular mortality	As	Inhalation	All	35	2×10^{-2}	µg/m3
Still birth	As	Ingestion	All	1	1.044×10^{-4}	µg/day
Still birth	As	Inhalation	All	1	2.088×10^{-3}	µg/m3
Osteoporosis	Cd	Ingestion	All	35	8×10^{-3}	µg/day
Osteoporosis	Cd	Inhalation	All	35	1.6×10^{-1}	µg/m3
Renal dysfunction	Cd	Ingestion	All	35	4×10^{-4}	µg/day
Renal dysfunction	Cd	Inhalation	All	35	8×10^{-3}	µg/m3
Lung cancer	CrVI	Inhalation	All	70	4×10^{-3}	µg/m3
Children's IQ	Pb	Food	Minors	5	3.2×10^{-1}	µg/day
Children's IQ	Pb	Drinking water	Minors	5	4.8×10^{-1}	µg/L
Children's IQ	Pb	Inhalation	Minors	5	1×10^{-1}	µg/m3
Anaemia	Pb	Food	All	1	4.8×10^{-5}	µg/day
Anaemia	Pb	Drinking water	All	1	2×10^{-4}	µg/L
Anaemia	Pb	Inhalation	All	1	1.3×10^{-3}	µg/m3
Children's IQ	Hg	Ingestion	Minors	1	1.49×10^{-1}	µg/day
CHS effects in adults - ataxia	Hg	Ingestion	All	35	1.3×10^{-3}	µg/day
CHS effects in adults - ataxia	Hg	Inhalation	All	35	1.4×10^{-4}	µg/m3
Renal dysfunction - preclinical effects	Hg	Inhalation	All	35	2×10^{-3}	µg/m3
Lung cancer	Ni	Inhalation	All	70	3.8×10^{-4}	µg/m3
Lung cancer	Cd	Inhalation	All	70	1.8×10^{-3}	µg/m3

2 INTRODUCTION – PURPOSE, BACKGROUND, STRATEGY FOR THIS REVIEW

2.1 Purpose

The purpose of the present report is to provide the scientific basis for a set of concentration-response functions (CRFs) to be used, in conjunction with other data, for quantifying health impacts from ambient air pollution in Europe (i.e. in the EU-27). The issues are important practically, because in previous work it has been shown that external costs of air pollution were dominated by public health impacts (Rowe et al. 1995, Abt 2004, ExternE 2005). The set of CRFs and associated background rates are needed to update previous work in the field of external cost assessments. The set of CRFs is the basic outcome of this work package in stream 1B of the IP NEEDS.

2.2 Background – literature reviews leading to CRFs for ExternE in the 1990s

The NEEDS project draws heavily on, and in turn extends, work previously carried out for the Commission under the various projects on the ExternE programme. This applies in particular to issues of air pollution and health, which are central to estimating quantitatively the effects of energy systems, including transport and electricity generation from fossil fuels, and where the ExternE methodology has been widely accepted and used, including by the Commission in its development of policies for air pollution control.

Within ExternE, the first comprehensive review of the literature on air pollution and health, with a view to quantifying health effects of pollution, was carried out in 1993-95. This review drew heavily on work by Bart Ostro for a parallel project for the US Department of Energy (Rowe et al. 1995), though with use, where practicable, of CRFs from studies in Europe. It focussed on the 'classical' air pollutants – particles (expressed as PM10) and the major combustion-related gases (ozone, SO2, NO2 and CO). Both gases and particles emitted directly and those derived later, in the course of atmospheric reactions were included. The review consisted of:

i. An assessment of causality of various pollutants;
ii. An assessment of which health endpoints were associated with the pollutants that were considered to be causal – PM and ozone;
iii. Recommendations on key framework issues such as whether the evidence supported a population-level threshold for health effects;

iv. Recommendations for concentration-response functions (CRF; i.e. % change in health effect per µg/m^3 pollutant) for quantifying the many specific pathways to be included; with these recommendations varying as necessary by sub-population (e.g. children, elderly, people with asthma);

v. Recommendations for estimating, for the various health endpoints, the background rates of morbidity in the target population, consisting of the 15 countries which then constituted the European Union; and

vi. Recommendations for aggregating impacts across pollutants and health endpoints.

The review and recommendations were published in the first ExternE Methodology Report (1995) and were the basis for ExternE work for a number of years. In practice the single most important CRF was that linking daily mortality with daily variations in ambient particulate matter, measured as PM10. This was important in practice partly because of the convention, in widespread use at that time, of attributing the full monetary value of a statistical life (VSL) to the associated deaths, even though there was an understanding that those most at risk were people with established cardio-respiratory disease, and so with a shorter life expectancy relative to the population generally at that age. The 1995 recommendations took account of then recent cohort studies, by Dockery et al (1993) and Pope et al (1995), showing associations between mortality risks and annual average concentrations of PM2.5 in US cities and metropolitan areas. It proposed a simple method of quantification, leading to estimates of attributable deaths; but suggested that the associated impacts – which were much greater than those associated with daily variations in PM – be included in sensitivity analyses only, because the underlying cohort studies were not yet widely used for quantification of impacts.

The second comprehensive review of the literature on air pollution and health, with a view to revising the core set of CRFs for quantifying health effects of pollution in Europe, was carried out in 1997-98. This was necessary because ongoing research on air pollution and health had led to new information, including a much wider set of results in Europe, especially from the APHEA project. Also understanding within the air pollution and health research community continued to mature. In particular it was becoming more widely accepted that the US cohort studies, showing that longer-term (i.e. annual average) exposure to ambient PM2.5 was associated with increased risks of mortality, were valid and expressed relationships that probably were causal. As noted above, the effects as estimated from the cohort studies dominated those of short-term exposures ('daily variations'); the more so when attempts were made to take into account the vulnerable health of those most at risk from death from short-term exposures. There were, however, important issues in how to estimate and value the associated impacts on mortality. The ExternE team – including the

present authors – had become uneasy about using 'attributable deaths' as the key outcome from the cohort studies, and had a major programme of development of methodology to express results in terms of life expectancy instead. In other respects, the review covered similar topics as before, but with use of updated information, and so with changes in recommended CRFs. Results and recommendations were published as part of the second ExternE Methodology Report (1999). These formed the basis of ExternE-related work in the subsequent years.

2.3 Developments from 2000 to the Clean Air for Europe Cost-Benefit Analysis (CAFE CBA)

The pace of new research on air pollution and health has continued to grow, as has the understanding of air pollution and health, and reviews and meta-analyses by expert groups. This has led to a range of important developments.

One is that other teams were developing quantification methods in parallel with those of ExternE. We noted earlier the work of Bart Ostro in the early 1990s. With co-workers, he has continued to be a key figure on health impact assessment (HIA) methods, in projects for the US EPA and for the World Bank. Within Europe, Künzli and colleagues carried out a major analysis of the health effects of air pollution in three countries and of transport-related air pollution (Künzli et al., 2000). The APHEIS project, while focusing on mortality and hospital admissions as health impacts, was doing important work, including on background rates of morbidity in various European cities; and the World Health Organisation (WHO), working within the Convention on Long-Term Transboundary Air Pollution (CLRTAP), was also addressing the issue.

The ExternE team recognised the need to link in more closely with the work of other teams such as these. One reason was to contribute to and to benefit from the exchange of experience in what was a rapidly developing field. Another was to help underpin the credibility, and so the acceptability, of the ExternE work. On that basis, Leo de Nocker and Rudi Torfs from VITO in Flanders, and Fintan Hurley from the IOM, visited Dr. Michal Krzyzanowski of WHO, then based at Bilthoven. Discussions with Dr. Michal Krzyzanowski of WHO were followed by a WHO workshop on quantifying the health effects of outdoor air pollution, held in Bilthoven in November 2000 (WHO, 2001), and a much closer subsequent collaboration with WHO and others, including within the Health Impact Assessment Working Group of AIRNET.

Finally, ExternE researchers, notably Ari Rabl in Paris, and Brian Miller and Fintan Hurley in Edinburgh, had continued to develop the methodology for estimating changes in life expectancy rather than 'attributable deaths' from long-term exposure to $PM_{2.5}$.

To some extent these and other 'new' issues were identified in the various follow-on projects of ExternE (e.g. DIEM, NEWEXT) and, where practicable, adjustments were made to the core ExternE quantification framework. That process continued through the MAXIMA project, whose final report in 2005 (ExternE, 2005) incorporated the most up-to-date version of the ExternE methodology at that time.

It was clear, however, when the NEEDS proposal was being prepared, that a third comprehensive review was necessary, with the twin aims of (i) updating the ExternE quantification framework for air pollution and health in the light of new evidence and understanding; and (ii) aligning the ExternE / NEEDS recommendations more fully with those of the World Health Organisation (WHO).

Fortunately, the health impact assessment (HIA) for the cost-benefit analysis (CBA) of the Commission's major Clean Air for Europe (CAFE) programme had similar requirements; and the CAFE CBA team was very strongly linked with ExternE, including having one of us (FH) leading on developing the methodology for HIA within CAFE CBA. This allowed joint effort on the two projects, i.e. NEEDS and CAFE CBA, and so a far more comprehensive analysis for each one that would have been possible with the funding for either project only.

Work on the present project can be seen as consisting of two parts. First, there was a comprehensive review of quantification issues, as a joint exercise of CAFE CBA and NEEDS. Secondly, within NEEDS we carried out further work on a range of issues. This present report includes a short account of the joint work between CAFE CBA and NEEDS; and follows this with chapters of further work since the CAFE-NEEDS literature review.

2.4 Work since the CAFE-NEEDS literature review; strategy for CRFs and impact functions in NEEDS.

Work since the CAFE-NEEDS HIA literature review has focussed on three broad sorts of issues.

i. We have re-visited some of the framework issues of CAFE CBA and the CRFs in PM and ozone used there.

ii. Secondly, we have considered some other pollutants, notably gases other than ozone, and metals. (paragraph 4.1 and 4.2)

iii. Thirdly, we have examined some issues not considered by CAFE, especially aspects of indoor air pollution (chapter 9). Where necessary we have repeated and extended our views on issues that keep being raised, like the transferability of epidemiological results from the US to a European HIA (chapter 7).

We have been cautious in changing the CAFE CBA recommendations in the light of these updates. This is because we recognise that CAFE CBA now has a particular status within air pollution HIA in Europe. It is, where relevant, consistent with recommendations of WHO in its own expert groups, and within the UNECE Convention on the Long-Range Transport of Air Pollutants (CLRTAP). Its methodology has been open to consultation and comment, including peer-review by a top US team. And it has been used by DG Environment on behalf of the Commission to inform policy development, including of proposals for the control of ambient PM within the EU. There is, clearly, an advantage in using the CAFE-CBA methodology in projects such as NEEDS. We recognise also, however, that the evidence on air pollution and health, and the understanding of it, continue to evolve and that there is an advantage in also maintaining consistency with earlier ExternE-related projects carried out by many of the partners of Stream 1b of NEEDS (but of course, where CAFE CBA and ExternE are different, it is not possible to be consistent with both at the same time).

Thus, our 'bias' towards maintaining consistency with CAFE CBA does not preclude us from making changes. Rather, it means that we will make changes only where

(i) the evidence in favour of a change is strong and

(ii) the change is practically important.

And even in these circumstances, we think that both the CAFE CBA and alternative recommendations be implemented, so that the effects of various options can be seen.

3 HEALTH IMPACT ASSESSMENT IN 'NEEDS' AND IN THE CLEAN AIR FOR EUROPE (CAFE) COST-BENEFIT ANALYSIS (CBA)

3.1 WHO-led work for CAFE

The CAFE programme was and is managed by DG Environment. In the period of overlap with the work for NEEDS, it was under the overall direction of Dr. Matti Vainio, while Dr. André Zuber had a special responsibility for health effects estimation. CAFE was a large-scale and complex exercise, of which the CBA work was only one part. In earlier phases of the CAFE project WHO, under the leadership of Dr. Michal Krzyzanowski, had carried out a series of reviews of the health effects of particles, ozone and NO_2. These reviews were in the form of answers to questions from the CAFE Steering Group (WHO, 2003) and answers to CAFE Follow-up Questions (WHO, 2004a). Both of these reviews focused on hazard assessment, with evidence-based commentary on issues such as whether or not there are thresholds of the effects of these pollutants, or what kinds of particles are responsible for the adverse health effects of PM. They did not provide specific CRF information for use in quantification.

Two other WHO initiatives at about the same time did, however, provide quantitative recommendations. One of these was a series of annual meetings of the WHO-led Task Force on Health (TFH) of the UNECE Convention on the Long-Range Transport of Air Pollutants. The TFH provided specific quantitative guidance to the RAINS Integrated Cost-Effectiveness Model of IIASA in Austria. The guidance was focused on two areas:

- Effects on mortality (life expectancy) of long-term exposure to ambient $PM_{2.5}$; and
- Effects on mortality (attributable lives shortened) of daily variations in ambient ozone.

As well as proposing specific functions for quantification of these mortality endpoints, TFH of CLRTAP also set out positions on a number of framework issues relevant to quantification. These included that:

1. Quantification of the classical air pollutant mixtures be restricted to CRFs expressing the effects of PM and of ozone;
2. Quantification of PM be based on anthropogenic PM, without threshold;
3. Components of the $PM_{2.5}$ ambient urban mixture be quantified (per $\mu g/m^{-3}$) the same as the overall mixture itself; i.e. that the known or suspected differences in toxicity between different components of $PM_{2.5}$ be ignored for quantification purposes; and that

4. Quantification of ozone also be without threshold, but that ozone effects be estimated only above 35 $\mu g/m^{-3}$ (8-hr daily max), on days when this level is exceeded.

In addition, Ross Anderson and colleagues in London carried out a WHO-sponsored meta-analysis of studies in Europe linking, variously, PM_{10} or $PM_{2.5}$ or ozone with (i) all-cause and cause-specific mortality; (ii) respiratory hospital admissions or cardiovascular admissions; (iii) days of additional medication use in people with chronic lung disease; and (iv) cough days in people with chronic lung disease (WHO, 2004b).

Strategy for CAFE CBA in relation to WHO reviews

It was decided that the quantification of health effects within CAFE CBA would be as consistent as practicable with all four of the above WHO initiatives (two qualitative and two quantitative). This was partly to maintain consistency within the CAFE process and partly because of the authority of WHO. It was recognised however that CAFE CBA would include a possibly wide range of morbidity endpoints, if suitable C-R functions could be found, and if background morbidity rates could also be estimated suitably. Thus, the situation had arisen whereby there was a perfect alignment of goals between the planned literature review for NEEDS and the planned literature review for CAFE CBA.

3.2 Scope, coverage and process of the CAFE-NEEDS literature review for HIA

As noted above, the sharing of review work for the two projects enabled a substantially more thorough outcome than would otherwise have been possible. This was fortunate because overall, the review work itself was substantially more than had been anticipated. There were several reasons for this.

Reasons related to mortality and long-term exposure to PM

The focus of early work on CAFE CBA was on reconciling two life table approaches to estimating the life expectancy gains of reducing ambient $PM_{2.5}$. These were, respectively, the approach of the CAFE CBA team, based on experience within ExternE and the approach of RAINS, together with some of the framework assumptions of WHO. While there were many similarities in the two approaches, there were some important differences also, and work was needed to understand these differences and their implications. The differences included:

- Differences in choice of coefficient from Pope et al. (2002);
- The WHO framework decision to apply the same coefficient to all PM in the $PM_{2.5}$ (or PM_{10}) size range, regardless of source, composition or other characteristics that might be relevant to toxicity.

In addition, whereas the ExternE team had for some year not estimated 'attributable deaths', on the grounds that the methods for doing so were suspect, a decision was taken (partly in the light of the comments of external reviewers) that 'attributable deaths' would be estimated. This led to unexpected methodological work in order to clarify the relationship between 'attributable deaths' and changes in life expectancy. The CAFE CBA/ NEEDS work has involved also examining two other methodological questions relevant to life tables. These are:

- The relationship between results obtained from a sustained reduction in pollution, with associated sustained reductions in mortality hazards, and a set of consecutive one-year 'pulse' reductions, with associated consecutive temporary reductions in mortality hazards. This is necessary because benefits and costs are compared on annual basis.
- The possible shape and importance of what is known as the 'cessation lag', i.e. the time-period between reductions in pollution and consequent reductions in mortality.

Other reasons

The existing ExternE CRFs were all fully reviewed. Some were dropped from consideration. Others were changed considerably in the light of new evidence in intervening years. Where CRFs were taken from the same source studies as previously used in ExternE, those source studies were re-reviewed in detail, including in the light of their usage by other HIA teams, and where appropriate somewhat different CRFs were proposed, or the rationale for the functions used was described in much greater detail. This applied in particular to two sets of studies whose results were known (from previous ExternE work) to have a substantial impact on final answers. These were:

- Estimates of the relationship between long-term exposure to PM and development of new cases of chronic bronchitis in adults, based on the US AHSMOG study; and
- Estimates of the relationship between PM and/or ozone and restricted activity days (RADs), or variants of the same.

Some new pathways were quantified, notably long-term exposure to PM and increased mortality in infants, and short-term exposure to PM and work days lost. Very substantial new work was carried out to produce better estimates than previously of background rates of morbidity across the EU-25. The entire process was subject to formal expert peer-review, the reviewer for health effects

estimation being Dr. Bart Ostro of the California EPA (Krupnick et al., 2004). This led to a number of revisions and improvements, and further under-pinned the reliability of the work. The entire process was also subject to close and detailed stakeholder involvement. This required resource and attention, including in due course a detailed response to the written comments of the industry body UNICE.

3.3 Outputs from the CAFE-NEEDS HIA literature review

Four detailed reports from the CAFE CBA/ NEEDS literature review have already been published on the World-Wide Web.

a. Of these, that by Hurley et al. (2005a) is the most relevant to the present report, in that it gives the detailed literature review, and the associated recommendations for CRFs and for background rates, that underpin the quantification of health in CAFE CBA. The report also includes discussion of a wide range of relevant methodological issues. A summary of the literature review is given in annex 1, including the list of selected CRF for PM and ozone.

b. Holland et al. (2005a) is an overview of the whole CAFE CBA Methodology, including a summary overview of health. Its description of health quantification is a good alternative for those who do not wish or need to know the full details.

c. Following detailed comments by UNICE as part of the consultation process, the CAFE CBA team prepared a detailed response (Hurley et al., 2005b). Although referring to an earlier draft of the overall methodology than was published as Hurley et al. (2005a), the UNICE comments and the CBA team's response are informative about uncertainties within the evaluation.

d. Holland et al. (2005b) is a complementary and quantitative analysis of uncertainties associated the quantification of health in CAFE CBA.

Of course these reports also benefited enormously from the involvement of other authors who are not part of this WP of NEEDS, especially Dr. Mike Holland (EMRC), Paul Watkiss (AEA Technology Environment) and Dr. Alistair Hunt (University of Bath/ Metroeconomica). In addition, summaries of the CAFE-NEEDS methodology and results are included in two WHO publications, one on particulate matter (WHO, 2006), the other on ozone (in preparation). We have drawn on those summaries in preparing the present report.

Table 3.1: Overview of the concentration response functions for PM and ozone. Functions for sensitivity analysis in italics.

Endpoint	Pollutant (1)	Age group	Risk group fraction	CRF (95% CI)	Units
CORE CRF					
Chronic mortality					
Life expectancy reduction	PM2.5	30+	1	651 (127; 1194)	YOLL per 10 µg/m³ per 100 000 people
Infant mortality					
Increased mortality risk	PM10	0-1	0.4%	4% (2%; 7%) 18	attributable cases per 10 µg/m³ YOLL per 10 µg/m³ per 100,000 people (all ages)
Acute mortality					
Increased mortality risk	O_3/SOMO35	all	0.99%	0.30% (0.1%; 0.43%) 0.75	attributable cases per 10 µg/m³ YOLL per case
Morbidity					
New cases of chronic bronchitis	PM10	27+	0.376%	26.5 (-1.9; 54.1)	per year, per 10 µg/m³, per 100,000 adults aged 27+
Respiratory hospital admissions	PM10	all	1	7.03 (3.83;10.3)	per year, per 10 µg/m³, per 100,000 people
Cardiac hospital admissions	PM10	all	1	4.34 (2.17; 6.51)	per year, per 10 µg/m³, per 100,000 people
Medication use / bronchodilator use	PM10	5-14	PEACE criteria (15% N&E-EU) (25% W-EU) Medication use 10%	180 (-690; 1060)	per year, per 10 µg/m³ per 1000 children meeting the PEACE criteria
	PM10	20+	Asthmatics 4.5% Daily medication use probability 50%	912 (-912; 2774)	per year, per 10 µg/m³ per 1000 adults 20+
Lower respiratory symptoms	PM10	adults	symptomatic adults (30%)	1.3 (0.15; 2.43)	symptom days per year, per 10 µg/m³ per adult with chronic respiratory symptoms
	PM10	5-14		1.86 (0.92; 2.77)	symptom days per year, per 10 µg/m³ per child 5-14
Restricted activity days (RADs)	PM2.5	15-64	1	902 (792;1013)	per year, per 10 µg/m³ per 1000 adults 15-64
Work loss days (WLD)	PM2.5	15-64	1	207 (176; 208)	per year, per 10 µg/m³ per 1000 adults 15-64
Minor restricted activity days (MRAD)	PM2.5	18-64	1	577 (468; 686)	per year, per 10 µg/m³ per 1000 adults 18-64
Respiratory hospital admissions	O_3/SOMO35	65+	1	12.5 (-5; 30)	per year, per 10 µg/m³ per 100 000 people 65+
MRAD	O_3/SOMO35	18-64	1	115 (44; 186)	per year, per 10 µg/m³ per 1000 adults 18-64
Medication use / bronchodilator use	O_3/SOMO35	20+	Asthmatics 4.5%	730 (-225; 1570)	per year, per 10 µg/m³ per 1000 asthmatic adults 20+
LRS excluding cough	O_3/SOMO35	5-14	1	0.16 (-0.43; 0.81)	days of LRS per year, per 10 µg/m³ per child 5-14
Cough days	O3/SOMO35	5-14	1	0.93 (-0.19; 2.22)	cough days per days, per 10 µg/m³ per child 5-14
SENSITIVITY FUNCTIONS					
New cases of chronic bronchitis	*PM2.5*	*27+*	*0.376%*	*53.3 (-1.7; 113.4)*	*per year, per 10 µg/m³, per 100,000 adults aged 27+*

Endpoint	Pollutant (1)	Age group	Risk group fraction	CRF (95% CI)		Units
Consultations with primary care physicians						
Asthma	*PM10*	*0-14*	*1*	*1.18*	*(0; 2.45)*	*per year, per 10 µg/m³ per 1000 children 0-14*
	PM10	*15-64*	*1*	*0.51*	*(0.2; 0.82)*	*per year, per 10 µg/m³ per 1000 adults 15-64*
	PM10	*65+*	*1*	*0.95*	*(0.32;1.69)*	*per year, per 10 µg/m³ per 1000 adults 65+*
Upper respiratory diseases	*PM10*	*0-14*	*1*	*4*	*(-0.6 ; 8)*	*per year, per 10 µg/m³ per 1000 children 0-14*
	PM10	*15-64*	*1*	*3.2*	*(1.6-5)*	*per year, per 10 µg/m³ per 1000 adults 15-64*
	PM10	*65+*	*1*	*4.7*	*(2.4-7.1)*	*per year, per 10 µg/m³ per 1000 adults 65+*
Acute respiratory symptoms	*PM10*	*all*	*1*	*4650*	*(210; 9090)*	*symptom days per year, per 10 µg/m³ per 1000 people*
Consultations with primary care physicians for allergic rhinitis	*O_3 /SOMO35*	*0-14*	*1*	*3.03*	*(1.89; 429)*	*per year, per 10 µg/m³ per 1000 children 0-14*
	O_3 /SOMO35	*15-64*	*1*	*1.6*	*(1.22; 2.03)*	*per year, per 10 µg/m³ per 1000 adults 15-64*
Medication use / bronchodilator use Northern and Eastern Europe	*O_3 /SOMO35*	*5-14*	*1*	*124*	*(18; 227)*	*per year, per 10 µg/m³ per 1000 children 5-14*
Medication use / bronchodilator use Western Europe	*O_3 /SOMO35*	*5-14*	*1*	*310*	*(44; 569)*	*per year, per 10 µg/m³ per 1000 children 5-14*

(1) SOMO35 is the 8hr-averaged maximum O3 concentration increment above 35 ppb (70 µg/m³), aggregated over all days of one year.

Since Work Loss Days (WLD), Minor Restricted Activity Days (MRAD and days in hospital because of cardiac hospital admissions (CHA) an Respiratory Hospital Admissions (RHA) are also RADs the net RADs are calculated as:

netRAD (per µg/m³ $PM_{2.5}$) = RAD – WLD – WRAD – (RHA and CHA due to PM_{10}) x 10 days, i.e.:

netRAD (per µg/m³ $PM_{2.5}$) = (0.0902-0.0207) x Adults_15_to_64 – 0.0577 x Adults_18_to_64 – (7.03 + 4.34) x 10^{-6} x 5/3 x Total_pop x 10 days per year , per µg/m³ PM2.5

4 SELECTION OF C-R FUNCTIONS FOR OTHER AIR POLLUTANTS

4.1 Quantifying a direct effect of gases other than ozone

Attributing effects to particles or to the gases

Separating out the roles of SO_2, NO_2 and PM_{10} is particularly problematic, given that they tend to vary together in most locations and studies. It is not clear to what extent the apparent effects of PM are in reality a reflection of effects of NO_2 or SO_2 or vice versa, or whether the presence of other pollutants affects the toxicity of PM. Thus there are uncertainties in applying CRFs in a situation where the ambient pollutant mixture is different from the one where the original epidemiological study was carried out.

Following recommendations of WHO and the work of CAFE, the current position of ExternE/ NEEDS is to use only CRFs for PM and O_3, but none for SO_2 or NO_2, a choice also made in several other health impact assessments. However, the situation is not clear and opinions could change as further evidence comes to light. In particular, the Hong Kong intervention study showed a sustained benefit in mortality reductions following reductions in pollution involving SO_2 mostly (Hedley et al., 2002). There could indeed be significant direct effects of SO_2, contrary to the current position of ExternE. Although WHO revised its air quality guideline downwards for SO_2 (WHO, 2005), it is also stated that this is primarily a precautionary approach, assuming that reduction in exposure to a causal and correlated substance is achieved by reducing sulfur dioxide concentrations.

There is also evidence from the APHEA study that NO2 has a modifying effect (Katsouyanni et al., 2001) on acute mortality risks of PM_{10}. It is not sure if this is because NO2 is toxic, or rather if it is an indication of a higher traffic-related PM contribution, thus hinting at components in PM emissions from cars being more toxic than more general ambient particles.

In previous ExternE work an acute and independent effect of SO_2 and NO_2 was examined. The evidence at the time did not reject the possibility of a small acute effect in addition to the acute effects of particles and ozone. In the first ExternE methodology report (1995) no effects of NO_2 were quantified. On the basis of the evidence at that time SO_2 was also considered to be a marker for particulate air pollution, and hence no acute effects of SO_2 were identified either. When the first APHEA results on acute mortality and morbidity and air pollution came out, it was decided in the second methodology report (1998) to take into account an acute effect of SO_2 on mortality and hospital admissions in the core set of CRF, and acute effects of NO_2 were

considered in a sensitivity analysis. After 1999 the role of particles in air pollution related health effects gained importance, and most epidemiological studies concluded that NO_2 and SO_2 related acute effects were in fact a substitute for particulate air pollution. In the recent ExternE 2005 report, again no CRF of SO_2 and NO_2 were used in the set of CRF. Although our position has changed, according to the most recent epidemiological evidence available, this has not played an important role in the overall health impact assessment. A ranking of energy technologies for example does not change when acute effects of SO_2 or NO_2 are included or excluded. In contrast the role of the SO_2 and NO_x derived secondary pollutants in the chronic effects of particulate air pollution does affect the outcome of a health impact assessment. Especially for gas-fired power plants, with virtually no particle and SO_2 emissions, but only NO_2 emissions, the relative importance of nitrates in the PM mixture, has a strong effect on the overall externalities of these technologies (see also discussion on chronic effects)

Nitrogen dioxide (NO_2)

But scrutiny remains about the exact role of NO_2 in ambient air pollution. Although NO_2 can be associated with respiratory effects in both adults and children, it is unclear whether these effects are caused by NO_2 or by the mixture of pollutants they represent. Traffic related emissions for example are well characterized by NO_2 in cities, but include a wide range of unmeasured (organic) pollutants as well, that might influence or trigger the health effects. It remains therefore difficult to separate the role of NO_2 in acute effects on health. Toxicological studies and studies of indoor exposure to NO_2 show in general that at ambient levels of NO_2 lung function changes or other acute respiratory effects are undetectable. Currently our position is based on the WHO review, and remains that there is no compelling new evidence to include acute effects of NO_2 into the health impact assessment.

There is also no basis, from the ACS study, to quantify an effect of long-term exposure to NO_2 on mortality. In a cohort study of 16209 men from Oslo Norway, indicators of home address NO_x exposure, were associated with an increased risk of respiratory (RR=1.16, 95% CI 1.06–1.26 per 10 μg/m3 increase in NO_x exposure), ischemic heart disease (RR=1.08, 95% CI 1.03-1.12 per 10 μg/m3 increase in NO_x exposure) and lung cancer mortality (RR=1.11, 95% CI 1.03-1.19 per 10 μg/m3 increase in NO_x exposure), but not for cerebrovascular disease (Nafstad 2003 and 2004). In the same study no effects were found for SO_2 exposure at home address. Particle or black smoke exposure was not assessed in this study and the authors also note that the contrasting results for NO_x and SO_2 might be an indication of traffic related air

pollution. It is our opinion that this study therefore is not sufficient to prove an independent long-term effect of NO_2 on mortality.

There is some evidence of an effect of long-term exposure to NO_2 on morbidity, especially the respiratory morbidity of children. In the Southern Californian Children's Study a statistical significant association was found between lung development and yearly average NO_2 concentrations in children living at different locations (Gauderman, 2004). However the association was also found with $PM_{2.5}$ and was even stronger with a measure of acid vapour, including nitric acid (HNO_3) and formic and acetic acid.

The SAPALDIA Study in Switzerland gives support to the association of NO_2 exposure and lung function decrements among adults living in different communities. But also here there is a potential for confounding with PM10. When the intra-urban exposure to NO_2 was taken into account there was still evidence of a –albeit smaller- chronic NO_2 effect on lung function, although the PM_{10} concentrations remained largely unchanged in the cities considered (Ackermann-Liebrich, 1997; Schindler, 1998). At this moment the discussion of the long-term morbidity effects of NO_2 is undecided. More studies and longer follow-up periods are needed to establish a clear relationship.

Sulphur dioxide (SO_2)

Similar to NO_2, SO_2 plays an important role in the mixture of air pollutants, as gas but also a precursor of secondary (fine and ultrafine) particles. The latter contribution to $PM_{2.5}$ is taken into account when calculating the long-term effects on mortality based on the Pope et al. (2002) extended follow up of the ACS cohort; and of course secondary sulphate particles more generally contribute to all anthropogenic particle effects, via their contribution to $PM_{2.5}$ and/or PM_{10}. This is discussed further in chapter 6.

The role of SO_2 as a gas, within the overall air pollution mixture, remains unclear and controversial. Early evidence, from air pollution episodes such as the London smog, involved greatly elevated levels both of particles (measured as black smoke) and of SO_2; and air quality guidelines were set to control the black smoke / SO_2 mixture. In terms of causality and mechanisms, there was speculation that acidity of the mixture was an important part of its toxicity.

Further insight was given by time series studies of the effects of acute exposure ('daily variations in pollution') on mortality and hospital admissions, through the 1990s. Evidence from North America pointed to particulate matter (PM) rather than to SO_2 as the main driver of these relationships. This was based on two kinds of evidence. One was that PM effects were identified in cities where SO_2 concentrations were very low; and the magnitude of the estimated PM effects seemed relatively invariant to whether SO_2 was present or not. Secondly, analyses from two-pollutant models, i.e. simultaneously including both PM and SO_2 as explanatory variables, favoured PM as the pollutant driving the relationship. Nevertheless, there remained some evidence, from areas of high SO_2 (i.e. north-east USA, south-east Canada), that acidity may be a factor. And analyses of single-pollutant models in Europe, as part of the APHEA programme, continued to pick up associations between daily variations in SO_2 and both mortality and hospital admissions. On the basis of this evidence, ExternE (1998) quantified a limited number of SO_2-health endpoints for application in Europe, while making clear that the evidence for direct effects of SO_2 as a gas remained limited and controversial.

Two sets of evidence in particular have led to some re-assessment in favour of a specific role for SO_2 as a gas. One is the set of studies examining the effects on mortality and morbidity of an intervention to reduce air pollution in Hong Kong. This required that, from 1 July 1990, *"all power plants and road vehicles in Hong Kong were restricted to use fuel oil with a sulphur content of not more than 0.5% by weight."* (Hedley et al., 2002). The key study, Hedley et al. (2002), examined differences in deaths over 5 years from July 1990, in districts of Hong Kong with and without sustained reductions in pollution (SO_2) relative to baseline – sustained being interpreted as the reductions measured as at 2.5y after the change. There was an immediate, substantial and sustained reduction in SO_2; an immediate reduction in sulphates which, because of regional effects, returned to baseline levels over the following 3-5years; and non reductions in PM_{10} or NO_2. Changes in the sulphur content of the fuel did however lead to changes in the nature of the PM mixture which may have affected its toxicity, including via transition metals on the particle surface (Lippmann and Ito, 2006). Monthly deaths had been *increasing* on average by 3.5% per annum. in the five years prior to the policy intervention. There was a clear and sustained reduction in this increase for all causes and all ages over the following five years. The change was greatest for respiratory causes and also for cardiovascular, with a much lesser reduction for lung cancer and for other non-cancer causes. Cancers other than lung cancer increased as before the intervention. The change occurred in the high SO_2 reduction areas, and not in the low SO_2 reduction areas.

The second set of evidence comes from analyses of the American Cancer Society (ACS) cohort. All the main papers – the original paper of Pope et al (1995); the re-analysis by Krewski et al (2000); and the extended mortality follow-up of Pope et al (2002) – give their principal results in terms of particles, expressed as $PM_{2.5}$ or, in the earlier reports, as sulphate particles also. However, the re-analysis by Krewski et al showed a robust association between SO_2 as a gas and mortality; and the risk coefficient for PM was reduced markedly in two-pollutant models including both $PM_{2.5}$ and SO_2. However, the association with SO_2 disappeared when analyses were carried out on a smaller spatial scale (Willis et al, 2003). The extended follow-up of Pope et al (2002) is the most powerful study, with three times as many deaths as the earlier analyses. Clear associations were found linking $PM_{2.5}$ with all-cause mortality, with cardio-respiratory causes and with lung cancer; but not with other causes. Statistically significant associations were also found linking both SO_2 and sulphates with all-cause mortality, with cardio-respiratory, with lung cancer and with all other causes. The finding of association with 'other' causes is curious. A possible explanation is that annual average SO_2 and sulphates are correlated with some unmodelled regional confounding factors that affect general mortality risks.

In terms of quantification, there are five main questions.

a. What commonly measured aspect of the air pollution mixture best captures the adverse effects on mortality, from long-term exposure? It is very widely accepted that particulate matter, measured as $PM_{2.5}$, is the best available single index. Like others, it is the index on which we base our main quantification.
b. Does a risk coefficient in annual average $PM_{2.5}$ capture the full mortality effects of long-term exposure to air pollution? Arguably it does not. Though hugely important for policy purposes, $PM_{2.5}$ is a relatively crude measure of particulate air pollution, in that it does not *per se* capture characteristics such as surface properties, transition metals, surface area or particle number, which may affect toxicity.
c. Does the evidence support an independent role for SO_2 as a gas, in addition to an effect of $PM_{2.5}$? The evidence regarding SO_2 from both short-term and longer-term exposures is assessed in the recent review by Lippmann and Ito (2006) as part of WHO's Air Quality Guidelines Global Update. There is some weight of opinion towards recognising a particular effect of pollution from combustion of sulphur-containing fuels and the associated global update of WHO air quality guidelines recommended lowering the SO_2

24-h average guideline value from 125 µg/m³ to 20 µg/m³. Reasoning behind this was a) that it was feasible in practice, given that a number of policy measures on fuel quality and stationary combustion sources were implemented, and b) that reduction of SO_2 would probably lead to reduction of correlated health relevant compounds. It is not at all clear, however, that this should translate into quantifying an independent or additional effect of SO_2, as part of the overall air pollution mixture. The WHO (2003, 2004) evaluations of air pollution hazards in the form of answers to questions for CAFE focussed on PM, NO_2 and ozone; they did not consider SO_2 specifically. The WHO-led Task Force on Health of the UNECE Convention on Long-Range Transboundary Air Pollution (CLRTAP) did not attempt to quantify an independent or 'additional' effect of SO_2, and so neither did CAFE Cost Benefit Analysis (CBA). Similarly, the US EPA quantifies long-term exposure and mortality in terms of $PM_{2.5}$ only, and not of SO_2 in addition. Given the weight of evidence, and current understanding of its interpretation, we have concluded to follow the current approach in the CAFE CBA not to quantify a long-term SO_2 effect on mortality.

d. Should the coefficient for long-term exposure to $PM_{2.5}$ and mortality be adjusted for the effect of SO_2? Not unless a main effect of SO_2 is quantified.
e. What about morbidity effects, or mortality from acute exposures ('daily variations')? Although ExternE (1995) did not quantify SO_2 effects, the evidence from APHEA tipped the balance for ExternE (1998) and, despite doubts about causality, ExternE (1998) did include some quantification of SO_2 effects, as if they were independent. If the apparent associations with SO_2 arise from sulphur-containing combustion rather than from SO_2 as a gas, from the point of view of health impact assessment this could however lead to double counting; and therefore, for this reason and for consistency with CAFE, it is decided not to quantify any direct SO_2 effect.

4.2 Quantifying an effect of metals

4.2.1 General Remarks

By far the most toxic metals that are emitted to the environment in significant amounts are: As, Cd, Cr, Hg, Ni and Pb. They cause a wide variety of health impacts. Of these cancers and neurotoxicity are of particular concern. As, Cd, Cr and Ni are carcinogenic, and Hg and Pb neurotoxic. The unit risk factors and slope factors for these metals are summarized in Table 4.1. Further remarks follow here.

Table 4.1: ERFs recommended for carcinogens. Slope factor = probability of cancer per daily unit intake during 70 years. Unit risk factor = probability of cancer from lifetime exposure to a concentration of 1 μg/m^3.

	Air unit risk per μg/m^3	Drinking water unit risk per μg/L	Oral slope factor per mg/kg$_{body}$/day
PCB [a, b]	1×10^{-4}	1×10^{-5}	4×10^{-2}
Dioxins TEQ [b]			1×10^{6}
Benzene	4×10^{-6}		
Benzo[a]pyrene	1×10^{-7}		
As (inorganic)	4.3×10^{-3} [d]	5×10^{-5} [e]	1.5 [e]
Cd	1.8×10^{-3} [d]		
Cr^{VI}	1.2×10^{-2} [d]		
Ni (refinery dust)	2.4×10^{-4} [d]		

[a] haematologic cancer, cancer of the liver, gall bladder, gastrointestinal tract and biliary tract, and skin cancer.
[b] the numbers for PCB and dioxins are probably an overestimate, and Searle (2005) suggests that they may be 5 times smaller; furthermore, mortality rate for such cancers may be around 50%.
[c] this is probably an overestimate, to be used for sensitivity analyses only.
[d] lung cancer, mortality rate about 90%.
[e] lung cancer, internal organ cancers and skin cancer.

In reviewing the information on ERFs for ExternE, one has to keep in mind that the project is to help policy makers formulate instruments for reducing environmental pollution that bring the greatest benefit. Therefore the ERFs should correspond to end points that can be evaluated in monetary terms. That is often a problem in practice because many, if not most of the end points for which information is available, are difficult or impossible to value in monetary terms, at least at the present time, for instance lung function reduction. Another consideration is the link to exposure: policy makers need to know the benefit of reducing the emission of a pollutant or the exposure to it. Again, this can pose a problem because some ERFs are available as a function not of exposure or dose, but of bio-indices such as the concentration of Pb in blood. Such information can be used in the present context only if the relation between the bio-index and the exposure or dose is also known. In the present review end points that do not meet all these criteria will not be examined in detail. Another source of information is the Espreme project (http://espreme.ier.uni-stuttgart.de/) were an independent review of metals was made. The Espreme project includes other endpoints than cancer. For lung cancer unit risks are different from the ones presented in Table 4.1 for As (1.5×10^{-3} in Espreme compared

to 4.3 $\times 10^{-3}$ above), Ni (3.8×10^{-4} vs.2.4×10^{-4} above) and CrVI (4×10^{-3} vs. 1.2×10^{-2} above). It illustrates differences between risk factors used by EPA (http://www.epa.gov/iris) and risk factors given in the WHO air quality guidelines (WHO, 2000) For completeness we have included the Espreme tables in our summary.

4.2.2 Arsenic

The effects of arsenic in humans are best known from studies of populations that have been exposed to arsenic in drinking water; some information has also been derived from studies of exposed workers. The most clearly identified impacts are lung cancers, due to inhalation and ingestion. Other effects include skin lesions, other cancers (liver, kidney, bladder and skin), cardiovascular mortality, hypertension, diabetes, and neurological effects; however, for these end points the ERFs at low exposures are less reliable and not considered sufficient for inclusion in an external cost assessment.

For cancers due to inhalation EPA IRIS (Integrated Risk Information System, http://www.epa.gov/iris/) lists a URF of 4.3×10^{-3} per $\mu g/m^3$. Ingestion is considered carcinogenic, with slope factor 1.5 per mg/(kg.day), but only for inorganic As; for organic As compounds EPA and the International Agency for Research on Cancer do not indicate any evidence for carcinogenicity. However, most of the ingestion dose is organic, with the exception of drinking water which is inorganic.

4.2.3 Cadmium

The effects of Cd in humans have been established from studies of populations in Japan who consume Cd in rice grown on contaminated soil, of populations who live near major industrial sources in northern and western Europe and who are exposed in air and in food, and of populations who are exposed to Cd in the work place. Most studies have assessed effects in relation to urinary or blood Cd levels. Other studies have assessed effects in relation to concentrations of Cd in food or estimated lifetime intake of Cd in food.

Among the numerous end points are lung cancer, osteoporosis, renal dysfunction, diabetes, and mortality. Of these only lung cancer meets the criteria for the present assessment, i.e. reliability of the ERF and availability of monetary values. EPA IRIS shows a URF for lung cancer due to

inhalation of Cd, $1.8\times10^{-3}/\mu g/m^3$ in ambient air, and it seems unlikely that dietary Cd contributes much to lung cancer risk.

4.2.4 Chrome

The effects of Cr in humans are best known from studies of workers exposed to airborne Cr. There is a relatively well established relationship between exposure to hexavalent Cr (Cr^{VI}) and adverse respiratory effects including cancer. There is no information about effects in populations exposed to Cr in water or food. Limited information about the toxicity of ingested Cr is available from animal studies. Most of the Cr present in ambient air, water and food is in the trivalent rather than the hexavalent state.

The ERF information available for Cr is predominantly for Cr^{VI}, with the exception of some limited information about "mild ill health" that is linked to total Cr exposure. There is no evidence that trivalent Cr is associated with cancer, but it is plausible that trivalent Cr could have adverse effects on kidney function. The relative potency of trivalent Cr in comparison with other heavy metals has not been established. The information for effects on the respiratory system is based on exposure to airborne Cr^{VI} and it seems unlikely that ingested Cr would have a substantial effect on respiratory health. EPA IRIS shows a URF for lung cancer due to inhalation of Cr^{VI} as $1.2\times10^{-3}/\mu g/m^3$ in ambient air. The ERF information for other health endpoints is based on ingested Cr and includes nasal damage, mild ill health, effects on fetal development, and impaired kidney function. But it is not sufficiently reliable for inclusion in external cost calculations.

4.2.5 Lead

There is an extensive literature linking exposure to lead to adverse effects in humans that includes studies of workers exposed to lead in workplace air and children and others exposed to lead in the general environment, often through ingestion of contaminated drinking water. In addition, ingestion of non-food items such as paint is a potentially important source of exposure for young children. Most studies have used concentrations of Pb in blood as an index of exposure. WHO provide some information linking Pb in blood to exposure although the relationships are variable and do not apply well at extreme (low or high) levels of exposure.

Children appear to absorb more Pb than adults for a given level of exposure and also seem to experience adverse effects at lower blood Pb levels than adults. Relatively few studies have attempted to establish dose-response relationships and most have focused on establishing threshold levels of exposure.

There are a variety of impacts on the nervous system, such as cognitive impairment in adults, hearing impairment in children, cognitive impairment in children, reduced IQ of children, effects on nerve conduction, amyotrophic lateral sclerosis (a degenerative motor neuron disease), and brain damage. There is considerable overlap between these endpoints and for most of them there are no monetary values. However, loss of IQ is includes most of these and it can be measured, even for young children; furthermore the monetary valuation for this end point is relatively firm. For these reasons we take reduced IQ of children as proxy for all neurotoxicity of Pb.

Other end points include anemia, hypertension, renal dysfunction, spontaneous abortion, and possible effects on male fertility, but the ERFs are not sufficiently well established at low doses.

The dose-response function for reduced IQ of children is quite well determined, thanks to numerous studies, including a meta-analysis by Schwartz (1994) who found a decrement of 0.026 IQ points for a 1 µg/L increase of Pb in blood, a relation that appears to be linear without threshold. More recently a study designed to identify effects at the lowest doses found an even larger effect, 0.055 IQ points per 1 µg/L, without any threshold (Lanphear et al 2000). Here we continue to use 0.026 IQ points per 1 µg/L, being based on a meta-analysis rather than a single study.

To relate blood level to exposure we use a relation between blood level Pb and ingestion dose, published by WHO (1995). Surprisingly the blood level per ingested quantity is higher at low doses, perhaps because of increased excretion at higher dose or storage in bones. Here we use a lower value in the range of blood levels per dose, 1.6 µg/L per ingested µg/day. Together with the above mentioned 0.026 IQ points per 1 µg/L increase of blood Pb this implies a loss of 0.026 IQ points × 1.6 (µg/L)/(µg/day) × (1 yr/365 days = 1.14E-04 IQpoints/(µg/yr) for a child who ingests a dose of 1.0 µg for 1 year.

One also has to consider the time window during which an exposure causes damage. The sensitivity of the brain to Pb is greatest during pregnancy and the first years of life, although the precise time distribution of the damage is not known. However, as we explain in this paragraph, this does not matter since the result of Schwartz expresses the total impact in a population due to a constant exposure. Furthermore, the half life of Pb in blood and other soft tissues is relatively short, about 28-36 days (although much longer in bones) (WHO 1995). Thus, for the purpose of damage calculations, one can equally well assume that the damage is incurred during a one year exposure by infants between the ages of zero and one only, or during a three year exposure between the ages of zero and three. To see that the effect is the same, note that the fraction of the population between zero and three is essentially three times the fraction between zero and one (the latter being 0.9% to 1.1% in the EU27, but the precise value does not matter for this argument). If the sensitive period is only one year, the loss due to a one year exposure is 1.14E-04 IQpoints/(μg/yr) × 1.1% of population. If the sensitive period is three years, the affected cohort is essentially three times as large but the damage rate three times smaller, so the loss due to a one year exposure is (1.14E-04 IQpoints/(μg/yr))/3 × (3 × 1.1% of population of EU), essentially the same. Therefore one can write the slope s_{ER} of the ERF in terms of a 1 year dose as

$$s_{ER} = f_{1yr} \times \text{1.14E-04 IQpoints/(μg/yr)}, \text{ to be applied to total population,}$$

where f_{1yr} is the fraction of the population between 0 and 1 yr of age.

4.2.6 Mercury

Among the various possible health impacts of Hg, the neurotoxic impacts on fetus and infants have been investigated most thoroughly at low doses and they appear to be the most worrisome and the least uncertain. Rice and Hammitt (2005) also quantify mortality due to coronary heart disease and strokes, both fatal and non-fatal, but we do not consider these ERFs sufficiently certain at low dose. The most important studies have followed cohorts of children among three populations (in New Zealand, the Seychelles, and the Faroe Islands) whose diet contains a particularly large portion of seafood. Based on these studies Trasande et al (2005) have recently estimated the social cost of the IQ decrement that can be attributed to Hg ingestion in the USA. In view of the large uncertainties they consider several possible forms of the ERF, both linear and logarithmic, but all with a no-effect threshold of 5.8 or 8.2 μg/L in the cord

blood of the newborn infant. They also consider several values of the ratio cord blood/maternal blood. Data for the percentage of women with blood concentration above different thresholds are available in the USA, and so Trasande et al can calculate the impacts even with a nonlinear ERF.

The four ERFs considered by Trasande et al are shown in Figure 4.1, together with the ERFs we recommend for external cost calculations. These authors evaluate the ERFs at the points indicated by the markers, but then use these values for the entire population segment with blood concentrations in the interval up to the next point; in other words, they apply the ERFs as if they were equal to the stair cases shown by the thin lines in Figure 4.1.

For the present paper we estimate a lower bound for the ERF by drawing a hockey stick with a threshold at the higher threshold considered by Trasande et al. As upper bound for the ERF we take a line through the origin with the same slope. Approximation by straight lines makes the calculations much simpler and it is certainly justified in view of the uncertainties. We have chosen the slope so that our ERFs are a fair representation of the ERFs of Trasande. The scale of the x-axis is the Hg concentration in cord blood, and the slope of our ERFs corresponds to a loss of 0.2 IQ points/($\mu g/L_{cord\ blood}$).

To apply the ERFs, we need to transform the scale to ingestion dose. First we transform to maternal blood. The concentration in cord blood is higher than in maternal blood, but there is considerable uncertainty about the ratio cord blood concentration/maternal blood concentration. In the present paper we assume a ratio of 1.65, the mean of the meta-analysis by Stern and Smith (2003). These authors find that the distribution of values for this ratio is lognormal with median (=geometric mean) of 1.45, which implies a geometric standard deviation σ_G = exp[(2*Ln(mean/median))^0.5] = 1.66.

In terms of maternal blood the slope is 0.33 IQ points/($\mu g/L_{maternal\ blood}$) if one assumes a value of 1.65 for the ratio of concentrations in cord blood and maternal blood. To relate this to dietary intake, we note that according to UNEP (2002) the ratio of the steady state blood concentration y (in μg/L) and the average dietary methyl-Hg intake x (in μg/day/person) is in the range y/x = 0.3 to 0.8. Here we take as central estimate the relation y/x = 0.44, the mean value of the meta-analysis by Lipfert (1997) as cited by Sullivan et al (2003). It implies a ERF slope of

s_{ER} = 0.2*1.65*0.44 = 0.145 IQpoints/(µg/day) .

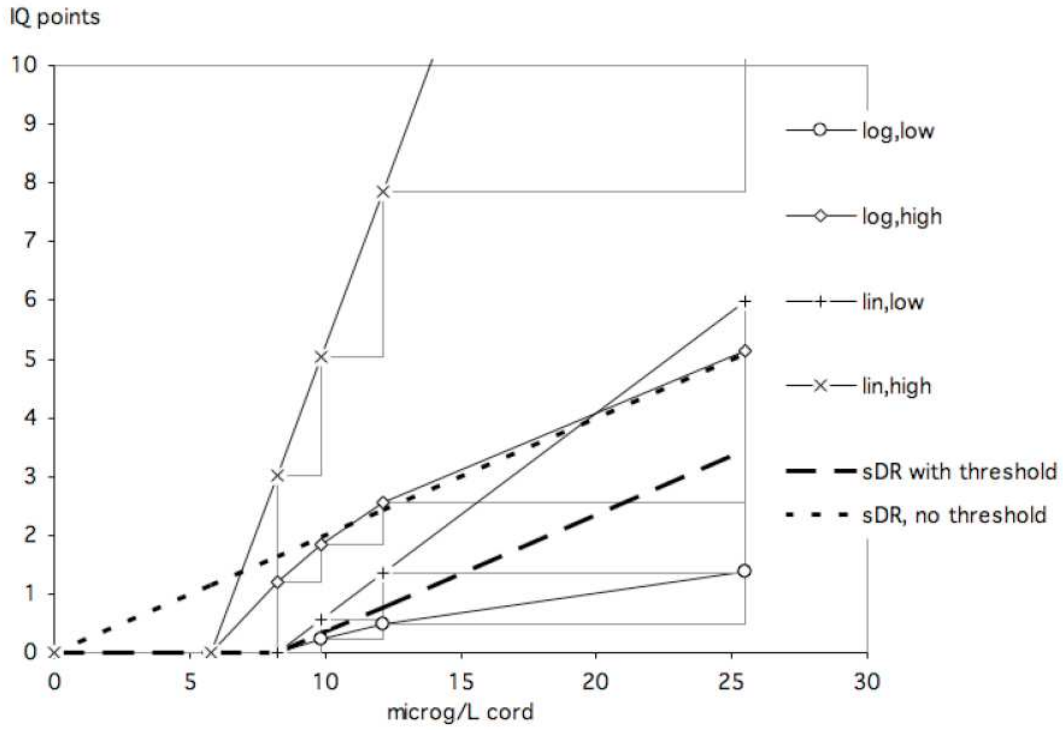


Figure 4.1 ERFs for IQ decrement due to Hg, in terms of µg per L cord blood. The lines with markers show the ERFs used by Trasande et al (2005). Corresponding to each of these four ERFs a thin line shows a stair step under the curve because that is how Trasande et al applied the ERFs to population segments with blood concentrations in the respective ranges. The thick dashed lines show the ERFs recommended here, with slope sER.

To apply the ERF with threshold we simply multiply the no-threshold result by the fraction f_{thresh} of the population that is above the threshold. EPA (http://www.epa.gov/iris) noted "*no evidence of a threshold arose for methylmercury-related neurotoxicity within the range of exposures…*". We find the possibility of a straight line without threshold not only plausible but probable. Hg is a neurotoxicant that damages the developing brain and reduces the IQ, just like Pb. Also, like Pb it is a substance that has only harmful effects, by contrast to other metals (for instance Cr and Se) that are toxic at high doses but of which the organism needs a certain minimum to survive. Furthermore, whereas in the past the ERF for IQ decrement due to Pb was believed to have a threshold, more recent studies have found that at the lowest doses the ERF for Pb is at least as high as the extrapolation of the high dose points, and quite possibly even higher (Lanphear et al 2005).

To apply the ERFs, it is necessary to consider the time window during which the brain is affected by Hg. The sensitivity of the brain to Hg is greatest during the early development of the body, but the precise time distribution of the damage is not known. Even though the ERFs of Trasande et al are stated in terms of cord blood and refer to the mother during pregnancy, they probably include also the effect of diet during early infancy because the IQ of the children was measured well after infancy. In effect they express the total impact on infants, starting with exposure of the mother before and during pregnancy.

Even though the detailed time distribution of the sensitivity to Hg is not known, it does not matter for the calculation of impacts, because the ERF of Trasande et al describes the total steady state impact on a population exposed to a specified steady state dose. Whereas the damage is incurred only during early development, it is assumed permanent and the entire population is affected. For these reasons we consider only the population average dose, not the dose to pregnant women or infants.

To see that the duration of the sensitive period f_{sens} does not matter, note that the fraction of the population between zero and f_{sens} years is very close to f_{sens} times the fraction between zero and one year, and the fraction between conception and birth is very close to 0.75 times the fraction between zero and one year, because the rates of still births and infant mortality are sufficiently small. The fraction between zero and one year is equal to the birth rate b, apart from a negligible correction due to infant mortality. If the sensitive period is only one year, the loss per μg/day of intake is $s_{DR} \times b$.

If the sensitive period is f_{sens} years, the affected cohort is essentially f_{sens} times as large but the damage rate is f_{sens} times smaller, so the loss is $(s_{DR}/f_{sens}) \times (f_{sens} \times b)$, the same result. f_{sens} drops out of the calculation. By the same token variations in food intake between individuals or over time do not matter because they are implicit in the ERF, having been determined by epidemiology as population averages.

4.2.7 Nickel

The effects of nickel in humans are reasonably well established for workers exposed to airborne nickel, although reported dose-response relationships are variable and appear to be

different for different nickel compounds. There is also a well established association between nickel and contact dermatitis; but there is no monetary valuation for this endpoint. There is very little information about the effects of community exposure to ingested or inhaled nickel apart from some information linking dietary exposure to dermal sensitization. The health endpoints associated with both ingestion and inhalation are presumed to be similar, with the exception of lung cancer which is expected to be primarily associated with inhalation. There is relatively little information about the relative efficiency of Ni following inhalation or ingestion. WHO (1991) cites limited information that suggests that about 75% of inhaled Ni is absorbed, about 25% of Ni ingested in water and about 1% of Ni in food. EPA IRIS indicates a URF of 2.4×10^{-4} per $\mu g/m^3$ for lung cancer due to inhalation.

Besides lung cancer Ni may cause neurotoxic effects such as lethargy and ataxia, neonatal mortality, kidney dysfunction, and worsening of eczema in sensitive subjects, but the ERFs are not sufficiently well established.

4.3 Quantifying an effect of benzene, PAHs and dioxins

4.3.1 Benzene

Benzene is recognized as a human carcinogen, for instance IARC (1995) classifies it as Category 1, the best established carcinogens. There are many occupational studies investigating exposure to benzene and development of cancer, especially leukaemia. However, a quantification of risks is complicated by uncertainties due to lack of quantitative data, short follow up at low exposure concentrations, and co-exposures to other potential carcinogens. Furthermore, there may be large individual variation in susceptibility or metabolism, the latter being relevant because the body breaks down benzene to metabolites which seem to be more toxic than the parent substance. There is no convincing evidence of chronic non-cancer effects at ambient concentrations.

As so often, different risk estimates have been derived, using different assumptions about the pattern of exposures, the shape of the CRF, and the extrapolation to low concentrations. In 1990 EPA gave a URF of 8×10^{-6} cancers/(pers·70yr·$\mu g/m^3$), not too different from the estimates of Crump (1994) who gives a range of 4.4 to 7.5×10^{-6} cancers/(pers·70yr·$\mu g/m^3$) for the URF of leukaemia. Currently the IRIS database of EPA shows a range of 2.2 to 7.8×10^{-6}

per μg/m^3. Here we recommend a URF of 4.0×10^{-6} per μg/m^3, with large uncertainty (about a factor of 2), as listed in Table xx.

4.3.2 Polycyclic Aromatic Hydrocarbons (PAHs) - Benzo[a]pyrene

These are ring compounds resulting from the incomplete combustion of organic material and which jointly share carbon atoms. They cover a wide range of substances including benzo[a]pyrene (BaP). The relationship between BaP and other PAHs differs for various types of emission but has been shown to be relatively similar in the ambient air of several towns and cities.

There is strong evidence, including from epidemiological studies (e.g. Redmond *et al*., 1972; Hurley *et al*., 1983; Armstrong *et al*., 1994), to suggest that certain components of PAHs, and specifically benzo[a]pyrene, are carcinogenic in humans; and that nitroaromatics as a group pose a hazard to health. In 1986 IARC and the US National Cancer Institute concluded that PAHs were a risk factor for lung cancer in humans. Benzo[a]pyrene specifically, rather than PAHs as a group, is labelled as a probable human carcinogen.

As these compounds form complex mixtures and are also absorbed onto particulates, it is difficult to quantify levels of human exposure and so is difficult to estimate risks reliably. Benzo[a]pyrene is the only PAH for which a suitable database is available, allowing quantitative risk assessment. The EPA unit risk factor of lung cancer for BaP is 1×10^{-7} per μg/m^3 (US EPA, 1990). Limitations in the use of benzo[a]pyrene as an indicator of PAH toxicity in air pollution are that some PAH is bound to particulates, and that some of the gaseous components are not included. WHO (1987) estimated a URF of 8.7×10^{-8} per μg/m^3; i.e. almost identical to that used by US EPA.

4.3.3 Dioxins and PCBs

Dioxins (more precisely polychlorinated dibenzo-*p*-dioxins) are a class of 75 individual compounds some of which are extremely toxic, acting as carcinogens and as endocrine disrupters. Closely related are the furans (more precisely polychlorinated dibenzofurans), a class of 135 different compounds, and the PCBs (polychlorinated biphenyls), a class of 209 compounds. They are “dioxin-like”, i.e. they have similar chemical structure, similar physical-chemical properties, and invoke a common battery of toxic responses. It is convenient to

characterize their relative toxicity in terms of the toxic equivalency factor (TEF), defined as the toxicity of a compound divided by that of the most toxic one, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). Using the TEF, the toxicity of a mixture of dioxins or dioxin-like substances can be expressed as toxic equivalent (TEQ) of the quantity of TCDD with the same toxicity. Usually the emissions, doses etc of dioxin mixtures are stated as TEQ. TEF data can be found in the dioxin reports of EPA (1994).

Dioxins are created in minute quantities as byproduct of certain processes where chlorine and organic matter can combine, for example the incineration of chlorinated plastics. Other sources of dioxins include the steel industry and the production of pesticides. However, thanks to stringent environmental regulations the emission of dioxins in Europe has been drastically reduced. However, because of their hydrophobic nature and resistance towards metabolism, these chemicals persist in the environment for many years. In particular they bioaccumulate in fatty tissues of animals and humans. Most of the dose comes from ingestion.

Dioxin is one of the most thoroughly studied of all of the pollutants. Several human epidemiological studies and numerous studies in experimental animals have been carried out. There can be acute as well as chronic effects. Dioxins cause changes in laboratory animals that may be associated with developmental and hormonal effects; however, the mechanism of carcinogenicity is unclear. To what extent such biochemical changes may result in adverse health effects in people and at what concentrations is not very well known. Among the acute effects is chloracne, a skin disease. However, the most troubling impacts are cancers and developmental changes, the latter because dioxins have certain similarities with hormones.

The hormonal effects of dioxins interfere with the endocrine system; however, there are still no ERFs are available for such effects, despite much research. The only quantifiable impact is cancer. Dioxins (2,3,7,8-TCDD and HxCDD) were said by EPA to be "the most potent carcinogen(s) evaluated by the EPA's Carcinogen Assessment Group". The slope factor is 1.0×106 cancers/(mg/(kgbody·day)) TEQ (EPA 2000).

PCBs had been used in the past as transformer oil, but that was outlawed and there are no new sources. But a remainder of PCB pollution is still lingering in the environment, especially in the sea. For PCBs http://www.epa.gov/iriswebp/iris/index.html states "Joint consideration of cancer studies and environmental processes leads to a conclusion that environmental PCB

mixtures are highly likely to pose a risk of cancer to humans“ and lists the following ERFs for cancer:

URF for exposure to air = 1×10-4 per µg/m3,

URF for drinking water = 1×10-5 per µg/m3 per mg/L,

and SF for ingestion = 4×10-2 per mg/kgbody/day.

They are an average over different congeners. The cancers found in epidemiological studies are hematologic cancer, cancer of the liver, gall bladder, gastrointestinal tract and biliary tract, and skin cancer. The mortality rate for such cancers may be around 50%.

These slope factors and URFs for dioxins and PCBs are probably an overestimate, and the real numbers may be 5 times smaller.

4.4 Quantifying an effect of radionuclides

In the ExternE 1995 series of reports an extensive analysis of the nuclear fuel cycle has been made (Dreicer and Tort, 1995). The human health effects due to the release of radionuclides were calculated starting from an aggregated population exposure (in man.Sv). Risk coefficients for cancer and hereditary effects were taken from the International Commission on Radiological Protection (ICRP) publication n°60. A linear dose-effect relationship was assumed, that is still valid. After a major review of the biological effects of low-dose ionizing radiation the United Nations Scientific Committee on the Effects of Atomic Radiation concludes that “an increase in the risk of tumour induction and of hereditary disease proportionate to the radiation dose is consistent with developing knowledge and that it remains, accordingly, the most scientifically defensible approximation of low-dose response. However, a strictly linear dose response should not be expected in all circumstances” (UNSCEAR 2000, annex G). Risk coefficients are expected to change. A new ICRP recommendation is nearly finalised, and is expected to be published in 2007. New information on the risks of radiation-induced cancer and hereditary effects has been used to change the risk coefficients, for the purpose of estimating the impact on a population level. The risk coefficient for hereditary effects has decreased with a factor of 6 for the whole population and with a factor of 8 for the subset of adults in the population. A summary of the new risk coefficients are given in Table 4.2

Table 4.2: Risk factors (in % per man.Sv) for cancer and hereditary effects (ICRP draft recommendations of 12/01/2007).

exposed population	cancer		Hereditary effects		Total	
	New recommend-dations	ICRP 60	New recommend-dations	ICRP 60	New recommend-dations	ICRP 60
Whole population	5.5	6.0	0.2	1.3	6.0	7.3
Adults	4.1	4.8	0.1	0.8	4.0	5.6

Note: the previous uncorrected cases per manSv (only adjusted for low doses) are 0.05 cases per manSv for fatal cancers, 0.12 cases per manSv for non-fatal cancers and 0.01 cases per manSv for hereditary defects. ICRP suggests to use an adjusted risk factor, that takes into account the life years lost (or the life quality loss). It then becomes (for the general adult public): 0.05 cases per manSv for fatal cancers, 0.01 cases per manSv for non-fatal cancers and 0.013 cases per manSv for hereditary defects. (See ICRP 60 (1991) tables 3 and 4).

In the new (draft) recommendations there is no longer a subset of non-fatal cancers risk coefficients available. The risk is based on cancer incidence, including fatal and non-fatal cancers. The risk of fatal cancers is roughly estimated to be 10% lower than the previous ICRP 60 recommendations, 4.4% for the whole population (compared to 5% in ICRP 60), and 3.6% for adults (compared to 4.0% in ICRP 60). Risks of non-cancer disease at low doses remain most uncertain and no specific judgement is possible. Finally ICRP is ambiguous about using these new values. In their recommendation, ICRP finds it of no practical use to lower their risk estimates, because of the uncertainties involved. For ExternE this means that an impact assessment based on the ICRP 60 recommendations is still valid, with the new recommendations as sensitivity analysis.

5 LIFE EXPECTANCY LOSS DUE TO AIR POLLUTION

5.1 Is total mortality a good policy indicator for $PM_{2.5}$?

In the ACS study the association between $PM_{2.5}$ and cardiopulmonary and lung cancer morality is stronger than for total mortality (Table 5.1). This finding seems to suggest that cardiopulmonary death and lung cancer are better indicators of the effect of particulate air pollution.

The number of people dying from cardiopulmonary disease or lung cancer is approximately 2 times smaller though in the US and in western Europe, compared to the total number of yearly deaths. This might result in different estimates of life expectancy loss in the general population, either by using the total mortality hazard, or the sum of cardiopulmonary and lung cancer hazards. We use the total mortality hazard from the ACS study to calculate the life expectancy loss due toe $PM_{2.5}$, for several reasons, explained below.

Table 5.1: Relative risks for different kinds of mortality for 10 µg/m³ $PM_{2.5}$ (Pope et al., 2002).

Mortality	**RR (95% CI)**
Total mortality	1.06 (1.02-1.11)
Cardiopulmonary mortality (ICD-9 401-440, 460-519)	1.09 (1.03-1.16)
Lung cancer mortality (ICD-9 162)	1.14 (1.04-1.23)
All other cause mortality	1.01 (0.95-1.06)

1. Reliable mortality statistics limit the use of cause-specific hazard calculations.

In a recent extensive analysis of the ACS data up to 1998 (Pope et al., 2004) respiratory mortality did not show a good statistical relation with $PM_{2.5}$. The researchers concluded that $PM_{2.5}$ causes inflammation and accelerates atherosclerosis, perhaps leading to ischemic heart disease, because there was confirmation that a strong association existed between $PM_{2.5}$ and ischemic heart disease (RR 1.18 (95%CI 1.14-1.23) for a 10 µg/m³ increase). Pope and colleagues also showed that PM2.5 alters the cardiac autonomic function, leading to dysrhythmias, heart failure, cardiac arrest, and this is also demonstrated with a RR of 1.13 (95% CI 1.05-1.21) for an increase of 10 µg/m³ PM2.5. But surprisingly the analysis did not

show significant associations between $PM_{2.5}$ and mortality due to COPD. A possible explanation according to the authors is that deaths of COPD patients are often misdiagnosed because they succumb from influenza or pneumonia. In fact disease of the respiratory system in general was not associated with $PM_{2.5}$. There was only borderline significance with deaths from pneumonia and influenza. This brings to attention that it might still be better to use all cause mortality risks for the chronic mortality effects of $PM_{2.5}$, to avoid the effects misclassification of the cause of mortality. LE loss due to the long-term effect of $PM_{2.5}$ on total mortality thus seems preferable for practical reasons because mortality statistics by underlying cause of death are less comparable and sometimes very uncertain or even lacking in different countries. Total mortality is easier to use, quite straightforward to monitor and hence more comparable across countries.

2. Total mortality captures the full health effect of $PM_{2.5}$.

Next to this practical reason for using total mortality as the endpoint, there are theoretical reasons as well. In his paper, Rabl (2006) argues that it is plausible that air pollution acts as a stressor on the human body and generally affects the reserve capacity of people. Those who have a lower reserve capacity are hence more susceptible to air pollution, and this is shown in the epidemiological studies. As a consequence susceptibility to air pollution is not only a factor for cardiovascular patients or for patients with severe respiratory disease. Also people suffering from other illnesses, like severe diabetes, and having a weak health and low reserve capacity, are potentially affected by air pollution. Intensive labour or sport activities might also lower the reserve capacity. This is for example demonstrated by a study showing higher effects of air pollution through ozone in children who are very active outdoors. McConnell and colleagues (2002) show that the incidence of new diagnoses of asthma is associated with heavy exercise in communities with high concentrations of ozone. Gauderman et al. (2004) show that lung function growth is significantly reduced in relation to air pollution and that larger deficits in lung function growth rate were observed in children who reported spending more time outdoors. Jerrett et al. (2005) find associations between $PM_{2.5}$ and unexpected causes of death like digestive causes.

On the basis of these findings, it is possible that a health impact assessment of $PM_{2.5}$, using only cardiovascular mortality, respiratory mortality or lung cancer as the endpoints, can underestimate the impact of air pollution. It might therefore be better to use total mortality and life expectancy loss as the 'true' indicators of $PM_{2.5}$ related health impacts. The hypothesis that

air pollution affects all susceptible people and not only those succumbing from cardiopulmonary effects, needs further investigation.

3. Life expectancy loss due to cause-specific hazards is comparable.

Nevertheless policy makers in Europe require robust indicators to evaluate their policies. Using life table methods we have compared the life expectancy loss in an English population for cause-specific mortality with the LE loss for total mortality. The resulting difference between the two approaches is a close to zero. This means that it doesn't matter whether life expectancy loss for total mortality or for cause specific mortality is calculated on the basis of the ACS coefficients. Given the 95% confidence interval for the hazard coefficients of the ACS study, results are comparable. Total mortality is thus a good indicator for a health impact assessment and for cost-benefit analysis.

		Life expectancy gain per 100,000 population
Cause	**Impact (reduction in hazard)**	**England/Wales**
All cause	6%	673.2
Cardiorespiratory	9%	477.0
Lung Cancer	14%	103.3
Cardiorespiratory & Lung Cancer	9%+14%	580.4
Starting population (million)		53.0

5.2 A harmonised approach for life expectancy loss due to acute and chronic exposures

There has been much debate about the significance of the mortality impacts (sometimes called "acute mortality") observed in time series (TS) studies, an issue often referred to as harvesting or mortality displacement (see e. g. Zeeger et al 1999, Schwartz 2000). The key question is whether the observed deaths have been advanced by only a few days or whether the loss of life expectancy (LE) is much larger. This issue is crucial for the monetary valuation and for policy implications.

For a new perspective on this issue and on the relation between TS studies, intervention studies and cohort studies, Rabl (2006) formulates the analysis directly in terms of LE change, after showing that such a formulation is mathematically equivalent to the conventional formulation in terms of mortality risk. An LE formulation offers several advantages: it automatically

accounts for the fact that everybody dies exactly once, regardless of pollution; it provides a unified framework for time series, intervention studies and cohort studies; and it yields directly a quantity of interest to policy makers.

The constraint of fixed total probability of death can be appreciated by comparing an accident that instantly kills individuals in normal health (the LE loss ΔL is equal to the entire remaining LE) with a mortality risk that reduces LE by a short amount of time ΔL. The time dependence of the mortality rates is different. Whereas for an accident the mortality rate changes only at the moment of the accident, for the risk with the short ΔL the mortality rate increases initially but then decreases (relative to a reference population without the risk) during the ensuing period ΔL because of the individuals who would have died then but whose deaths were advanced. The delayed decrease can be called "compensating change". Even though TS studies until now have not taken this constraint into account, it has not affected the results. For TS the compensating change becomes a more or less uniform background as a result of the fluctuations in concentration because there is a wide range of individual ΔL. In cohort studies the constraint is implicit in the study design, because they observe the net effect of chronic exposure. But the constraint is crucial for understanding the LE change in TS studies and in intervention studies.

A unified framework for TS and cohort studies has also been proposed recently by Burnett et al (2003) who show that both types of study measure essentially the same risk function. However, these authors do not take into account the time variation of the risk function due to the compensating change, i.e. that the increased mortality due to a pollution peak now implies a decreased mortality at a later time.

Rabl (2006) shows that the mortality fluctuations observed in TS studies are proportional to the instantaneous time derivative of the life expectancy. They are the result of exposures both in the recent and the distant past, but a strong correlation with the most recent exposure is observed since the fluctuations due to past exposures tend to average to zero. The acute LE loss due to a pollution peak can be calculated by integrating the mortality rate over the observation window of a TS study (typically 1 day) and results are presented for O_3 and PM_{10} (including studies that have extended the observation window to 60 days). In intervention studies the (approximately) constant difference between exposures before and after the intervention makes it possible to determine the LE change by integrating the change of the mortality rate over

time. Cohort studies measure a long term relative risk from which one can calculate (Brunekreef 1997, Rabl 1998, Leksell and Rabl 2001, Miller and Hurley 2003) the ultimate LE gain that can be achieved by a permanent reduction of air pollution; it is equal to the LE change at the end of a sufficiently long intervention study.

Rabl (2006) also addresses the determination of deaths that can be attributed to air pollution. By contrast to the acute LE loss due to a pollution peak, the corresponding total number of deaths (in the sense of all deaths that are advanced by the peak) cannot be measured by epidemiology. The customary multiplication of mortality rate and relative risk increase of TS studies yields only a lower bound. This also prevents the determination of the LE loss per air pollution death.

Unfortunately the available data are not sufficient to determine all quantities of interest, for example the relation between the results of TS, intervention and cohort studies, and the contribution of acute mortality to the total LE loss from chronic exposure. Since one needs models for the processes by which the body repairs air pollution damage, the remaining sections of the paper are somewhat speculative. Whereas the phenomenon of repair is well documented by studies of smoking cessation (USDHHS 1990, Doll et al 1994, Lightwood and Glantz 1997), less is known about repair of air pollution damage (for a review see Röösli et al 2005). In view of the available information it is plausible to assume that the LE change due to air pollution is proportional to the time integral of past concentrations weighted by exponential decay factors. Using model parameters suggested by the data, results are plotted for the evolution of mortality after an intervention. They indicate that the change in mortality rate is largest soon after the intervention. After a time long compared to the longest time constant of the repair processes the mortality rate returns to a level close to the one before the intervention (even though the LE gain is permanent), a consequence of the fact that everybody will die sooner or later.

With these models the results of TS, intervention and cohort studies are remarkably compatible with each other. The contribution of acute mortality to the total LE loss of chronic exposure is equal to the relative risk increase times the time constant of the repair processes that are significant immediately after a pollution peak.

5.3 Intervention studies

Intervention studies probably provide the best proof of the effect of air pollution on health. The ban of residential coal use in Dublin, Ireland, has resulted in a step change in air pollution, but also in a significant reduction of deaths in subsequent winters (Clancy et al., 2002). The reduction was significant in every season but more pronounced in winter. The reduction in mortality rate was largest for people younger than 60. Averaged over a period of 6 years after the coal ban, mortality rates decreased with 5.7% whereas black smoke was reduced with about 35.6 $\mu g/m^3$. The reduction in risk is larger than what would be expected from studies on the acute effects of mortality, indicating that a reduction in air pollution also influences long term risks. The biggest 'return' in public health is realised in the first years after the coal ban, indicating a direct and reversible effect of air pollution policies.

In Hong Kong a reduction of sulphur in fuels from 1 july 1990 resulted in a significant and persistent reduction in SO_2 (Hedley et al., 2002). PM_{10} and sulphates were reduced short after the measure, but increased again in the following years, possibly due to regional pollution from China. Still a remarkable absence of winter peak (respiratory) mortality in the year after the sulphur reduction was noted. This 'acute' effect was accompanied by a long term reduction in mortality rates. It is unclear whether the effect is caused by SO_2. It is suggested that emissions of metals like vanadium and nickel are probably reduced as well due to the change in fuels, and that these metals are perhaps causing the health effect (Lippmann et al., 2006). Finally an important study of Pope and colleagues in Utah, US, shows that during a 13 month strike in a steel mill, both morbidity in the community decreased and mortality rates dropped markedly (Pope et al., 1996). Analyses of the dust samples taken before, during and after the strike, and bronchiolar lavages of people who were exposed to these samples indicate that a larger fraction of metals when the factory is in operation is responsible for the inflammatory effects of the particles (Ghio and Devlin, 2001).

6 COMPONENT SPECIFIC TOXICITY - RELATIVE TOXICITY OF DIFFERENT KINDS OF PARTICLES

6.1 Introduction

Short overview of air pollution (especially particles) and health

Numerous epidemiological studies have shown associations between ambient particulate matter (PM), as measured at fixed-point monitors, and the risks of mortality and cardio-respiratory morbidity. The evidence is strongest for acute exposures, i.e. daily variations in particulate pollution, where effects have been found in numerous cities and regions worldwide, with different pollution mixtures and different climatic conditions; but there are also now numerous cohort studies linking annual average PM concentrations and risks of mortality. This very substantial epidemiological evidence is supported by plausible mechanisms of effect, based in the evidence of toxicological, and by studies of policy interventions.

Consequently it is now very widely accepted that the general ambient air pollution mixture, and PM in particular, cause adverse health effects in populations exposed to them.

However, particulate matter is in itself a mixture of different components, and it remains a critical issue whether or not (or to what extent) it is the chemical composition of particles, the mixture of air pollutants co-existing with particles, or only the physical properties of particles that are driving the health effects. There is for example a lack of evidence for the toxicity of sulphates and nitrates that make up a large fraction of the mass of particulate matter in ambient air. In particular there is a lack of epidemiological studies of nitrate aerosols because until recently this pollutant has not been monitored by air pollution monitoring stations. Indeed, most of the available epidemiological studies are based simply on the mass of PM without any distinction of the components or characteristics (acidity, solubility, …). Moreover particles in ambient air are generally accompanied by other pollutants, the more classical being NO_x, SO_2, ozone, but also a series of volatile organic compounds like benzene, aromatic compounds. Together, these considerations have implied that there is not a clear view of what aspects of ambient PM are principally responsible for the adverse health effects found in many epidemiological studies.

Quite generally it is difficult for epidemiologists to attribute a particular health impact to a particular pollutant, because populations are exposed to a mix of different pollutants that tend

to be highly correlated with each other. The conclusion that air pollution damages health is much more certain than the attribution of damage to a particular pollutant. For that reason some epidemiologists keep emphasizing that any individual pollutant is merely an indicator of pollution and that the attribution of an impact to a specific pollutant is very uncertain (ERPURS 1997). Informed opinion varies on this; others are less agnostic. For example, many leading American epidemiologists have tended to attribute the damage mostly to PM, although in recent years they have also recognized the possibility of a larger role of the other pollutants. A plausible view is that PM and ozone both have a causal relationship to mortality and morbidity; that the effects of these two pollutants are independent of one another; and that together they account for the greater part of the adverse health effects of the general pollution mixture found in cities worldwide.

In this chapter we build upon existing knowledge and on the approach first suggested by Rabl (1999, 2001), to incorporate alternative assumptions about the toxicity of particles in relation to co-pollutants in ambient air, and to evaluate the consequences of weighing components of PM differently. First the theoretical approach of Rabl (2005) is repeated, and then extended to include other pollutants or components. Reviews of the toxicity and epidemiology of particle constituents are briefly summarised in annex 3. Finally, we make a recommendation for NEEDS.

While this chapter describes a theoretical model that is practicable and able to address different components of PM or the mixture of ambient air pollution, the range of application of the model at this time is limited by two important factors, quite different in nature. One of these are the limitations in the modelling of particulate matter dispersion from emissions of energy sources (or any other source of air pollution). The second is the view of many individual air pollution researchers, and of established working groups e.g. of the World Health Organisation, that although the toxicity of different kinds of particles may well vary, per unit mass, it is not possible, based on current evidence, to quantify reliably any differences in toxicity. This, for example, was the view of the WHO experts in answers to the Follow-up Questions for CAFE (WHO, 2004), and of the peer reviewers of the CAFE CBA methodology. Consequently, in its quantitative estimates, CAFE CBA distinguished between PM_{10} and $PM_{2.5}$, but did not otherwise differentiate in toxicity between different components of PM.

Nevertheless, we consider that the model described here remains important as a framework for quantifying differential effects of different kinds of particles. And it is fit for purpose, (i) to evaluate alternative assumptions about the components and mixture that is being modelled with

air quality models like the EMEP model, and (ii) also to demonstrate the importance of including other fractions not modelled in EMEP, like metals, or organic components.

The equations presented here describe a possible way forward to model component specific toxicity, but at this stage it is over-specified, meaning that they contain too many terms relative to what we can estimate from scientific evidence or scientific consensus (i.e. the model is over-specified relative to the available evidence). The full version is thus not usable at this stage. But the framework, and the use of a limited version for illustrative purposes, provide a means of sharpening our thinking about the issue of component specific toxicity and quantification of source specific air pollution and health damages. It certainly highlights where we need additional information from scientific studies.

6.2 A model to evaluate the toxicity of PM and co-pollutants (PM, NOx, SO2, CO and O3)

Statement of the problem

Several health impact assessments estimate the health impacts of exposure to ambient levels of air pollution. That is sufficient for informing policy makers about the benefit of reducing the concentration values recommended as guidelines for ambient air quality. For such an assessment the results of epidemiological studies can be used without any hypotheses about the toxicity of different components of ambient PM because both the studies and the assessments are based directly on typical compositions of ambient PM, especially in cities where the majority of the population reside.

Unfortunately, current epidemiological and toxicological studies do not provide sufficient evidence to identify which air pollutant is responsible for which health impact. In such a situation it is natural to recommend sensitivity studies to evaluate different hypotheses. However, that approach is not a practical one for health impact assessment (HIA) programmes such as ExternE because it would lead to an excessive profusion of numbers that would have to be presented as final results of the study. A more manageable alternative is to chose a single most probable set of hypotheses, specified in terms of certain parameters, and estimate a central value and an uncertainty range for each of these parameters. Thus one can calculate a single central result, bracketed by a confidence interval obtained by Monte Carlo analysis. As starting point for this framework we assume that all the concentration-response functions (CRF) are linear without threshold.

By contrast with a health impact assessment of ambient air pollution, ExternE is a bottom-up methodology and starts from the source of the pollutants, calculating the damage attributable to each emitted pollutant (called primary pollutant). The need for this kind of information becomes obvious when one recognizes that, in order to actually attain lower ambient concentrations, specific regulations must be put in place to force the polluters to reduce their emissions. For the optimal formulation of such regulations one needs to compare the benefits of reducing the emission of a pollutant and the cost of such a reduction for all abatement technologies under consideration. In some cases tradeoffs must be made between the reductions of different pollutants; for example certain automotive technologies reduce the emission of PM while increasing the emission of NO_2 (e.g. the diesel particulate filter which increases the NO_2/NO ratio). Thus the optimal formulation of environmental policies requires more detailed information on the health effects of specific pollutants: one needs to know the incremental impact of an incremental kg of each pollutant that is emitted by particular source such as a power plant or a car. Both policy makers (representing the general public) and industries would benefit from a more disaggregated assessment of external costs, to balance against the scarce public or private resources to reduce the most damaging emissions as efficient as possible.

1st steps towards formulating a general model

To proceed it is convenient to write the incremental impact ΔI for a particular end point as a sum of the contributions of the individual pollutants i (each with CRF slope s_i and concentration increment Δc_i)

$$\Delta I = s_i \, \Delta c_i \quad ; \qquad (1)$$

the unit of I is cases per year per average person. The Δc_i are calculated for each location where there is human population, and the impacts are summed over all locations to obtain the total for the entire region that is affected.

How ExternE has quantified the effects (per µg/ m3) of particles from different sources

Although there is not yet a critical mass of air pollution researchers who are willing to quantify differently the effects (per $\mu g/m^3$) of particles from different sources, there have been some attempts in this direction. In particular, the ExternE reports of 1999 tried to differentiate between primary and secondary particles. There, the assumption was made that the toxicity of all sulphates is equal to that of the general $PM_{2.5}$ mixture and the toxicity of particulate nitrates

equal to that of PM_{10}. This distinction between sulphates and nitrates was based only on size, noting that nitrates need other particles to condense on, whereas sulphates self-nucleate and are therefore smaller on average. The ratio of CRF slopes $s_{PM10}/s_{PM2.5}$ was taken as 0.6, because this is a typical value of the ratio of concentrations of $PM_{2.5}$ and PM_{10}. We continue using this assumption.

$$s_{PM2.5}/s_{PM10} = 1/0.6 = 1.67 \quad . \qquad (2)$$

ExternE also tried to differentiate between different kinds of primary particles. The size, composition and toxicity of primary PM emitted by different sources can be quite different; for example, automotive PM is almost entirely organic or carbonaceous whereas PM from coal combustion contains in addition a sizable portion of minerals. Particles from internal combustion engines are all $PM_{2.5}$, whereas those from power plants are larger (mostly PM_{10}, with some particles being even larger than 10 µm). Since the available emissions data are simply stated in terms of PM mass, the best one can do is distinguish different typical PM compositions according to their source. In 1999 and 2001 ExternE treated power plant emissions as being equivalent in toxicity to PM_{10} and vehicle emissions as equivalent to $PM_{2.5}$. Sulphates were treated like $PM_{2.5}$ and nitrates like PM_{10}, based on their typical sizes. In terms of the above equations one can summarize the assumptions of ExternE 1998 and 2000 for the health impact ΔI due to a concentration increments Δc_i as

for ExternE (1998) (2)

$$\Delta I = s_{PM10} (\Delta c_{PMpower} + 1.67\, \Delta c_{PMtrans} + 1.67\, \Delta c_{sulf} + s_{PM10}\, \Delta c_{nitr}) + s_{O3}\, \Delta c_{O3}$$
$$+ s_{SO2}\, \Delta c_{SO2} + s_{CO}\, \Delta c_{CO} + \text{other}$$

where

$\Delta c_{PMpower}$ = concentration due to primary combustion PM from power plants,
$\Delta c_{PMtrans}$ = concentration due to primary combustion PM from transport, and
"other" = analogous terms for carcinogens such as benzene.

For the most recent methodological report from the ExternE series (ExternE, 2005) the assumptions about the toxicity of the different PM types have been changed after a careful review of the latest epidemiological and toxicological literature. Evidence has been accumulating to underline the high toxicity of combustion particles and especially of particles from internal combustion engines. For the secondary particles the evidence is less convincing.

In particular for nitrates there is still not much evidence for harmful effects, whereas for sulphates quite a few studies, including the very important cohort study of Pope et al (2002), do find associations, though it is unclear to what extent these associations are causal in sulphates *per se*, rather than sulphates acting as a marker of pollution with sulfur content. Therefore ExternE (2005) now treats

- nitrates as equivalent to 0.5 times the toxicity of PM_{10};
- sulphates as equivalent to PM_{10} (or 0.6 times $PM_{2.5}$)
- primary particles from power stations as equivalent to PM_{10};
- primary particles from vehicles as equivalent to 1.5 times the toxicity of $PM_{2.5}$ (1.5*1.67=2.5 times that of PM_{10}).

Effects of O_3 are considered independent of PM and added, whereas direct of effects of CO, SO_2 or NO_x are not taken into account. In equation form this can be written as

for ExternE (2005)

$$\Delta I = s_{PM10} (\Delta c_{PMpower} + 2.5\ \Delta c_{PMtrans} + \Delta c_{sulf} + 0.5\ \Delta c_{nitr}) + s_{O3}\ \Delta c_{O3}. \quad (4)$$

For example with s_{PM10} = 0.06% per $\mu g/m^3$ and s_{O3} = 0.03% per $\mu g/m^3$ for acute mortality in adults this would translate into an increased mortality risk ΔI of $0.06\%(\Delta c_{PMpower} + 2.5\ \Delta c_{PMtrans} + \Delta c_{sulf} + 0.5\ \Delta c_{nitr}) + 0.03\%\ \Delta c_{O3}$.

6.3 Extension of the model for component specific toxicity of PM.

To formulate a better approach for ExternE, we add modifying factors f_i in front of each term $s_i\ \Delta c_i$. The CRF slopes s_i are set equal to the associations reported by the respective epidemiological studies for a particular health end point. The values of the modifying factors f_i, together with estimates of their confidence intervals, could be chosen by expert judgment of epidemiologists and toxicologists in an attempt to obtain the most probable estimate of the health impact. Thus the f_i indicate with what weight the corresponding CRF should be counted. If a pollutant, for instance CO, is considered not to be causally linked to an end point, one sets the corresponding factor equal to zero, $f_{CO} = 0$ in this case. We also add terms for additional pollutants to allow for the possibility that they could have an effect, for instance NO_2 for which a few European studies report significant associations.

There is also the issue of HNO_3, a gaseous nitrate that has not been taken into account so far. Is there reason to believe that HNO_3, a strong oxidizing acid, is less harmful than the neutral but particulate NH_4HO_3? The oxidizing impact could be comparable to particulate nitrates even if the impact of acidity at the low concentrations in Europe and North America maybe negligible because the respiratory tract is sufficiently basic to neutralize it. One of the reasons for the lack of epidemiological evidence about nitrates is the difficulty of measuring ambient nitrate concentrations; few stations have monitored them routinely. Also, it is difficult to distinguish the effect of too many pollutants in an epidemiological study. The oxidizing potential of nitrates suggests that their impact might be similar to O_3; to account for that possibility we add a nitrate term to the ozone concentration, also with modifying factors f_i for each of the terms. For completeness we should try to indicate that metals and organic fractions of PM might play a role. This leads to a generalisation of equations 3 and 4:

$$\begin{aligned}\Delta I = \; & s_{PM10}\, f_{PMpower}\, \Delta c_{PMpower} + s_{PM2.5} f_{PMtrans}\, \Delta c_{PMtrans} + s_{sulf}\, f_{sulf}\, \Delta c_{sulf} \\ & + s_{snitr,P}\, f_{nitr,P}\, \Delta c_{nitr} + s_{HNO3,P}\, f_{HNO3,.P}\, \Delta c_{HNO3} \\ & + s_{snitr,O}\, f_{nitr,O}\, \Delta c_{nitr} + s_{HNO3,O}\, f_{HNO3,O}\, \Delta c_{HNO3} \\ & + s_{O3}\, f_{O3}\, \Delta c_{O3} + s_{SO2}\, f_{SO2}\, \Delta c_{SO2} + s_{NO2}\, f_{NO2}\, \Delta c_{NO2} + s_{CO}\, f_{CO}\, \Delta c_{CO} \\ & + s_{Metal}\, f_{metal}\, \Delta c_{metal} + s_{OM}\, f_{OM}\, \Delta c_{OM}.\end{aligned} \qquad (5)$$

The subscripts P and O for nitrates and HNO3 distinguish whether the impact is due to their particulate or their oxidizing nature.

Since there are few CRFs for sulphates, and none for nitrates, HNO3, metals and organic matter we can either leave them out of the model or assume proportionality with the CRFs for particulates and group the corresponding terms by setting their slopes (s_i) equal to that for PM_{10}.

$$s_{sulf} = s_{nitr} = s_{HNO3} = s_{PM10} = s_{Metal} = s_{OM} = s_{PM10} \quad . \qquad (6)$$

That option is preferred to be in line with general epidemiological results associating effects with PM_{10} mass concentrations. Moreover there is evidence from literature that findings for these components are correlated with findings for PM_{10} (e.g. Gauderman 2004, where acid vapour, including HNO_3, is correlated with PM).

$$s_{nitr,\,O} = s_{HNO3,O} = s_{O3} \qquad (7)$$

Assuming the same CRF slopes in Eqs.6 and 7 is no limitation because the f_i factors can be chosen to set the actual slopes to any desired value, if, as we assume here, the CRFs are linear without threshold.

Thus we can write Eq.5 in the form

$$\Delta I = s_{PM10} (f_{PMpower} \Delta c_{PMpower} + f_{PMtrans} \Delta c_{PMtrans} + f_{sulf} \Delta c_{sulf} + f_{nitr,P} \Delta c_{nitr} \quad (8)$$
$$+ f_{HNO3.P} \Delta c_{HNO3} + f_{OM} \Delta c_{OM} + f_{metal} \Delta c_{metal})$$
$$+ s_{O3} (f_{O3} \Delta c_{O3} + f_{nitr,O} \Delta c_{nitr} + f_{HNO3,O} \Delta c_{HNO3})$$
$$+ s_{SO2} f_{SO2} \Delta c_{SO2} + s_{NO2} f_{NO2} \Delta c_{NO2} + s_{CO} f_{CO} \Delta c_{CO} .$$

The f_i factors should be chosen by consensus of the experts. Unfortunately at the present time not enough of the experts are willing to commit themselves to making such a choice, or, more precisely, to making a choice other than the one implicit in the CAFE methodology.

In choosing the weighting factors f_i for PM one should note that there is a constraint to ensure consistency with the observed CRF, since the measured CRFs for PM correspond to the ambient PM_{10} composition in the cities where the studies have been carried out. The observed impact ΔI due to an ambient concentration increment $\Delta c_{PM10amb}$ is

$$\Delta I = s_{PM10} \Delta c_{PM10amb} \quad (9)$$

and by Eq.8 it is also equal to

$$\Delta I = s_{PM10} \Delta c_{PM10amb} (\%_{PMpower} f_{PMpower} + \%_{PMtrans} f_{PMtrans} + \%_{sulf} f_{sulf} + \%_{nitr} f_{nitr,P} \quad (10)$$
$$+ \%_{HNO3} f_{HNO3,P} + \%_{PMother} f_{PMother})$$

where % with subscripts indicates the fraction of $\Delta c_{PM10amb}$ that is of the type indicated by the subscript, and “other” refers to other PM constituents such as OM, metals and soil particles. Thus the expression in ( ) must be equal to unity. However, in practice this constraint is difficult to apply because it depends on the PM composition at each site. It seems more appropriate to treat the f_i factors as independent and consider deviations from this constraint as being part of the uncertainty of the CRF slopes.

Once we have established the CRF slopes s_i and the f_i factors, we can evaluate the impact for each location where (and if) the incremental concentrations of PM_{10} (or $PM_{2.5}$) ($\Delta c_{PMpower}$, Δc_{sulf}, Δc_{nitr} , Δc_{HNO3} , Δc_{metal} , Δc_{OM}), O3 (Δc_{O3}), SO_2 (Δc_{SO2}), NO2 (Δc_{NO2}) or CO (Δc_{CO}) are modelled.

Table 6.1: Choice of the modifying factors f_i (confidence intervals) for chronic mortality (examples).

	f_{PM10}	f_{sulf}	$f_{nitr,P}$	$f_{HNO3,P}$	f_{metal}	f_{OM}	f_{O3}	$f_{nitr,O}$	$f_{HNO3,O}$	f_{SO2}	f_{NO2}	f_{CO}
ExternE 1998	1	1.67	1	1	1	1	1	0	0	0	0	0
ExternE 2005	1	1	0.5	0.5	1	1	1	0	0	1	0	0
CAFÉ	1	1	1	1	1	1	1	0	0	0	0	0
…	…	…	…	…	…	…	…	…	…	…		
Expert n	0.5	0.1	0.05	0.1	0.1	0.15	0.5	0	0.5	0.6		
Expert n+1	…	…	…	…	…	…	…	…	…	…		
Average												

In annex 3 a concise literature overview of the limited information on component-specific toxicity is given. Since chronic mortality makes by far the largest contribution to the damage cost, it requires the greatest care. For the largest study (Pope et al 2002) the strongest association is with PM2.5. Significant associations were also found with sulphates and with SO2, but they should not be added with the entire coefficient because they were found in single-pollutant models rather than in a multi-pollutant model together with PM2.5. The principal evidence for direct SO2 effects comes from the Hong Kong intervention study (Hedley et al 2002) where the mortality was found to decrease significantly during the year following the interdiction of high sulphur fuel in Hong Kong and the resulting sharp decrease of SO2 concentrations (sulphates decreased only slightly because as secondary pollutant they are much more dependent on regional SO2 emissions which did not change). The long persistence of the mortality decrease, far longer than the few days during which acute mortality is seen in time series studies, indicates an appreciable effect on chronic mortality of the change in pollution mixture, characterised by the reduction in SO2.

Our recommendation in NEEDS is in line with the CAFÉ approach in Table 6.1. In core analyses we will use particle mass within size ranges, i.e. $PM_{2.5}$ or PM_{10}, and we will not differentiate further than that, quantitatively. In sensitivity analyses the impact of this choice

of weighting factors can be explored, through application of different weighting factors to different constituents of $PM_{2.5}$ and/or PM_{10}. Especially, for sulphate and nitrate particles we will treat these according to the general principle, above; i.e. their effects will be quantified insofar as they contribute to $PM_{2.5}$ and/or PM_{10} (in $\mu g/m^3$). Organic constituents are treated similarly, but it is possible that limitations on the air quality modelling implies some under-estimation of health effects. Overall this is a change from the most recent ExternE (2005) position. The position above is the position of CAFE CBA, WHO, US EPA, COMEAP…. We see a benefit in consistency with these other expert groups, unless we have a (very) strong basis for an alternative. As noted (e.g in annex, and in the ACS study (Pope et al., 2002)), there is some basis for an alternative but no critical mass of support for it – hence no differentiation in the core analyses. In addition, we think that a position which simultaneously played down the toxicity of sulphates and nitrates and did not quantify direct effects of SO_2 and/or of NO_2 would imply the unlikely situation that SO_2 and/or NO_2 are to be ignored.

The above all underpins the view that 'core' quantifications in NEEDS should be based on treating all components of $PM_{2.5}$ (and of PM_{10}) as having the same toxicity in mass terms, and so using the same core CRF. There remains a question of whether differential quantification is justified and useful, even for sensitivity analyses. It could be argued, based on the opinions above, that it is not. However, it is useful to have a differential sensitivity analysis, for two main reasons:

a. From the viewpoint of a particular application, it highlights in a very specific way that there is at least one alternative viewpoint on the relative toxicity of various components of $PM_{2.5}$ (and of PM_{10}). By expressing that alternative viewpoint quantitatively, we can see if the policy conclusions are sensitive to whether components of PM are quantified differentially (by whatever means we adopt), or not.

b. From the viewpoint of advancing the methodology as a whole, then we make progress by quantifying in the light of evidence, *but in advance of the evidence being compelling*. This then gives a starting-point for improvement, as new evidence emerges, or as the implications of current evidence get understood better.

Sensitivity analyses: what differential quantification should we use, and what status should it have?

The proposed differential quantification of ExternE (2005) might be somewhat extreme; i.e. the evidence, if and when it settles down into something where there is more consensus, may well settle into less extreme quantification than that given by ExternE (2005).

A possible intermediate position is to have:

- Primary particles at 1.3 times the toxicity of the $PM_{2.5}$ mixture;
- Secondary particles are at 0.7 times the toxicity of $PM_{2.5}$

We do not support a quantification that regards either nitrates or sulphates as inert, i.e. with zero toxicity. Other sensitivity analyses are possible but any use of a differential quantification needs to emphasise that this is for illustrative / exploratory purposes only; and that there is no consensus about the exact values chosen. Because there is such a strong swell of opinion against differential quantification, we propose that we do not focus strongly on the quantified results of the sensitivity analyses, but rather on whether the sensitivity analyses lead to the same policy conclusions as the core analyses.

7 TRANSFERABILITY

7.1 Introduction

When applying epidemiological concentration response functions (CRF) in a different context, the transferability of the original study results to the new situation is a matter that needs some attention. The issue of transferability applies to

1. CRFs, expressed as the relative risk, or the % change in health endpoint per unit ($\mu g.m^{-3}$) change in pollutant;
2. background rates of morbidity and mortality, expressed as number of events per year per unit population (e.g. per 1,000 or per 100,000 people); and
3. impact functions, which incorporate both CRFs (as % change) and background rates, giving estimates of the number of attributable events per year per $\mu g.m^{-3}$.

Here we focus on the transferability of CRFs. Transferability of background rates is perhaps more important but has been tackled in CAFE to tailor the impact assessment to the needs of the Commission for EU policies for the whole European Union. In CAFE estimating background rates in different locations across Europe was needed. Recent methodological work in CAFE, in particular for hospital admissions (both respiratory and cardiovascular), drawing on European studies like APHEIS; and for asthma, drawing on general respiratory health studies such as ISAAC (children) and ECRHS (adults) has made real advances on previous methodology reports for ExternE (e.g. 1995, 1998) where, for many health endpoints, it had been necessary to estimate background rates from specific studies conducted elsewhere (e.g. the studies that provided the CRFs), or from at most one or two locations in Europe. These advances in estimating background rates have been included in the proposed set of CRF (see table in paragraph 3.3, and annex 1).

Evaluating the importance of differences between locations – what really matters

Inevitably there are differences between locations and populations. Of primary interest here, however, is to make a judgement – based on evidence – about whether these differences really matter; i.e. do they exert a large, or only a small difference on the final risk and impact estimates? And if a large difference, what is the balance of evidence that factors affecting transferability may lead to over-estimation rather than under-estimation in the new locations?

And finally, if there is evidence of a systematic effect, is it possible to estimate a scaling factor which can be applied to the transferred estimate, and so to improve its accuracy?
The concern that is most often raised is that by transferring results from one location to another, the effects of air pollution in the target population may be over-estimated. It is important to consider that transferring CRFs (or background rates) between locations may also lead to under-estimation of effects – though an under-estimation is less severe than if CRFs had not been transferred at all, leaving the particular pollutant-endpoint combination unquantified and so in effect estimated as zero.

Special attention will be given to the study of Pope et al. (2002) of the chronic mortality effect due to $PM_{2.5}$, and whether application in Europe is justified. This is an important concern, that has been addressed by WHO and others in relation to e.g. the CAFE health impact assessment of $PM_{2.5}$ – important because the estimated effect on mortality of longer-term exposure to PM, characterised as $PM_{2.5}$, is clearly the single dominant quantifiable effect of air pollution on health. Factors that may affect transferability of CRFs, and more specific of the CRF for long-term mortality due to PM2.5 from the ACS study, according to the WHO (2000) or to the European Scientific Committee on Health and Environmental Risks (2005), are

1. the way of measuring or modelling the exposure to a mixture of air pollutants and, specifically, the variation in composition of pollutants like particles; and
2. the variation in population characteristics, including their time-activity patterns and their indoor exposure.

When selecting a set of CRF for use in ExternE or in European air quality policy studies (like in the cost benefit analysis for CAFE) these considerations have played a role. Here we are dealing with these considerations more explicitly, to make the use and applicability of CRF in other contexts more transparent.

Apart from these transferability issues, there is another important concept that has been questioned (for example by Gamble, 1998): the use of ambient concentrations as a proxy of exposure to PM_{10} or $PM_{2.5}$. Although not strictly a transferability issue (it is more an issue of epidemiological study design), it confuses a lot of people because of the fact that people spent more than 80% in indoor environments, and are not directly influenced by outdoor air pollution. It leads to the prejudice that the epidemiological studies are wrong and therefore impossible to use in health impact assessments. We are not zooming in on the issue of causality, which at the time of Gambles' critique was of most concern. The evidence in favour

of causality has been strengthened by medical research, sophisticated new epidemiological analyses (a.o. by Pope and colleagues (2004)), and most important by intervention studies worldwide (Clancy et al., 2002). A causal effect – in terms both of short term exposures (daily variations) and longer-term exposures – is now widely accepted with respect to the ambient air pollution mixture as a whole, and generally accepted with regard to ambient PM and ozone, in particular. Nevertheless, there are uncertainties and, to some extent, variations in interpretation, regarding the role of different constituent pollutants of the mixture as a whole, and of ambient particles in particular. These issues are addressed in the chapter 6, on alternative toxicological assumptions.

7.2 Transferability of study results of acute effects.

For time series results the simple way to treat this issue of transferability is to look at the similarities between results of time series studies on daily mortality and hospitalization world-wide, given the completely different background rates and population characteristics. The percent change in daily mortality per unit exposure remains remarkably invariant to changes in population, location and population mixtures. Time-series studies have been undertaken around the world -- including a large set of cities in Europe (in the APHEA project), over 90 cities in the US (The NMMAPS study), in South and Middle America (Santiago, Chile (Ostro et al., 1996), Mexico City (Borja-Aburto, 1998), Sao Paulo, Brazil (Saldiva et al., 1995)), Seoul, Delhi, Bangkok (Ostro et al., 1999), Sydney (Morgan et al., 1998), … Such cities span a wide range of activity patterns, temperature – air pollution co-variations, housing characteristics, and baseline health conditions. In the APHEA study (Katsouyanni, 2001) the central estimate of 0.6% increased daily mortality per 10 µg/m³ was the result of a meta-analysis of 29 city results. In the same study the effect modification of NO2 pollution, climate, age distribution and mortality rates was highlighted, and explained a large part of the heterogeneity in the different single city results. The risk therefore varies between approximately 0.2% and 0.8% per 10 µg/m³ PM_{10}. In the US recent analysis of the NMMAPS city results concluded that the daily mortality increased with about 0.21 to 0.27% per 10 µg/m³ PM_{10}. Summary estimates for studies in South-east Asia are 0.4 to 0.5% per 10 µg/m³ PM_{10} (HEI, 2004). These numbers compare sufficiently close in view of the uncertainties with the value of 0.6% per 10 µg/m³ PM_{10} that we have chosen. This observation of similarities between risk estimates around the world is a good argument in favour of the validity of using ambient PM_{10} concentrations as a measure of exposure tot outdoor air pollution, and of transferring results of epidemiological

studies of acute effects of air pollution to target areas, when no original information for this area is available, irrespective of where ambient monitors are located or of how measurements are performed. Furthermore, note that the effect of daily variations in PM on mortality, as estimated from time series studies, is higher in Europe than in the USA. This gives some reassurance that, when transferring to Europe from the USA an estimate of the effect of longer-term exposure to PM on mortality, we may be under-estimating, rather than over-estimating, the corresponding true effects in Europe.

7.3 Is the way we measure or model the exposure a transferability issue?

In general, exposure is approximated by the concentration of a certain pollutant. This concentration is either measured or modelled. When transferring the CRF (expressed as the % change per unit of exposure) from the US to a European situation, and applying a measured or modelled concentration can induce errors, but is strictly speaking no transferability issue. Measuring pollutants in Europe and applying CRF from the US, where other measurement techniques might be applied to study the CRF, will result in additional uncertainties. In ExternE (NEEDS) air dispersion modelling techniques are being used, and this might also induce additional uncertainties. The uncertainty of the absolute value of a pollutant concentration due to dispersion modelling has been dealt with in previous ExternE methodology books (see e.g. ExternE methodology 2005, p.253-255).

The composition of a pollutant like $PM_{2.5}$ differs from location to location. When specific components are responsible for the health effects of $PM_{2.5}$ it is crucial to have these components included in a health impact assessment in Europe, using a CRF from the US. However, despite a large research effort the last 10 years, there is still limited evidence that points to very specific components of PM as the driving factor of the health effects of PM. Therefore we have taken the position of quantifying components of PM_{10} or $PM_{2.5}$ according to their relative contribution to the mass of PM_{10}/$PM_{2.5}$ (see chapter 6). In the ACS study $PM_{2.5}$ was measured in very different metropolitan areas across the US, with varying $PM_{2.5}$ concentrations and composition. It is therefore unlikely that a different composition of $PM_{2.5}$ in Europe will affect the CRF derived from the ACS study. But it remains important to have as many components as possible included in the modelled $PM_{2.5}$ concentrations. The same considerations apply for air pollution mixture as a whole. In general we consider the modelling as a source of additional uncertainty, but not as a factor that limits the transferability of CRF from the US. There are also good arguments to consider the use of the ACS CRF as an underestimation of effects in Europe.

Modelled concentrations as the index of exposure

Recent European studies (in CAFE) use modelled $PM_{2.5}$ concentrations, like the RAINS integrated assessment model that uses results from the EMEP air quality model. Amann et al. (2004) discuss some of the shortcomings when using RAINS, especially when applying a risk assessment for chronic exposure to $PM_{2.5}$. Composition of PM has not been taken into account, and the model results are limited to anthropogenic sources and precursor emissions of $PM_{2.5}$.

(This is consistent with the recommendations of WHO, also used in CAFE, that effects of $PM_{2.5}$ be estimated without threshold, but for the anthropogenic fraction of $PM_{2.5}$ only.) The model then calculates the dispersion and concentration field on a regional scale (grid size 50x50 km²) of primary $PM_{2.5}$ particles, and of secondary inorganic aerosols (SIA), mainly consisting of (ammonium-)nitrates and sulphates.

The most important shortcoming is probably the absence of a modelled secondary organic fraction. This is relevant to transferability to Europe of the CRF from Pope et al. (2002), because the measures of $PM_{2.5}$ underlying the Pope et al. CRFs do include the secondary organic matter (OM). Although there is uncertainty, debate and ongoing research into exactly which constituents of $PM_{2.5}$ are most responsible for the adverse mortality effects of $PM_{2.5}$, there also is some pattern to the available evidence. Thus WHO (2004 - follow-up answers for CAFE) concluded from the available evidence that primary combustion-derived $PM_{2.5}$ particles have a higher toxic potential. These particles are often rich in transition metals and organic compounds, and also have a relatively high surface area. It is unlikely that the secondary OM (which are not included in the EMEP model) are the single contribution to the associations with $PM_{2.5}$ detected in the ACS study (Pope et al., 2002), given the potential role of some of the metals that are adsorbed onto particles, the potential important role of elemental or black carbon nuclei, and the role of physical properties of particulate matter, like particle size, and surface reactivity. However if the toxicity of OM is comparable to that of primary combustion-derived particles, then an impact assessment using concentrations modelled by EMEP will underestimate the effect of $PM_{2.5}$ on a mass concentration basis.

It is possible to conjecture the implications under different scenarios concerning the effects of secondary organic particles. One possibility is that, per µg/m³, the various identifiable components of $PM_{2.5}$ all have similar effects. Though perhaps unrealistic, this is the basis of current quantification, as recommended by WHO and as applied in CAFE CBA. Assume also that the coefficient for $PM_{2.5}$ as a whole, as estimated e.g. by Pope et al. (2002), is correct. Consider finally a policy which reduces the concentrations of various kinds of $PM_{2.5}$. Then, the estimated overall benefits of reduction will be based only on the $PM_{2.5}$ modelled by EMEP, because the benefits of reducing the secondary inorganic fraction will have been omitted; i.e. the overall benefits will have been under-estimated. Under these assumptions, the size of the under-estimation is proportional to the reductions in annual average concentration (in µg/m³ $PM_{2.5}$) of secondary OM.

Note that this under-estimation is not *per se* an issue of transferability. Rather, it is a consequence of a mismatch between modelled PM concentrations and the measured values used in the underlying epidemiological studies.

Extrapolation beyond the range of the Pope et al. study is probably not an important issue given the linearity between $PM_{2.5}$ and survival probability across the range of annual average concentrations experienced by metropolitan areas in the ACS study. RAINS applies the results of the ACS study up to levels of 80 µg/m³ $PM_{2.5}$ annually, whereas the original study $PM_{2.5}$ values reported between 5 and 33.5 µg/m³. It is plausible, however, e.g. by analogy with time series studies, that the effects of ambient PM reduce, per µg/m³ $PM_{2.5}$, at high concentrations. Application of the US ACS coefficient throughout a range up to 80 µg/m³ $PM_{2.5}$ annually may therefore imply some *over*- estimation of effects in those regions in Europe which currently experience highest annual average concentrations. As a proportion of the overall population in Europe, these regions are small, and the impact of associated possible over-estimation of effects is therefore small also.

The ACS study is US-wide and so it includes results from regions with $PM_{2.5}$ from quite different sources. The key ACS papers do not try to give different coefficients for the mortality effects of $PM_{2.5}$ in different regions of the USA. These facts together strengthen the case for extrapolating the ACS results to Europe. One systematic difference in $PM_{2.5}$ pollution between US and Europe is the greater contribution in Europe from primary combustion diesel particles from traffic in cities. These are generally thought to be relatively toxic, per µg/m³. This implies that the differences in composition between the US and Europe, together with using the ACS study CRF in Europe, might lead to under-estimation of mortality effects in Europe. In addition, time series studies in Europe from APHEA2 have identified that the effects of ambient PM (on mortality) vary according to background level of NO_2, and specifically, that the estimated effects of PM are higher when background NO_2 is higher. NO_2 is often used as an indicator of pollution from mobile sources (traffic) and is correlated with particle number. The finding on PM and NO_2 may therefore reflect a relatively greater toxicity, per µg/m³, of particles from traffic sources (including diesel particles). This supports the view that compositional differences between $PM_{2.5}$ in the US and Europe might result in an underestimation of effects in Europe.

7.4 Is a difference in indoor exposure and time-activity patterns of a population in Europe compared to the US a factor that limits transferability of CRF?

Is the indoor-outdoor relationship for $PM_{2.5}$ and PM_{10} comparable in the US and in the EU? We can have greater confidence in the transferability to Europe of the information (coefficients) from epidemiological studies in the US if either (i) the penetration indoors of $PM_{2.5}$ from outdoor sources is not markedly different in the EU and the US (because ambient concentrations are then a good proxy for the assessment of personal exposure and health effects); or (ii) the estimated coefficient linking ambient PM and mortality does not vary markedly between regions with different outdoor-indoor penetration ratios.

Substantial research has been carried out on personal exposures to particulate matter and the contribution of particles from outdoor sources to total personal exposure, an area of investigation corresponding to Topic 1 of the US National Research Council's Committee. Studies have been carried out in the United States and in Europe that have involved personal monitoring of various population groups of interest in order to characterize the contribution of outdoor particles to personal exposure, and particularly to variation in personal exposure over time. This research was initiated, in part, to better understand the implications of using ambient monitors for population-level studies of particles and health, and also to consider exposure-based control strategies. The monitored populations have included children, healthy adults, and adults in groups within the population considered to be at risk for health effect of air pollution, including persons with chronic obstructive coronary heart disease and pulmonary heart disease. These studies have documented that particles in outdoor air penetrate indoors and make substantial contributions to day-to-day variation in total personal exposure, a critical finding with regard to interpreting the time-series studies. This work has not yet addressed personal exposures to particles in relationship to specific characteristics of the particles, based on size or chemistry; most have involved monitoring for PM2.5. An overview of several studies is given in annex 2.

In conclusion: ambient concentrations of $PM_{2.5}$ can serve as the adequate index of exposure in epidemiological time-series studies, intra-personal differences being explained in the original exposure studies through the indoor penetration of $PM_{2.5}$ and the indoor sources of $PM_{2.5}$.

7.5 Is a difference in population characteristics important when transferring CRF?

Is there a difference in socio-economic factors? Is the age structure, the prevalence of susceptible groups and the background incidence rate of disease and mortality comparable. And again, is the difference important enough to refrain from using the CRF, or can we incorporate this uncertainty in the impact assessment result? Detailed analyses of the ACS study have shown that the relative risks of mortality from long-term exposure to ambient PM2.5 are robust to a range of population characteristics. For example, the estimated relative risks of ambient PM per $\mu g/m^3$ PM2.5 do not vary with gender, or with age, or with smoking habit, though of course the absolute risks of mortality do vary with all three of these factors. Educational attainment is the one population characteristic which has been clearly shown, in the ACS study, to modify the relative risks of PM and mortality, with higher risks among people with lower educational attainment. It is understood that this may well not be an effect of education per se, but rather that educational status is here acting as a surrogate measure for socio-economic factors more generally.

This result has two implications regarding transferability. First, the group of participants in the ACS study is slightly higher educated than the average population, and this might have effected the risk estimated in the sense that the real risk across the US population as a whole might even be higher than estimated in the ACS study (Krewski et al., 2000). The US EPA however does not consider this a serious limitation to the use of the risk estimate of ACS study in air quality standard policy making for the whole of the US population, though it is useful to note that, once again, this issue suggests that use of the ACS study under-estimates the true effect. Secondly, it is useful to check whether differences in population characteristics between Europe and the US are important enough to take into account when assessing the chronic effect of PM on mortality. We have no direct comparisons between socio-economic factors (with educational status as a surrogate) between US and Europe but it seems reasonable to conclude that the population represented in the ACS study is relatively well off, compared with the population of Europe, on average, and so use of the ACS study in Europe might imply some under-estimation of mortality effects associated with socio-economic differences implied by differences in educational status. The ACS study is adjusted for a multitude of (44) potential individual confounders, which in practice have little or no effect on the estimated coefficient linking longer-term exposure to PM and mortality. This gives reassurance in transferring the ACS coefficient for use in Europe. It may further be a reassurance that overall mortality rates

and most important causes of death are quite similar, in the US and Europe, though within the ACS study – as noted above – the estimated effects of PM2.5 on mortality do not vary clearly or systematically with age, gender or smoking habits, whereas of course background rates of mortality vary markedly by these factors.

Environmental epidemiological studies are confronted with the aspect of socio-economic status (SES) as an important confounder of effects of air pollution. Social or material deprivation is a known driver of poor health, poor diet and higher smoking prevalence. The latter two factors were taken into account in the ACS study (Krewski et al., 2000, Pope et al., 2002). SES however remained an important confounder after the reanalysis of the original data. Possible explanations according to Jerrett (2004) are:

1. People of higher SES are more mobile, therefore the observed correlation of their risk with pollution is reduced, being averaged over sites with different exposures. It is shown in the reanalysis that the risk is not changed when filtering out the mobile subjects.
2. Contextual deprivation occurs in cities where it seems that the neighbourhood itself where people live determines their health status. The reanalysis and the follow up study did not find such an effect, even after including contextual (ecologic) variables, and assumptions with regards to spatial clustering.
3. If it is occupational exposure that causes the increase in risk, than this would also true for European populations. This kind of residual confounding is however a more general concern and needs better adjustment. It is not expected that this confounding effect will affect the risk estimates significantly, nor does it prohibit transferring the coefficients of the ACS study to Europe.

A comparison of European time-series studies in the APHEA2 study show a pronounced effect modification due to difference in age structure of the population in different cities in Europe, due to climate difference and due to difference in average NO2 concentrations (Katsouyanni, 2001). The latter leads to low estimates (0.19% mortality increase per 10µg/m³) in 'low NO2' cities or high estimates (0.8% increase per 10 µg/m³) in 'high NO2' cities of the acute mortality risk, compared to the average risk of 0.6 % per 10 µg/m³. It's not really an issue of transferability but it would be useful to incorporate effect modification where possible as a sensitivity analysis of the average results. This is not done because we don not use the acute mortality effect estimate, due to overlap with the chronic effect estimate. At this moment we also lack information on effect modification for other (morbidity) endpoints.

7.6 The use of ambient concentrations as the index of exposure in cohort studies

The discussion and conclusions for time-series studies are only partly applicable to cohort studies that use long-term averages and spatial exposure contrasts to derive relative mortality risks. When using the results of the prospective cohort study of Pope et al. (2002) in a health impact assessment in Europe, the same transferability requirements apply as discussed in the introduction. The similarities between populations and pollution mix prevail here also. The appropriate use of ambient yearly averaged concentrations of $PM_{2.5}$ is discussed here. In the case of cross-sectional comparisons between cities, we need some kind of correlation between the spatial average yearly concentrations of $PM_{2.5}$ within a city with the long-term average personal exposure to ambient $PM_{2.5}$. Two separate issues are distinguished. First the variability of exposure to $PM_{2.5}$ within a city will, when captured by choosing other exposure metrics, increase the relative risk. Second the same exposure measurement error (the variability of $PM_{2.5}$ exposure of individual study subjects) will not affect the estimate of mortality risk between cities, because of the large number of cities. Intervention studies provide an easy argument in favour of the use of ambient concentration measurements as a proxy for long term effects. The intervention study in Dublin (Clancy et al., 2002; see paragraph 5.3 for more information on this study) is a good example of using ambient concentration as the proxy for exposure. It is clear that the proxy (ambient concentration) is associated with the personal exposure, and that using ambient concentrations is a suitable way to assess health impacts or benefits. A similar study in Hong Kong shows the benefit of reducing sulphur in fuels. Here too a long term effect is observed, that is most strongly associated with ambient SO_2 reductions (Hedley et al., 2002). Long term changes in health in east Germany are also considered to be associated with ambient air quality improvements (Heinrich et al., 2000). Also the SCARPOL study in Switzerland is informative of the use of ambient concentrations (Bayer-Oglesby et al., 2005). In a follow up study the risk of air pollution is reduced due to improved air quality, demonstrating again that ambient concentrations, measured at fixed sites, are a good exposure metric to be used in health impact assessments.

The variability within cities, what does it do to effect estimates for a city?

There is a possibility that variability between measurement sites within a metropolitan area is larger than the variability between cities, and that this will lead to underestimation of the real risk in cohort studies. Newer analyses using intra-urban interpolated concentration maps demonstrate this. In brief the reasoning behind this is:

1. Spatial uniformity is not always true within a city. The review by Wilson and colleagues (2005) gives a methodology to make a thorough study and good classification in this respect.
2. Pope et al. (1995, and 2002) and the reanalysis (Krewski et al., 2000) use central site ambient monitoring stations as the exposure metric. Probably some of these metropolitan areas in the ACS study have a uniform monitoring system, and some have a heterogeneous set of measurements, showing higher variability of $PM_{2.5}$ within a city than between cities.
3. This leads in the case of heterogeneous exposure situations to misclassification of exposure, where people in the "high polluted metropolitan areas" actually live in clean neighbourhoods and vice versa, and this is not picked up by the analysis of the ACS study (Pope et al. 1995 and Krewski et al., 2000), nor in the follow-up (Pope et al., 2002).
4. Exposure classification because of this intra-urban variability of $PM_{2.5}$ leads to underestimation of the risk. Therefore the new spatial analysis of air pollution and mortality in Los Angeles, taking into account this intra-urban variability, gives higher risk estimates (Jerrett et al., 2005). The potential of exposure misclassification is reduced.

8 UNCERTAINTY AND SENSITIVITY

8.1 General Remarks

The uncertainties of environmental damages are far too large for the usual error analysis of physics and engineering (using only the first term in a Taylor expansion). Rigorous systematic assessment of the uncertainties is difficult and few studies have attempted it. Most merely indicate an upper and a lower value, but based on the range of just one input parameter or by simply combining the upper and lower bounds of several inputs, without taking into account the combination of uncertainties (e.g. of atmospheric dispersion, dose-response function and monetary valuation). Many damage assessments involve so many different inputs that an analytical solution was usually not considered, and of the uncertainty analyses that have been done, almost all use Monte Carlo techniques and numerical calculations (see e.g. Morgan & Henrion 1990). The Monte Carlo approach is powerful, capable of treating any problem, but it is computationally intensive and the result is "black box": it is difficult to see how important each of the component uncertainties is or how the result would change if a component uncertainty changes – especially for a reader who does not have access to the details of the calculations.

As a simple and transparent alternative Rabl & Spadaro (1999) have developed an analytic approach based on lognormal distributions (for an update see Spadaro & Rabl 2006). The justification lies in the observation that the calculation of damage costs involves essentially a product of factors, and that the resulting uncertainty of the product is approximately lognormal for most damage costs of pollution. Thus it suffices to specify geometric mean μ_g and geometric standard deviation σ_g, or equivalently, multiplicative confidence intervals about the geometric mean (which is usually close to the median for damage costs). Compared to a Monte Carlo analysis, this approach yields typical answers that are easy to apply and communicate; the calculation is simple enough to allow the reader to modify the assumptions and see the consequences.

Whatever the method, an assessment of the uncertainties of damage costs must begin with a detailed examination of the uncertainties of each of the inputs to the impact pathway analysis,

estimating standard deviation and shape of the probability distribution of their uncertainties. This involves expert judgment with its unavoidably subjective aspects. These component uncertainties are then combined to obtain the total uncertainty of the damage cost. Calculating the uncertainty of ERFs by themselves is not interesting: one needs to evaluate the entire chain of an impact pathway analysis. The discussion in this chapter considers only the contribution of the ERFs; for complete results the reader is referred to the report by Spadaro & Rabl (2006). Since this method is based on lognormal distributions, one needs to specify the component uncertainties in terms of geometric standard deviations σ_g.

8.2 Exposure-Response Functions

The uncertainty of dose-response functions varies widely from case to case. Best established are the ones for health impacts from radionuclides, the ERFs for certain health impacts from the classical pollutants (PM_{10}, SO_2, NO_2 and O_3), and the ones for impacts of SO_2, NO_2 and O_3 on certain crops whose economic importance has prompted laboratory studies.

The confidence intervals of ERFs for health impacts are usually reported for 95% probability, and they are approximately symmetric (of the form $\mu \pm 2\sigma$) around the mean μ. The underlying probability distributions (implicit in the regression software used in the respective studies) are usually not lognormal, hence it is necessary to estimate the corresponding geometric standard deviations σ_g.

If one knows the probability distribution of the residues in the respective studies, one could calculate the geometric standard deviation exactly from its definition. If one does not, but the reported confidence intervals are symmetric, it is reasonable to assume a Gaussian distribution. Strictly speaking the resulting σ_g is complex because the Gaussian is nonzero at negative values. However, negative values are not plausible on physical grounds (for health impacts of air pollutants a beneficial effect is not plausible), and the distribution should be cut off at zero. Furthermore, if one uses only ERFs that are statistically significant at the 95% level, the contribution of the negative values represents at most 2.5% of the normalization integral of the Gaussian, and the effect on the resulting σ_g would be negligible.

A much simpler alternative is the following approximation. Suppose that $\mu \pm \sigma$ corresponds to a 68% confidence interval, as for a Gaussian distribution. Then one fits a corresponding lognormal distribution such that its 68% confidence interval equals $[\mu-\sigma, \mu+\sigma]$, which yields σ_g as

$$\sigma_g = \sqrt{\frac{\mu+\sigma}{\mu-\sigma}} \ .$$

We have evaluated this equation for all the ERFs of ExternE for NO_x, SO_2, PM and O_3; typically σ_g is in the range 1.2 to 1.8. Specifically for chronic mortality Table 2 of Pope et al (2002) indicates, for the average exposure during the observation period, a relative risk RR given by RR – 1 = 0.06 with 95% confidence interval [0.02, 0.11]. With μ = 0.06 and σ = 0.023 we find σ_g = 1.48. In the following we will take 1.5 as a typical value.

For chronic mortality one also needs to determine the relation between the YOLL (years of life lost) and the change in the age-specific mortality rate that has been reported by studies of chronic mortality. Leksell & Rabl (2001) have examined the uncertainties of this calculation; their results suggest a σ_g of 1.3 for the calculation of the YOLL, given the relative risk.

There is, however, another type of uncertainty due to the difference between the PM in ambient air on which epidemiology is based and the primary and secondary PM in the damage calculations. Ambient PM is a mix of primary PM from combustion and secondary PM, especially nitrates (due to NO_x emissions) and sulfates (due to SO_2 emissions). For the damage calculations one needs assumptions about the relative toxicity of the different components of ambient PM. The uncertainty of these assumptions is difficult to estimate, see e.g. a recent workshop on this problem (COST 2006). To deal with this issue we introduce a factor for the respective toxicities of primary particles, nitrates and sulfates relative to ambient PM, and we assume a σ_g for these toxicities (our choices are a subjective judgment based on extensive discussions with epidemiologists and toxicologists). There could also be important direct impacts of NO_2 and SO_2 but currently the dominant thinking among epidemiologists is that they are negligible compared to those of PM and O_3 (see e.g. WHO 2003).

To sum up, we assume

σ_g = 1.5 (range 1.2 to 1.8) for the morbidity ERFs due to ambient PM

σ_g = 1.5 for the mortality risk (RR-1) due to ambient PM

σ_g = 1.3 for the calculation of the YOLL for a given mortality risk

σ_g = 1.8 for chronic bronchitis due to ambient PM

σ_g = 1.5 for the toxicity of primary particles relative to ambient PM

σ_g = 2 for the toxicity of nitrates and sulfates relative to ambient PM.

These elements enter the calculation in multiplicative fashion. Because of the quadratic combination of uncertainties, terms that make only a small contribution to the total damage cost can be neglected. In particular, it is sufficient to consider only mortality and chronic bronchitis because they make by far the dominant contribution.

9 CASE STUDY: EXPOSURE TO INDOOR WOOD STOVE COMBUSTION SOURCES

9.1 Introduction

The fact that people spent more than 80% of their time indoors keeps raising questions about the relevancy of studying outdoor air pollution only. Does indoor exposure confound with outdoor exposure? How does indoor exposure correlate with exposure to ambient concentrations or with personal exposure? Much attention to this question has been given in recent years, especially in the US (by the US-EPA in its PM-research programme, http://www.epa.gov/pmresearch/, and by the Health Effects Institute) and findings from these new studies and reviews have been highlighted in chapter 7, on the use of ambient concentrations as a surrogate for exposure. Apart from this there are numerous sources of indoor air pollution, that interact or act independently from the outdoor air pollution. In the context of NEEDS a case study is developed to illustrate the relative importance of energy-related sources of indoor air pollution. Wood stove[2] combustion is selected as a typical case study, for which the exposure to particles indoors is elaborated in terms of exposure. It is clear that indoor air pollution is far more complex, involving many sources, among which the most important are cigarettes, consumer products and building materials, and involving a wide spectrum of pollutants. Pollutants like VOCs, formaldehydes and other aldehydes, NO2, CO, benzene, PAHs, toluene, xylenes are among the most important. A limitation to particles from woodstoves is thus automatically an underestimation of the full indoor exposure to air pollution.

Next to identifying sources, pollutants and effects from energy related sources indoor, there is the question of whether these sources and their pollution constitute an externality. An external cost is a cost not included in the market price of a good or a service being produced, i.e. a cost not borne by those who enjoy the benefits of that good or service. Not only does producing something sometimes involve an external cost, but consumption can also have this kind of cost. Now, one could argue that for occupants of residential buildings the burden of using heating appliances or of cooking is born by those who benefit from heating and cooking at the same time. Health costs due to the voluntary decision to use a wood stove for example are paid by who's consuming the benefit of that wood stove. One could however distinguish costs born by

[2] the term 'woodstove' here refers to both indoor (closed) window woodstoves and indoor open fireplaces

society. In the case of health costs, part of these are paid by health insurance systems. Systems that are financed by member contributions or by the government (taxes). It is defendable to assume that the impact on children's health due to indoor cooking or heating is an external cost, since they do not have part in the decision process. Moreover it is in general even for adults a decision that is not based on complete information, nor on a rational balance of costs and benefits. The possible impact on health is not used to inform the consumer and is thus not internalized in the cost of using e.g. a wood stove. Most people are ignorant about the possible effects of indoor air pollution due to the appliances they have bought, and even if they understand these effects, there is often little or nothing they can do about them if they need the appliance in question because they cannot modify the appliance.

In the following paragraphs we apply a modelling approach, and scenario analysis to overcome the difficulty of finding adequate and representative data. The case study is illustrative of the importance of indoor PM sources, but generalisation remains difficult. Woodstove inventories in EU Member States are scarce and vary according to the data source used (e.g. there is a difference between WHO estimates and estimates based on the EU energy statistics report 2006, see annex 5).

9.2 Modelling indoor PM concentrations

The most important indoor source of PM is environmental tobacco smoke (ETS); also cooking, wood smoke and other combustion source are known to contribute to indoor PM (Jones, 1999). Although the focus of the current study is on woodstoves, the role of ETS, unflued gas appliances and cooking on indoor generated PM will also be included in the indoor model in order to put the magnitude of woodstove related PM in perspective of other indoor sources. When modelling the exposure to pollutants from combustion sources we can follow two approaches. We can review more literature to find typical concentrations that can be allocated to combustion sources. We then have a simple model combining exposure with time spent in those concentrations. With the use of available concentration-response functions, we can estimate the health effect due to typical concentrations in residences stemming from as specific class of combustion source. The second approach is using a mathematical steady state description of the different contributions to exposure indoors. This approach is not new, originating from the PTEAM study in the US (Özkanyak et al., 1996), for particles but also applicable for non reactive gases. The complexity of the model increases when trying to include detailed materials balances and deposition or removal rates for particles and gases per

material used in the residence (construction materials, furniture materials etc.). Under steady state conditions the major indoor sources for PM contribute as follows:

$$C_{indoor\ sources} = \frac{N_{cig} S_{cig}}{(a+k)Vt} + \frac{S_{cook} t_{cook}}{(a+k)Vt} + \frac{S_{wood\ stove}\ t_{wood\ stove}}{(a+k)Vt} + \frac{S_{combustion}\ t_{combustion}}{(a+k)Vt} \quad \text{Eqn. 1}$$

The first term describes smoking, with N_{cig} the number of cigarettes smoked in the time t, and S_{cig} a source strength for cigarettes (µg particles per cigarette). V is the volume of the room. Disappearance of particles due to ventilation and deposition are accounted for in the factors aeration (a) and average deposition (k) or removal rates in equation 1. Values for a and k are taken from literature (see annex 5). The influence of aeration characteristics on $C_{indoor\ sources}$ is also illustrated in annex 5.

Cooking is also separated in the equation with S_{cook} the source strength and t_{cook} the time of cooking activities. Combustion sources related to heating are split in two term, i.e. a source strength factor for wood stoves ($S_{wood\ stove}$) and a strength factor for other combustion sources (e.g. natural gas, fuel oil). Time of wood stove use and time of heating with other combustion sources are respectively $t_{wood\ stove}$ and $t_{combustion}$.

Model parameterization data and scenario analyses are given in annex 5. The steady state model predicts concentrations in the expected order of magnitude. This relative simple indoor sources concentration model is a useful tool to predict indoor source generated PM concentrations if no measured concentrations are available.

9.3 Indoor-outdoor PM concentration and exposure relations

To understand the validity of using ambient concentrations in order to describe the health effects it is necessary to understand the relation between indoor and outdoor concentrations of (PM) air polluants. The previous paragraph described the effect of indoor sources on indoor PM concentrations. However, a major contribution to indoor PM is infiltration of outdoor PM next to indoor generated PM. The total indoor concentrations can be expressed with equation 2 (Hänninen et al., 2004) which includes a left term related to outdoor concentration and a right term representing the contribution of indoor sources (more in detail described in Equation 1):

$$C_{indoor} = \frac{Pa}{a+k} C_{outdoor} + C_{indoor\ sources} \quad \text{Eqn. 2}$$

The infiltration factor (known as F_{INF}) can be derived in the absence of indoor sources, assuming the following relationship between concentrations indoor (C_{in}) and outdoor (C_{out}) (see also Wallace, 1996; Allen et al., 2003 and Yeh et al., 2002),:

$$F_{INF} = \frac{C_{indoor}}{C_{outdoor}} = \frac{Pa}{a+k} \qquad \text{Eqn. 3}$$

with P the penetration factor. a is the air exchange rate (/h) and k is the deposition, removal or sorption rate (/h). The penetration factor P is the dimensionless fraction of the pollutant in ambient air that penetrates into the indoor environment. Wallace (1996) concludes that the penetration factor is 1 for gases and close to one for PM_{10} and $PM_{2.5}$. This is confirmed by Allen et al (2003) for $PM_{2.5}$. Overall, the infiltration efficiency for particles varies between 0.5 and 0.75 (more data is given in annex 5).

A person's total personal exposure to PM (*T*) is composed of exposure fractionated over different micro-environments, where the concentration of the micro-environments in which that persons spend its time is rescaled with the time fractions that the persons spend in the corresponding micro-environments:

$$T = \sum_{micro-environment\ j} E_j = \sum_{micro-environment\ j} (t_j * C_j) \qquad \text{Eqn. 4}$$

with t_j the time spent in micro-environment *j* and C_j the concentration in micro-environment *j*. Common micro-environments are dwellings, offices, schools, vehicles and outdoor environment. A Dutch study revealed that persons in the category 13-64 y spend on average 14.9 h/day indoors at home, 4.6 h/day indoors at other places (e.g. offices), 4.47 h/day outdoors and 1.3 h/day in vehicles (Kruize et al., 2000). Children (0-12 y) spend on average 17.2 h/day indoors at home, 2.61 h/day indoors at other places (e.g. day care center, school,), 4.13 h/day outdoors and 0.93 h/day in vehicles (Kruize et al., 2000). Time activity data vary across different countries, but differences are small (see for example the expofacts database at http://cem.jrc.it/expofacts), and do not alter the general conclusions from this modelled exposure assessment to indoor PM from woodstoves. This discussion is limited to exposure inside of residential dwellings, although the evidence about sources, pollutants and effects is also applicable to public spaces and offices for example. We do not discuss occupational exposure in specific, but we can not ignore the literature on occupational health effects due to indoor occupational exposure. The use of energy sources for heating will contribute to outdoor air pollution as well, adding to the ambient mixture of air pollutants, but the focus here will be on indoor exposure.

For each indoor micro-environment j indoor exposure from ambient origin (A_j) should be disentangled from the non-ambient exposure (N_j) (Wilson et al., 2006):

$$E_j = t_j * C_j = t_j(F\inf_j C_{j,outdoor} + C_{j,indoor\ sources}) = A_j + N_j$$

with

$$A_j = t_j(F\inf_j C_{j,outdoor}); \qquad N_j = t_j C_{j,indoor\ sources}$$

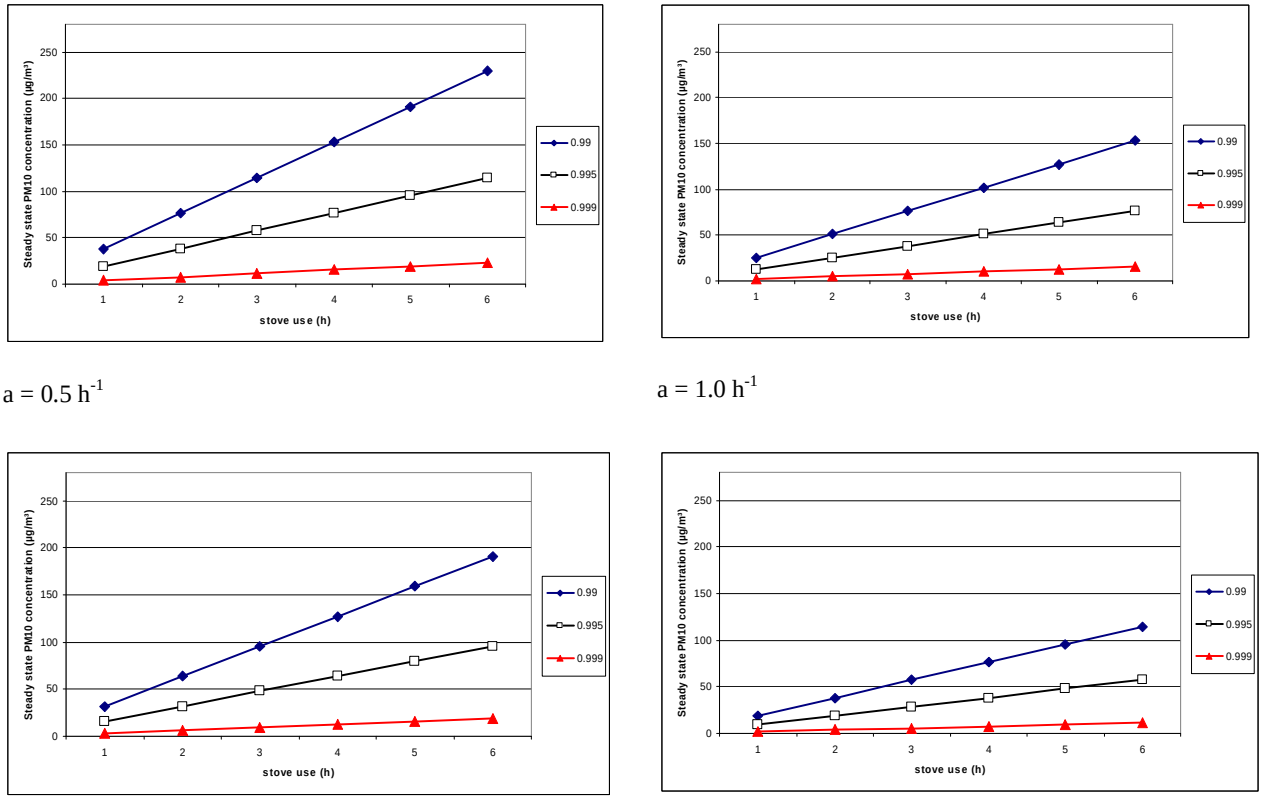
and

$$A = \sum_{micro-environment\ j} A_j; \qquad N = \sum_{micro-environment\ j} N_{j,indoor\ sources}$$

with C_j the concentration in environment j and $Finf_j$ the infiltration factor of indoor environment j. In this case study, the term N_j (with t_j: time spent indoors, and $C_{j,\ indoor\ sources}$ = woodstove) is of interest, and its magnitude relative to the total personal exposure T. More info on the ratio A to N is given in annex 5 as well.

Using this model and taking into account (i) stove PM concentrations modelled; (ii) average time activity patterns of adults and (iii) some assumption on average outdoor concentration of PM enables an assessment of the relative importance of this indoor source to the overall, population average exposure. In annex 5 the time window for woodstove users in the EU is discussed. From the EU energy report (2006), we estimate that on average an equivalent of 14 % inhabitants of the former EU-15 is exposed to indoor wood smoke during 14 % of their time. 86% of people in this part of Europe is hence not exposed to wood stove PM, whereas 100% of people is exposed to ambient PM.

This steady state model has been used to estimate the indoor PM_{10} concentration due to combustion sources. Different scenarios have been calculated (see annex 5 for other scenarios including cooking, smoking and gas stove use). In Figure 9.1 results are summarised for wood stove PM_{10} concentration under different chimney efficiencies and different aeration factors, but for a given emission strength and in a particular case of a 125 m³ inhabited volume.



a = 0.5 h^{-1} a = 1.0 h^{-1}

a = 0.7 h^{-1} a = 1.5 h^{-1}

Figure 9.1: Indoor steady state PM_{10} concentrations from wood stove combustion, for different aeration factors (a) and chimney efficiencies (R = 0.99; 0.995 and 0.999).

To answer the question whether wood stove use might interfere with the overall exposure to PM_{10}, a scenario analysis was made, taking into account an ambient average PM_{10} concentration of 40 µg/m³, and using a fraction 14% of the population that can be exposed to wood stove PM (Figure 9.2). For example, if on average (across all stoves in Europe) the wood stove PM_{10} concentration indoor would be 50 µg/m³, this would then contribute 4% to the total PM_{10} exposure, if ambient average concentrations would be 40 µg/m³ and given the stove use pattern and time use described above.

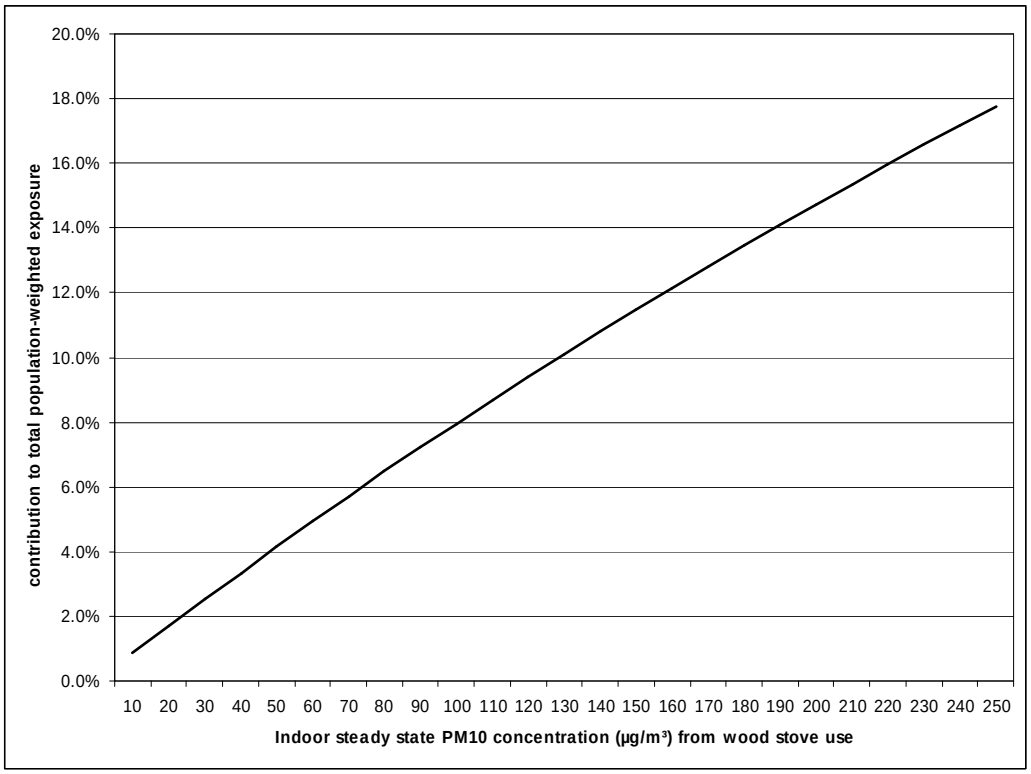


Figure 9.2: Contribution of wood stove use in Europe to total population average PM10 exposure (scenario analysis).

At the individual level, the PM woodstove exposure can amount to >10 % of the personal PM exposure. At average population level, the relative toxicity of wood stove PM must be very high to be of any significant concern when assessing population based health impacts from ambient air pollution. As will be shown further (9.4), there is not sufficient evidence available on the differential toxicity of wood stove PM versus ambient (fossil fuel) PM. There is no good reason to believe that wood stove PM would be more toxic, given the fact wood contains less sulphur and less metals compared to fossil fuels. Note that exposure to some individuals in Europe might still be very high and of concern. Indeed, the negative health impact of woodstove use in a number of studies performed at present times in the EU (see below in 9.5) confirm this. But from this population averaged contribution of PM to personal exposure example, we can safely assume that the use of ambient concentrations –either modelled or measured- are valid in an overall health impact assessment of air pollution, because it dominates the total exposure. Of course in cases where multiple sources are involved, and especially for ETS, the indoor contribution to total exposure increases.

9.4 Ambient versus non-ambient PM: differences in composition, toxicity and health effects?

The vast majority of epidemiological particulate matter-health studies is related to ambient PM. Much less is known about health effect of indoor generated PM (Jones, 1999). Extrapolating the ambient PM concentration/exposure - health effect response to indoor PM assumes that ambient and non-ambient PM are equally toxic. This assumption is however questionable since the composition of indoor PM may deviate of outdoor PM. For example, the particulate organic matter fraction of indoor PM2.5 was near twice as that of outdoor PM2.5 in 173 homes of the RIOPA study (Polidori et al., 2006). Characterization of indoor and outdoor PM in 6 residences in Hong Kong gave similar results (Cao et al., 2005). The proportion of trace elements in PM2.5 was lower indoors relative to outdoors at 20 residences and 6 school rooms in California (Na et al., 2004). In addition, Lunden et al. (2003) demonstrated that changes in composition occur during outdoor-to-indoor transport.

Due to differences in composition and size distributions, it is possible that indoor-generated particles may be more or less toxic than ambient particles. The source of PM was found to play a role in health effects in outdoor PM studies. Lanki et al. (2006) suggested that the PM fraction originating from combustion processes, notably traffic, exacerbates ischemic heart diseases associated with PM. Analogously, it seems reasonable to assume that the difference between indoor and outdoor PM, especially if the PM sources between the two strongly differ, will affect the health impact.

We could not find any study that compares the concentration/exposure – health impact relationship between indoor and outdoor PM at epidemiological level. Long et al. (2001) published an in vitro study comparing toxicity between indoor and outdoor PM and these authors suggested that indoor-generated particles may be more bioactive than ambient particles. These researchers performed an in vitro test on 14 paired indoor and outdoor PM2.5 samples in 9 homes in Boston to investigate the relative toxicity of indoor and outdoor PM. The tumor necrosis factors (TNF) in rat alveolar macrophages (AM) were higher in indoor PM samples compared to outdoor samples, despite that the endotoxin levels did not differ between indoor and outdoor PM. Endotoxins are considered as the major bioactive component of PM2.5. However, after normalization for endotoxins, the contrast between indoor and outdoor PM – TNF response was even enhanced, suggesting that there are other proinflammatory components of indoor particles aggravating indoor PM toxicity compared to outdoor PM toxicity .

It is emphasized that implications of these results with respect to in vivo effects are very uncertain. In vitro exposure conditions are clearly not representative of particle inhalation and deposition in lungs. Even if the vitro toxic effects were representative for in vivo conditions, the higher indoor PM toxicity compared to ambient PM toxicity may become invisible or insignificant in human health studies if ambient exposure dominates non- ambient exposure.

Indeed, the few human health studies in which ambient and non-ambient exposure are separated do not confirm the results of this in vitro study. Koenig et al. (2005) investigated differential health effects of indoor and ambient PM exposure to 19 children with asthma. They found the opposite trend as the in vivo study of Long et al. (2001): the ambient-generated component of PM2.5 exposure was consistently associated with increases in exhaled NO (eNO), a marker of airway inflammation, whereas the indoor-generated component was less strongly associated with eNO. This is in accordance with the recent study of Ebelt et al. (2005). Among 16 patients with chronic obstructive pulmonary disease in Vancouver, non-ambient exposure was not associated with health outcomes, whereas ambient exposure was associated with decreased lung function, decreased systolic blood pressure, increased heart rate and increased supraventricular ectopic heartbeats (Ebelt et al., 2005). It is noted that the non-ambient exposure N is not dominated by A in the study of Ebelt et al. (2005), thus the hypothesis that indoor PM could be more toxic than ambient PM but masked by the much larger contribution of the latter is not plausible.

However, due to the limited number of participants and the biased health status of the participants in the studies of Koenig et al. (2005) and Ebelt et al. (2005), it would not be fair to generalize the smaller health effect of indoor PM than of outdoor PM to population level. In assessing the "strength" of air pollution health effects data, Bates (1992) emphasized that coherence between epidemiological and toxicological data is a prerequisite for drawing firm conclusions. Given the lack of coherence here, at present, the difference between indoor and outdoor generated PM can not be confirmed neither rejected. More research is needed to further unravel this issue.

9.5 Exposure –response functions for woodstove related PM

In general, the health impacts of outdoor exposures to respirable particles are better studied than those associated with indoor exposures (Ostro and Chestnut, 1998). There are less logistical efforts needed to establish a $C_{outdoor}$ – health impact response relationship at epidemiological scale than for C_{indoor} –health impact response. Quantifying the relation

between indoor PM concentration and health impact requires, on the one hand, exposure data, i.e. outdoor PM concentration, indoor PM concentrations, personal PM concentrations, personal time-activity patterns (indoor, outdoor,..), and on the other hand, endpoints for health impacts (respiratory infections, otitis media, emergency department visits, hospital admission,… Indicators for exposure should be recorded at the same moment, or with a short lag time, before the registration of health impact indicators for acute symptoms. Chronic symptoms also require past exposure data.

In addition, epidemiological health impact studies require a very large set of study objects. It is obvious that in practice not all above-enumerated parameters can be quantitatively gathered for the large set of study objects that are required in epidemiological studies. In practice, it is be more feasible and cost-effective to measure surrogates of exposure, such as use frequency of PM sources such as fireplaces and woodstoves. The major part of health impact studies of PM is of this type. The information to obtain these qualitative indicators for exposure is generally retrieved from interviews and questionnaires. Based on such data is not possible to establish a quantitative $N_{woodsmoke}$ exposure – health impact relationship.

Health effects related to woodstove use in developed countries: literature review

Exposure to indoor sources of particulate matter, from combustion of biofuels (wood, charcoal, agricultural residues, dung) have been clearly associated with respiratory disease in developing countries (Smith et al., 2000; Ezzati & Kammen, 2001; WHO, 2002), although at generally much higher concentrations. Given the differences in combustion sources, in building characteristics and time-activity patterens between these developing countries and developed countries it is difficult to extrapolate these high exposure – health effects to typical low exposures common for woodstove use in developed countries. However, European studies on health effects of wood smoke exposure are very scarce. A Spanish study reports that among 120 women hospitalized in the Hospital de Mar in Barcelona for an exacerbation of chronic obstructive pulmonary disease (COPD), there was a strong association between exposure to wood and charcoal exposure and COPD, after adjustment for age and smoking (Orozco-Levi et al., 2006) (

Table 9.1). The majority of the other large-scale (5979 subjects) recent European studies on this topic (Lissowska et al., 2005) was in Central and Eastern Europe (Czech Republic, Hungary, Poland, Romania, Russia and Slovakia), though the United Kingdom was also included. In that study, the odds ratio (OR) of lung cancer associated with solid fuel use was 1.22 (95 % CI :1.05-1.44) for cooking or heating, 1.37 (95 % CI: 0.90-2.09) for solid fuel used only for cooking, and 1.24 (95 % CI:1.05-1.47) for solid fuel used for both cooking and heating. The odds ratio is defined as the ratio of incidence of a health effect (here: lung cancer) in the target group (e.g. solid fuel users, namely wood and coal) to the incidence of the control group (here: non-users of coal and wood). Among the persons that used solid fuels for cooking during their whole lifetime, the OR increased to 1.8 (95 % CI: 1.36-2.41).

Notwithstanding that some OR's are significantly above 1 in the study of Lissowska et al. (2005), the outcome is probably not representative for the entire EU-25 (especially not for the EU-15) since woodstove use (both frequency and technical aspects such as chimney efficiency in terms of indoor PM removal) in Poland, Hungary, Romania,… are probably different from the remainder of the EU.

Table 9.1 gives an overview of studies related to health effects related indoor wood smoke in these regions. The major part of these studies did not report indoor PM concentrations but prevalence of woodstove/fireplace use. Most of these studies deal with population groups that are rather vulnerable to air pollution, i.e. children and asthmatic persons.

Table 9.1: overview of European and N-American studies related to wood smoke health effects.

Reference	Location	Study population (number)	Study group	Health outcome	OR[a] (*or RR)	Sign.?[b]	Remark
Orozco-Levi et al., 2006	Barcelona	120	females (hospitalized for COPD)	COPD	4.5 1.8 1.5	yes no no	both wood and charcoal wood only charcoal only
Lissowska et al., 2005	Central & Eastern Europe	5979	2861 lung cancer patients + 3118 controls	lung cancer	1.22 1.37 1.24 1.80	yes no yes yes	cooking or heating cooking only cooking and heating whole-life users for cooking
Honicky et al., 1985	Michigan	62	young children	mild respiratory symptoms moderate respiratory symptoms severe respiratory symptoms	- - ?	no no yes	
Butterfield et al., 1989	Boise ID	59	children < 5,5 yrs	wheeze, cough and nocturnal awakening	?	yes	
Morris et al., 1990	Arizona	?	children	lower respiratory tract infection	4,2	yes	
Robin et al., 1996	Navajo	45	children 0-2 yrs	acute lower respiratory illness	5	no	
Koenig et al., 1993	Seattle	326	children < 12 yrs, no asthma children < 12 yrs, with asthma	lung function decrement lung function decrement	? ?	yes no	decrement 18 ml/µg/m³
Daigler et al., 1991	Springville	499	children	otitis media	1,7	yes	

Reference	Location	Study population (number)	Study group	Health outcome	OR[a] (*or RR)	Sign.?[b]	Remark
Maier et al., 1997	Seattle	925	children	asthma & wheezing	-	no	multivariate analysis
Ostro et al., 1994	Denver	164	18-70 yrs; with asthma	shortness of breath	1,3	yes	
Eisner et al., 2002	N. California	349	asthma patients	emergency department visits	1,1	no	multivariate analysis
				hospital admissions	0,7	no	multivariate analysis
Triche et al., 2002	Connecticut and Virginia	890	babies (first life year)	# wheeze episodes	0.28*	no	fireplace
				total days wheeze	0.25*	no	fireplace
				# cough episodes	0.97*	no	fireplace
				total days cough	0.99*	no	fireplace
				# wheeze episodes	1.11*	no	woodstove
				total days wheeze	1.08*	no	woodstove
				# cough episodes	1.05*	no	woodstove
				total days cough	1.10*	yes	woodstove

[a] Odds ratio or relative risk (marked with *)
[b] Significance of difference between exposed and non-exposed groups

In a review paper, Naeher et al. (2005) compiled a large number of northern U.S. and Canadian studies related to health impacts of residential wood smoke. In most of these studies, the occurrence of health problems (asthma, lower respiratory illness, shortness of breath, otitis media,…sometimes categorized in terms of mild or severe symptoms) is compared between exposed persons (i.e. woodstove use in their homes) and control persons without woodstove use. For example, Honicky et al. (1985) compared respiratory symptoms in 31 children who lived in homes with wood stoves with 31 children who lived in homes without wood stoves in Michigan. The two groups did not different with respect to mild symptoms but differed significantly for severe symptoms ($p < 0.001$). A similar study was conducted in Boise ID in 1989 by Butterfield et al. (1989). Respiratory symptoms were tracked in 59 children under the age of 5 1/2 years during a winter season. Symptoms such as wheeze, cough, and nocturnal awakening were associated with wood smoke exposure. Morris et al (1990) evaluated the health of children living on the Navajo reservation in Arizona by assessing use of a well-child clinic. All cases of respiratory illness were evaluated for wood stove use in the home. The odds ratio for a lower respiratory tract infection in children with presence of a wood stove was 4.2 ($p < 0.0012$). Triche et al. (2005) found an association between woodstove use and total days of cough among 890 newborns during their 1st year of life in Connecticut and Virginia (RR: 1.10; 95 % CI: 1.02-1.19). The relative risk (RR), which was in that study based on an 8-hour/day increase in source) was not significant different from 1 for association between woodstove use and total wheeze days, number of wheeze episodes and number of cough episodes. The RR for fireplace use was not different from 1 for any of these 4 considered health outcomes. Interesting in that study, was the information on intermittent use patterns of home heating sources, allowing better estimates for exposure than in case if use patterns averaged over the whole winter period would be used. It revealed from this information that fireplaces tended to be used infrequently and for shorter periods of time than woodstoves.

Health effects of indoor air pollution including woodstove use on adult persons in Denver (18 to 70 years) were reported by Ostro et al. (1994). The use of wood fires was associated with an increase in daily moderate or severe shortness of breath. Use of wood stoves or fireplaces was second only to presence of smokers in the home, and more strongly associated with shortness of breath than use of gas stoves or occupational exposures. On the other hand, there are also some studies that have failed to find associations between woodstove emission exposure and respiratory health. Eisner et al. (2002) found no clear relationship between gas stove use or woodstove exposure and asthma health outcomes for a sensitive population group, i.e. 349 asthma patients. In

Table 9.1, some other studies that could not find a significant negative effect of woodstove use on health outcomes are listed (e.g. Robin et al.,1996; Maier et al.,1997), as well as studies that find negative effects of some health endpoints, but not on others (e.g. Honicky et al.,1985; Triche et al.,2002). In addition to woodstove use, Eisner et al. (2002) also investigated the influence of environmental tobacco smoke (ETS) on asthma health outcome. Whereas wood smoke did not affect the odds ratio, this ratio was significantly affected by ETS in the same population group. Neas et al. (1995) also reported increased incidence of lower respiratory symptoms (OR= 1.25) by ETS in a study on 2994 7-11 years children in six US cities. These studies in

Table 9.1 do have some limitations. They all are limited to indoor environments and none has personal exposure information. To our knowledge, the only studies that separates ambient and non-ambient exposure in relation to health effects are those by Ebelt et al. (2005) and Wilson et al. (2006). The health outcome in that study was correlated with ambient PM2.5 exposure (*A*) but not with non-ambient PM2.5 exposure (*N*). Another important finding in the study of Wilson et al. (2006) is that the coefficient of determination (R^2) for *N* versus $C_{ambient}$ (both for PM2.5) near zero is. This is important for the interpretation of health effects due to indoor sources. It suggests that the ambient concentration does not confound the non-ambient exposure and vice versa. In other words, the intercept of the non ambient exposure- health response curves is attributable to ambient exposure but the slope of the curve is independent on the ambient exposure. It is the slope of that curve that tell us the impact of non ambient particles on health outcomes.

The next step should involve a meta-analysis of these data in order to quantify the effect of wood smoke on human health. Among the studies in Table 9.1 there are differences in proportional seriousness. The indicators of adverse effects run from increases in respiratory symptoms to lung function decreases to visits to emergency departments and finally hospitalization. Overall, indoor pollution – health effects studies are still limited and endpoints too diverse to enable a real meta-analysis. One of the difficulties in the meta-analysis is poor quantification of 'exposure' to PM in indoor environments. In outdoor pollution studies quantifiable indicators of exposures such as ambient PM concentrations are commonly used (and can be linked with health data of a large community such as a city near the outdoor PM monitoring station). In most indoor studies however, exposure is expressed in terms of 'woodstove use' or 'no woodstove use' and does not discriminate for frequency of use, appliance,… This is however essential when one wants to compare different studies. For example, disease X might be not significantly affected by woodstove use in study Y_1 whereas disease X is related to woodstove use in study Y_2. The difference might be explained by the difference in woodstove use frequency or technical aspects such as PM indoor spilling between the 'wood stove users' in study Y_1 and 'woodstove users' in study Y_2.

In conclusion, some studies point out that woodstove use has negative effects on human health in developed countries. However, due to the lack of quantification of exposure to woodstove PM in most health outcome studies, it was impossible to derive an exposure-health burden relationship (or woodstove-use – health burden relationship).

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ANNEX 1

1 SELECTION OF C-R FUNCTIONS : CAFÉ-NEEDS-HIA LITERATURE REVIEW

1.1 Quantifying the adverse effects on health of ambient particulate matter (PM)

1.1.1 Mortality in adults aged 30y+ from long-term exposure to PM

It is widely recognised now that the Pope et al (2002) update of the American cancer society (ACS) cohort study is the key study for quantifying the effects on mortality of long-term exposure to PM. Following the task force on health (TFH) of WHO-UNECE, CAFE CBA used as its principal C-R function for quantification an increase of

6% change in mortality hazards (95% CI 2-11%) per 10 µg/m^3 $PM_{2.5}$

from Table 2 in Pope et al. (2002). These are estimates derived from using the average of annual average concentrations from two periods of measurement, one of them early (1979-83) and the other late (1999-2000) in the follow-up period of the ACS study. Given that in the USA ambient $PM_{2.5}$ declined over the follow-up period, use of the 'average' pollution measures is consistent with a short cessation time-lag between changes in ambient PM and consequent reductions in the risk of mortality.

An alternative estimate, also from Table 2 of Pope et al (2002), is

4% change in mortality hazards (95% CI 1-8%) per 10 µg/m3 PM2.5,

derived from using annual average concentrations at the start of the follow-up period. This is consistent with a view of a longer cessation lag. WHO-UNECE recommended that this value be used in sensitivity analyses. Note that recent ExternE-based evaluations (e.g. DIEM, NewExt) used a coefficient of 5%, i.e. the average of the WHO-UNECE 1st and 2nd choice coefficients, above. The changes in risk in any of these coefficients are applied at ages 30 or more, because the ACS study was based on adults in that age range. Primary implementation is via life table methods, using e.g. the RAINS implementation (IIASA, 2002) or the ExternE methods (ExternE, 1999; Miller and Hurley, 2003). As noted earlier, CAFE CBA also included estimates of attributable deaths. Also, these and all other C-R or impact functions for PM below, were applied to anthropogenic PM ($PM_{2.5}$ or PM_{10}), without threshold, irrespective of the source or composition of the PM.

1.1.2 Mortality at all ages from short-term exposure to PM

An issue in quantification is whether deaths as estimated from time series studies of short-term exposures (daily variations in PM) should in some way be included also. The issue is to avoid double-counting while capturing the full effects of PM on mortality. The WHO meta-analysis of studies in Europe (Anderson et al, 2004) provides a suitable coefficient, with % change estimated as:

0.6% (95% CI 0.4%, 0.8%) per 10 µg/m3 PM10

all-cause mortality (excluding accidents), all ages

Note that some of the attributable deaths from time series studies are not already included in mortality as estimated from cohort studies, because the time series studies capture effects at ages less than 30 years, and also (by using the metric of PM_{10}) capture some direct effect of the coarse fraction of PM. These effects are small, however, compared with mortality in adults associated with longer-term exposure to $PM_{2.5}$. To avoid double-counting, Hurley et al (2005a) recommended not to add time series mortality effects attributable to PM to those from cohort studies.

1.1.3 Mortality in infants (aged less than 1 year) from long-term exposure to PM

Woodruff et al (1997), a US cohort study of 4 million infants, showed that post neonatal infant mortality, between the ages of one month and one year, was associated with mean outdoor concentrations of PM_{10} in the 1st two months of life, giving a CRF for change in (all-cause) infant mortality of

4% per 10 µg/m3 PM10 (95% CI 2% - 7%)

It is unclear to what extent these infant deaths associated with and presumably attributable to air pollution occur among young people who are already very frail, and so unlikely to survive into adulthood. This complicates assessment both of public health importance and of monetary valuation. Following Kaiser et al (2004), Hurley et al (2005a) estimated attributable deaths rather than life expectancy, though this transfers the problem to valuation rather than solving it. In any case, in terms of life expectancy the loss of life expectancy due to infant mortality is small compared to the total loss of life expectancy due to particulate matter air pollution of the general population (Rabl 2003).

1.1.4 Morbidity – general methodological remarks

The general approach to estimating the effects of PM (or ozone) on morbidity uses a CRF expressed as % change in endpoint per (10)µg/m^3 PM_{10} (or $PM_{2.5}$) and links this with (i) the background rates of the health endpoint in the target population, expressed as new cases (or events) per year per unit population – say, per 100,000 people; (ii) the population size and (iii) the relevant pollution increment, expressed in µg/m^3 PM. Results are then expressed as estimated new or 'extra' cases, events or days per year attributed to PM. Note that the percentage change in probability can be combined with background rates to give a single *impact function* expressed as:

number of (new) cases, events or days per unit population
(say, per 100,000 people)
per (10) µg/m^3 annual average PM_{10} (or $PM_{2.5}$) per annum

For many health endpoints, reliable data on background rates of morbidity in the EU-25 target population are not readily available. One strategy then is to use other general epidemiological studies of that health endpoint – not necessarily studies of air pollution and health – to provide estimates of background rates, for example the International Study of Asthma and Allergies in Children (ISAAC) and, for adults, the European Community Respiratory Health Study (ECRHS).

Another approach is to estimate an impact function from where the relevant epidemiological studies were carried out and then transfer and use that impact function for quantification in the wider European target population. The two approaches have been used (for different health endpoints) in the CAFE-NEEDS methodology. Otherwise, few if any morbidity endpoints would have been quantifiable.

1.1.5 New cases of chronic bronchitis and long-term exposure to PM

The US Seventh Day Adventist Study (AHSMOG: Adventist Health Smog) study examined people on two occasions, about ten years apart, in 1977, and again in 1987/88. Chronic bronchitis was defined as reporting chronic cough *or* sputum, on *most* days, for at least three months of the year, for at least two years. *New cases of chronic bronchitis* were defined as those who met the criteria in 1987/88, but not in 1977. Using a C-R function from Abbey at al (1995a, Table 6), and a background incidence rate (adjusted for remission of chronic bronchitis symptoms) of 0.378% estimated from Abbey et al (1993, 1995a), Hurley et al. (2005a) derived an estimated impact function of

New cases of chronic bronchitis per year per 100,000 adults aged 27+

= 26.5 (95%CI -1.9, 54.1) per 10 µg/m3 PM10

1.1.6 New cases of chronic cardiovascular disease

It is to be expected then that ambient PM also affects the development and/or the worsening of chronic cardiovascular disease. However, we have not found suitable studies of long-term exposure to quantify these impacts, other than those impacts which result in earlier mortality. Though recently a study on the incidence of cardiovascular disease in post-menopausal woman in the US demonstrated an effect of PM on the development of chronic heart diseases (Miller et al., 2007). In this study the risk of a first cardiovascular event increased with 24% per 10 $\mu g/m^3$ $PM_{2.5}$ in woman with a median age of 63 years.

1.1.7 Respiratory hospital admissions (RHAs: ICD 460-519)

Hurley et al (2005a) used all-ages data, both for CRF and for background rates, derived from APHEIS-3 (Medina et al, 2004), based on eight European cities. Together they imply an impact function:

Annual rate of attributable emergency RHAs

= 7.03 (95% CI 3.83, 10.30) per 10 µg/m3 PM10 per 100,000 people (all ages)

1.1.8 Cardiac hospital admissions (ICD 390-429)

CAFE-NEEDS quantified an effect of PM_{10} on cardiac admissions, using a CRF based on APHEA-2 results from eight cities in Western and Northern Europe (Le Tertre et al, 2002) and a Europe-wide annual rate of emergency cardiac admissions estimated as the arithmetic mean of rates from eight European cities derived from the Appendices of the APHEIS-3 report (Medina et al, 2004). Together these imply an impact function:

Annual rate of attributable emergency cardiac hospital admissions

= 4.34 (95% CI 2.17, 6.51) per 10 µg/m3 PM10 per 100,000 people (all ages)

1.1.9 Emergency room visits

Hurley et al (2005a) did not attempt to quantify a relationship between emergency room visits and PM.

1.1.10 Consultations with primary care physicians (general practitioners)

Studies in London have linked daily variations in ambient PM with consultations with primary care physicians for asthma (but not for lower respiratory diseases) (Hajat et al, 1999) and for upper respiratory diseases, excluding allergic rhinitis (Hajat et al, 2002). These studies were based on numbers of people consulting (including home visits) in a 3-year period 1992-94, among about 282 000 registered patients from 45-47 general practices in the Greater London Area. Because of differences in health care systems, it is difficult to know to what extent these relationships are transferable within Europe. Hurley et al (2005a) therefore proposed that they be used in sensitivity analyses only, to help assess if these endpoints are important.

Consultations for asthma

Separately by age-group, (i) CRF for warm season, adjusted for other factors (Hajat et al, 1999), (ii) mean daily numbers of consultations for asthma in the warm season and (iii) numbers of registered patients were linked and the results expressed as annual impact functions, to give

1.18 consultations (95% CI 0, 2.45) for asthma, per 1000 children aged 0-14

0.51 consultations (95% CI 0.2, 0.82) for asthma, per 1000 adults aged 15-64

0.95 consultations (95% CI 0.32, 1.69) for asthma, per 1000 adults aged 65+

per 10 µg/m3 PM10, per year.

Consultations for upper respiratory diseases (URD), excluding allergic rhinitis (ICD 460-3; 465; 470-5 and 478)

Analyses by Hajat et al (2002), adjusted for season, day-of-the-week effects and climate, showed statistically significant associations between PM_{10} and consultations by adults and by elderly people. Estimates for children, not statistically significant, but quite close to it, are included for completeness. These results, and background rates, were used to derive the following impact functions, for attributable consultations for upper respiratory disease (URD) (excluding allergic rhinitis) per 10 $\mu g/m^3$ PM_{10}, per year:

4.0 consultations (95% CI -0.6, 8.0) per 1000 children aged 0-14

3.2 consultations (95% CI 1.6, 5.0) per 1000 adults aged 15-64

4.7 consultations (95% CI 2.4, 7.1) per 1000 adults aged 65+

1.1.11 Restricted activity days and associated health endpoints

Ostro (1987) and Ostro and Rothschild (1989) used data on adults aged 18-64 from six consecutive years (1976-81) of the US Health Interview Study (HIS), a multi-stage probability sample of 50,000 households from metropolitan areas of all sizes and regions throughout the USA (Ostro and Rothschild, 1989). Within the HIS, RADs are classified according to severity 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.

Restricted activity days (RADs)

Ostro (1987) studied both RADs and work loss days (WLDs) among adults aged 18-64 in separate analyses for each of the six years 1976-81. A weighted mean coefficient for RADs was linked to estimated background rates of, on average, 19 RADs per person per year (ORNL/RFF, 1994) to give an estimated impact function of:

Change of 902 RADs (95% CI 792, 1013) per 10 µg/m3 PM2.5

per 1,000 adults at age 15-64:

In the main analyses of CAFE CBA, this impact function was applied to people at ages 15-64, as in the original study. In sensitivity analyses, the same impact function was used but applied to all ages, on the grounds that it is unlikely that health-related restrictions on activity do not cease at age 65.

Minor restricted activity days (MRADs) and work loss days (WLDs)

As an alternative, Hurley et al (2005) also derived impact functions for work loss days (WLDs) from Ostro (1987) and minor RADs from Ostro and Rothschild (1989), to give, respectively,

Change of 207 WLDs (95%CI 176-238) per 10µg/m3 PM2.5 per year

per 1000 people aged 15-64 in the general population

and

Change of 577 MRADs (95% CI 468-686) per 10µg/m3 PM2.5 per year

per 1000 adults aged 18-64

1.1.12 Medication (bronchodilator) usage by people with asthma

WHO (2004) concluded that there is sufficient evidence to assume a causal relationship between air pollution exposure and aggravation of asthma in children. On that basis, the CASFE-NEEDS quantification of Hurley et al (2005a) proposes impact functions for increased medication usage in people with asthma, although the specific evidence is weak. Separate results were given for children and for adults.

Effects in children aged 5-14 years

Hurley et al (2005) linked an estimated C-R functions from the WHO meta-analysis (Anderson et al, 2004), dominated by the PEACE study and not statistically significant, with estimates of the mean daily prevalence of bronchodilator usage among panels of school-children who meet the PEACE study criteria, to give an impact function of:

Annual change in days of bronchodilator usage

= 180 (95% CI -690, 1060) per 10 µg/m3 PM10

per 1000 children aged 5-14 years meeting the PEACE study criteria

European data from the International Study of Asthma and Allergies in Childhood (ISAAC Steering Committee, 1998) were used to estimate that approximately 15% of children in Northern and Eastern Europe, 25% in Western Europe, met the PEACE study inclusion criteria.

Effects in adults aged 20+

A C-R functions from the WHO meta-analysis (Anderson et al, 2004) was linked with estimates of (i) the mean daily prevalence of bronchodilator use by people with asthma and (ii) the percentage of adults with asthma of a severity comparable to that of the Dutch panels on whom the CRF was based, to give an estimated impact function for change in bronchodilator usage days:

912 (95% CI -912, 2774) per year per 10 µg/m3 PM10

per 1000 adults aged 20+ with well-established asthma

(say, 4.5% of the adult population)

1.1.13 Lower respiratory symptoms (LRS), including cough, in adults with chronic respiratory disease

A random effects meta-analysis of results from five panels was linked to both (i) estimates of the mean daily prevalences of LRS, including cough, in symptomatic panels, based on the studies underlying the CRF, and (ii) estimates of the percentage of people qualifying for a such panels, using data from ECRHS (1996) to give an estimated impact function:

Annual increase of 1.30 (95% CI 0.15, 2.43) symptom days

(LRS, including cough) per 10 µg/m3 PM10

per adult with chronic respiratory symptoms (approx 30% of the adult population)

1.1.14 Lower respiratory symptoms (LRS), including cough, in children in the general population

The recent systematic review by Ward and Ayres (2004) very strongly suggests that effects of PM on respiratory symptoms should be quantified for children generally, and not be confined to children with chronic symptoms. Hurley et al (2005a) combined CRF from Ward and Ayres (2004) with an estimate of the mean daily prevalence of LRS, including cough, based on two general population Dutch studies of children (van der Zee et al, 1999; Hoek and Brunekreef, 1995), to give an estimated impact function:

Change of 1.86 (95% CI 0.92, 2.77) extra symptoms days per year

per child aged 5-14, per 10 µg/m3 PM10.

1.1.15 Acute respiratory symptoms in the population generally

Hurley et al (2005a) quantified acute respiratory symptoms in adults with chronic respiratory disease (Section 6.13) rather than in adults generally. However, *for sensitivity analyses only*, CAFE-NEEDS included also some estimates of the effect of PM on symptom days in the general population, based on Krupnick et al (1990), which had previously been used e.g. in ExternE (1995) to give

Annual change in symptom days per 1000 people at risk (all ages)

= 4650 (95% CI 210, 9090) per 10 µg/m3 PM10

It is likely that this is a high estimate of the effects of PM on respiratory symptoms, especially for application in Europe. It was included in CAFE CBA with the intention that it be used *only* for sensitivity analyses, to indicate how big an effect *might* be.

1.2 Quantifying the adverse health effects of ozone

1.2.1 Effects on morbidity of long-term exposure to ambient ozone

There is no strong or quantifiable evidence that long-term exposure to ozone is associated with health effects additional to those which are the aggregate over time of the effects of short-term exposure, i.e. of daily variations in ozone. Consequently, no impact functions linking long-term exposure to ozone and health were proposed by Hurley et al (2005a).

1.2.2 Framework issue: ozone pollution metric used

The WHO evaluations (WHO 2003, 2004) concluded that there was no evidence for a threshold in the relationship between daily variations in ozone and mortality. However, these evaluations also recognised that, at lower concentrations of daily ozone, there was little evidence on which to base any judgement. Consequently, TFH of WHO-UNECE decided that, in the core analyses, the effects of daily ozone on mortality should be quantified only at ozone concentrations higher than 35 ppb (70 µg/m^3), considered as a daily maximum 8-hour mean ozone concentration. In practice, this means that effects are quantified only on days when the daily ozone concentration (maximum 8-hour mean) exceeded 70 µg/m^3, and then only the increment exceeding 70 µg/m^3 is used for quantification. This increment, aggregated over all days of the year, was called SOMO35 and is the exposure metric used for quantification in CAFE-NEEDS. WHO-UNECE emphasised that the use of a cut-off should not be interpreted as acceptance of a threshold, and recommended also that, as sensitivity analyses, effects be estimated with a cut-off of zero. In CAFE-NEEDS these recommendations regarding no threshold but yes a cut-off for daily ozone, originally developed in the context of daily mortality, were subsequently applied to all impact functions.

1.2.3 Mortality at all ages from short-term exposure to O3

The WHO meta-analysis (Anderson et al., 2004) provided a CRF of an increase in all-cause mortality of

0.3% (95% CI 0.1-0.43%)

per 10 µg/m3 increase in the daily maximum 8-hour mean O3.

This CRF, which applies to all ages, was used in CAFE CBA, in line with guidance from TFH of WHO-UNECE.

It was noted in the CAFÉ CBA report on health impact assessment that "*there have been two very recent publications of the effects of ozone on mortality, one from NMMAPS in the USA (Bell et al., 2004), one from APHEA2 in Europe (Gryparis et al., 2004).*" Gryparis et al. (2004) confirm the mortality risk used here. An analysis of ozone and mortality in 23 cities in Europe resulted in an increase in mortality of 0.33% (0.17%-0.52%) per 10 µg/m³ increase in 1-hr max ozone concentration in summer, while in winter no significant effects were observed. Interestingly for the first time robust information is now available on cardiovascular deaths due to ozone (0.45% increase (95% CI 0.22%-0.69%) per 10 µg/m³ 1-hr max. O3) and for respiratory deaths (1.13% (0.62%-1.48%)). The results in summer seem linear and independent from SO_2 or PM, but the lack of significant effects in winter may indicate some lower effect level for ozone. Effects were studied for 0 and 1 days after exposure. The NMMAPS study (Bell et al, 2004) takes into account a distributed lag of effects after exposure to ozone the previous week. 1-hr max ozone was associated with mortality (0.17% increase per 10 µg/m³ O3, (95% CI 0.11%-0.23%). And the risk increased when only cardiovascular and respiratory mortality were included. There was a significant association with a data subset of days where daily average values were below 120 µg/m³ (60 ppb). The results were also robust to PM_{10} adjustment and insensitive to season and exclusion of high temperature days. These recent findings, on balance, confirm the earlier estimates used by Hurley et al. (2005a).

Table 1.1 : Comparison of ozone mortality risks (% increase per 10 µg/m³ (95% CI) using 1 ppb = 1.997 µg/m³).

	1-hr max	**8-hr max**	**daily average**
NMMAPS (Bell et al. 2004)*	0.17 (0.11-0..23)	0.21 (0.14-0.29)	0.26 (0.14-0.39)
APHEA (Toulomi, 1997)	0.58 (0.2-0.8)		
APHEA2 (Gryparis, 2004)#			
Summer	0.33 (0.17-0.52)	0.31 (0.17-0.52)	
Winter	0.09 (-0.25-0.37)	0.12 (-0.12-0.37)	
WHO meta-analysis		0.3 (above 35 ppb)	

*: distributed lag; #: a priori lags 0 and 1

1.2.4 Respiratory hospital admissions (RHAs)

Anderson et al (2004) used results from five cities in Western Europe to estimate the change in all RHAs in various age groups in relation to daily variations in O_3 (8-hr daily average) with and effect – close to statistical significance – for elderly people only. giving a CRF of 0.5% (95% CI -0.2%, 1.2%) per 10 µg/m^3 O_3 (8-hr daily max) in people aged 65+. Background rates in people aged 65+ were taken from the APHEIS second year report (APHEIS, 2002), giving an impact function (see Section 6.4):

Annual rate of attributable emergency RHAs per 100,000 people at age 65+

= 12.5 (95% CI -5.0, 30.0) per 10 µg/m3 O3 (8-hr daily average)

1.2.5 Cardiovascular hospital admissions

There is no strong or quantifiable evidence that daily variations in ozone are associated with cardiovascular hospital admissions or, indeed, with other cardiovascular morbidity endpoints.

1.2.6 Emergency room visits

Hurley et al (2005a) did not attempt to quantify a relationship between emergency room visits and ozone.

1.2.7 Consultations for allergic rhinitis (ICD9 477), with primary care physicians (general practitioners)

Hajat et al (2001) studied consultations for allergic rhinitis (ICD9 477) and found that relationships with ozone (8-hr daily max) were strongest using a cumulative index incorporating O_3 over four consecutive days, with lags 0-3 days, based on numbers of people consulting (including home visits) in a 3-year period 1992-94, among about 282 000 registered patients from 45-47 general practices in the Greater London Area. Hurley et al (2005) used these results, applying them as if to a single day's pollution, and linked them to mean daily numbers of consultations and numbers of registered patients to give estimates of change in annual consultations for allergic rhinitis per 10 $\mu g/m^3$ O_3 of:

3.03 consultations (95% CI 1.89, 4.29) per 1000 children aged 0-14

1.60 consultations (95% CI 1.22, 2.03) per 1000 adults aged 15-64

Because of differences in health care systems, it is difficult to know to what extent these relationships are transferable within Europe. We recommend that they be used in sensitivity analyses only, to help assess if these endpoints are important.

1.2.8 Minor restricted activity days (MRADs)

For current urban workers, aged 18-64, Ostro and Rothschild (1989) reported relationships between minor restricted activity days (MRADs) and ozone (two-week averages of the daily 1-hr max, in $\mu g/m^3$). The weighted mean coefficient for ozone, adjusted for $PM_{2.5}$, from separate analyses of each of the six years 1976-81 was linked with a mean background rate of 7.8 MRADs per year among people in employment aged 18-64 (Ostro and Rothschild, 1989) to give an estimated impact function:

Increase in MRADs per 1000 adults aged 18-64 per year

= 115 (95% CI 44, 186) per 10 µg/m3 ozone (8-hr daily average)

Issues of uncertainty are addressed, as for other endpoints, in CAFE CBA Vol 3 (Holland et al, 2005a); in UNICE's letter of concerns about the CAFE Methodology, and in the CAFE CBA team's response (Hurley et al, 2005b).

1.2.9 Medication (bronchodilator) usage by people with asthma

As for PM (Section 6.12), the CAFE-NEEDS quantification of Hurley et al (2005a) proposes impact functions for increased medication usage in people with asthma, although the specific evidence is weak. Separate results were given for children and for adults.

Effects in children aged 5-14 years

A CRF was derived from Just et al (2002), a small study of 82 children with medically diagnosed asthma in Paris in early summer 1996, and the only European study identified as a relationship between daily ozone (8-hr daily mean) and medication use in children with asthma. Background rates were derived from Gielen et al (1997) and from Just et al (2002), with different functions reflecting higher prevalences of childhood asthma in Western Europe than in Northern and Eastern Europe (ISAAC, 1998). These results were combined to give an estimated impact function of:

Annual change in days of bronchodilator usage

per 10 µg/m3 O3 per 1000 children age 5-14 years (general population):

124 (95% CI 18, 227) in Northern and Eastern Europe;

310 (95% CI 44, 569) in Western Europe.

Two points should be noted. First, while the effects occur only in children with asthma, the impact function was derived to apply to the general population. Secondly, as noted, Just et al (2002) is a small study, in one location. Furthermore, the estimated odds ratio is very high, compared with other endpoints. The study may well be unrepresentative; it may be best to consider it as an upper limit, e.g. for sensitivity analysis only.

Effects in adults aged 20+ with asthma

Hiltermann et al (1998) gave results linking daily max 8-hr moving average O_3 with daily prevalence of bronchodilator usage was positive (OR 1.009 per 10 $\mu g/m^3$ O_3; 95% CI 0.997, 1.020, i.e. not statistically significant) at the selected lag of 1 day, though when 7-day cumulative ozone was considered, the estimated effect was higher and statistically significant. Background rates were estimated using results from Hiltermann et al (1998) and from the European Community Respiratory Health Survey (ECRHS, 1996). These data were linked to give an estimated impact function:

Change in days of bronchodilator use of

730 (95% CI -255, 1570) per 10 µg/m3 O3

per 1000 adults aged 20+ with well-established asthma

(approximately 4.5% of the adult population)

1.2.10 Acute respiratory symptoms in children in the general population

Work in progress by the Committee on the Medical Effects of Air Pollutants (COMEAP) in the UK suggests that there is convincing evidence that daily variations in ozone are associated with lower respiratory symptoms (LRS), including cough; and that these effects are not restricted to people with chronic respiratory symptoms such as asthma (Heather Walton, 2004, personal communication). The CAFE-NEEDS Methodology Report (Hurley et al, 2005a) used a small general population study of 91 children in Armentieres, Northern France (Declerq and Macquet, 2000), to quantify relationships linking (i) daily prevalence of cough and phlegm and (ii) lower respiratory symptoms (LRS), excluding cough with 8-hr daily max O_3. The relevant C-R functions were linked with background rates derived from Hoek and Brunekreef (1995) to give impact functions:

a change of 0.93 (95% CI -0.19, 2.22) cough days

and 0.16 (95% CI -0.43, 0.81) days of LRS (excluding cough)

per child aged 5-14 years (general population), per 10 µg/m3 O3, per year

1.3 Selection of CRF functions

Table 3.1 in chapter 3 represents the set of concentration response and impact functions from the joint CAFE-NEEDS HIA literature review, as used in CAFE CBA.

ANNEX 2

2 Exposure metrics: ambient concentrations, indoor and personal concentrations

In a HEI funded research project Brunekreef and colleagues (2005) measured $PM_{2.5}$, particle reflectance (as a metric for elemental carbon) and composition of PM at a fixed site outdoor. At the same time indoor and personal levels of PM were also measured. The study population consisted of 37 cardiovascular patients in Amsterdam and 47 in Helsinki. In total 337 personal and 409 indoor measurements were collected in Amsterdam, and 306 personal and 503 indoor in Helsinki. Median personal, indoor and outdoor $PM_{2.5}$ concentrations (24-h. average) were 13.6, 13.6, and 16.5 µg/m³ in Amsterdam, and 9.2, 9.2 and 11.1 µg/m³ in Helsinki. Median values for **indoor–outdoor ratios** were 0.73 for $PM_{2.5}$ and 0.87 for EC in Amsterdam and respectively 0.79 and 0.74 in Helsinki, in cases were no ETS was present. These median values are the result of all measurements taken together. Looking at the distribution of the median indoor–outdoor ratio per individual the median is around 0.75, and higher than 0.5 in 95% of cases. From the set of paired indoor, personal and outdoor measurements per individual, a Spearman correlation is calculated. The distribution of these individual **correlation values for $PM_{2.5}$ personal – outdoor** has a median value of 0.78 in Amsterdam and 0.73 in Helsinki, in cases without ETS, with 75% of correlation values higher than 0.57. To accept the hypothesis at the 5% level of significance that there is a correlation between personal and outdoor concentrations, the correlation values should be higher than 0.5 (in cases with 10 or more paired measurements) up to 0.9 in the few cases with only 4 paired measurements. Details of the individual cases were not available, but the conclusion is that personal and indoor concentrations were correlated with $PM_{2.5}$ outdoor (median correlation R = 0.7-0.8). Most elements, especially S and EC were highly correlated as well. The study proves that fixed site measurements are a good proxy of the personal exposure to $PM_{2.5}$ in **time-series studies**, especially for components that have no indoor sources (like S). (**Gas) cooking** seemed to contribute 3.4 µg/m³ (SE 1.5 µg/m³) to personal $PM_{2.5}$ and 2.5 µg/m³ (SE 1.3 µg/m³) to indoor concentrations. The PTEAM (Wallace, 1996) study estimates a much higher contribution of 9 µg/m³ to the indoor 24-h averaged $PM_{2.5}$ concentrations). The findings are not representative for the general population, because this susceptible group of age between 50 and 84 years stays more indoor. It does not necessarily imply that other groups of the population that spent more time outdoor, but also in other indoor environments, experience the same correlation between personal and outdoor concentrations. The findings are certainly not to be used when people are exposed to ETS. Furthermore the fixed site measurements are performed at locations nearer to the places of residence than in general would be the case when using fixed site ambient monitors in urban areas. This increases the chance of being well correlated with indoor concentrations.

The personal-outdoor correlation found in the study of Brunekreef et al. (2005) is comparable to correlations found in a study of 18 COPD patients in Boston, USA (Rojas-Bracho et al., 2000). In this study a 12 hour sample during daytime in the summer was used. In a study of elderly people in a retirement home Williams et al (2000a, b) also found high correlations between personal and outdoor concentrations, based 24-hour samples of $PM_{2.5}$. Ebelt et al. (2000) however did only find a modest correlation between personal and outdoor $PM_{2.5}$, in a study of 16 COPD patients in Vancouver. Sarnat et al. (2000) demonstrated a large difference in correlation of personal and outdoor $PM_{2.5}$ concentrations for different seasons. The better ventilated the buildings are (in summer) the higher the correlation. The correlation in winter however was very poor in this study. The work of Brunekreef (2005) builds on earlier work of Nicole Janssen. It confirms previous studies of the same design on children (Janssen et al. 1999) and on elderly (Janssen, 2000). It is also worth mentioning that studies involving healthy adults, with a more varied time activity pattern generally find lower longitudinal correlations between personal and outdoor concentrations to $PM_{2.5}$ (Adgate et al., 2003). So caution is necessary when applying ambient concentrations in health impact assessment in all circumstances. But overall there is good reason to use central site monitoring stations as a proxy of real exposure in assessments of the acute impact air pollution can have on susceptible groups in the populations. Zeger et al (2000) analyse the potential errors when using ambient (and even imprecise ambient) concentrations on the estimation of the acute mortality risk due to exposure to particles. It is concluded that ambient concentrations most likely underestimate the true risk, and that only when indoor sources generate particles with the same composition and toxicity that correlate with outdoor concentrations this may be a major source of error. Wilson and Suh (1997) however demonstrate that there is no correlation between indoor sources and outdoor concentrations.

ANNEX 3

3 Summary of reviews of the evidence: to what extent might component-specific toxicity be quantifiable?

Purpose of this limited review

The model outlined in chapter 6 gives a formal framework within which the specific contributions of different pollutants can be considered, individually and together. We have formulated the model on general terms, because in principle the range of issues to be addressed by it is very large. We recognise however that, on current evidence, the model as developed here is too detailed to be used as such. By this we mean that there is not sufficient evidence on which to base quantification of many of its component parts. In particular, there is insufficient evidence for quantifying most of the necessary f_i. Indeed, the current established view (WHO, 2004), is that evidence at present does not allow *any* differential slopes for different sorts of particles, except a differentiation between PM_{10} and $PM_{2.5}$. In addition, and perhaps partly as a consequence, most air pollution scientists are unwilling to commit to subjective estimates of the factors f_i.

While recognising that evidence is limited, we consider that the established position is overly pessimistic, given the importance of the issue of differential quantification. We consider that the position regarding importance is as follows.

i. There is some substantial evidence that particles from different sources and/or of different characteristics (other than size) have different toxicities, per unit exposure.

ii. Studies of general urban air pollution provide estimates of the effects of the urban particulate mixture. These associated coefficients are what is needed when, for example, the aim is to estimated the burden of disease and mortality associated with current levels of ambient air pollution.

iii. Some of these studies provide estimates also of the relative effects of different components of ambient particles. Where such estimates are provided, judgement is needed about whether the relevant particulate pollutant is itself the causal agent, or whether it is a marker of pollution from e.g. a particular source.

iv. Estimating the overall burden of disease is however only one application of HIA methods in the context of ambient air pollution. The principal use of quantified HIA and CBA is in evaluating the likely benefits of policies to reduce air pollution (or the possible damage to health of policies which, as an unintended side-effect, may increase pollution). By definition, these are policies which lead to changes in the concentrations of air pollution experienced by the populations exposed. The purpose of the HIA is then to evaluate the effects on health of that change in air pollution.

v. The key point is that *typically, the pollution mixture that has been changed is different in significant ways from the general urban air pollution mixture that has been studied*

epidemiologically. This is true both for the relationship between particles and gases, and for the constituent parts of the particulate mixture.

vi. Currently, the only workable method of evaluating (estimating) the effects of the 'change mixture' is to disaggregate it into its component parts; estimate the effects of the parts individually; take account, wherever practicable, of the fact that these components are experienced as part of a mixture; and then re-aggregate effects across the component parts.

vii. This puts a much greater emphasis on estimating correctly the contribution of individual components than does a HIA to establish burden of disease.

viii. Because this is so important, we think that it is progressive to attempt differential quantification, including of different kinds of particles, even when the evidence to support differential quantification is limited, rather than simply use as default an assumption which is widely believed to be wrong, i.e. that, within a given size range, all particles have similar toxicity.

Against that background, the purpose of the present brief review is to indicate what scope there may be for differential quantification of components of PM.

Particles and health

There are numerous detailed and well-established reviews of particles and healthy, most recently as part of the WHO Air Quality Guidelines Global Update. It is now better understood that different components of PM are toxic. In general however toxicology doesn't enable us to quantify the contribution of specific components of PM to the overall health impact of PM. Epidemiological studies that measure the exposure to specific components are limited. We give a brief overview of the different potential toxic constituents of PM. Where possible we link the toxicological evidence with epidemiological results. The following text draws mainly on reviews by the Health Effects Institute (2002) by epidemiological and toxicological experts (Schlesinger et al., 2006) and by the US EPA (2003)

From dosimetry it is concluded that particles with an aerodynamic diameter between 10 to 50 nm are most likely to deposit in the alveolar region of the lungs. The largest fraction of particles bigger than 1 to 2 µm deposit in nose, throat and mouth. The very fine particles with magnitudes around 1 nm diameter are mainly deposited in the extra-bronchial zone of the breathing system (WHO, 2004). Deposition in the alveolar zone is suspected to induce cardiovascular effects, perhaps through translocation of ultra fine particles to the bloodstream (but evidence is limited, see Nemmar (2002) and Mills (2006)). Ultra fine particles are still a potential explanation for some of the health

effects of particles. Experiments with TiO2 and graphite point to an increasing toxicity of particles with decreasing diameter (Macnee and Donaldson, 1999, and Donaldson and Stone, 2003). Both PM_{10}, $PM_{2.5}$ and ultra fine particles are capable of inducing oxidative stress in the lungs, leading to inflammatory reactions. The important characteristic seems to be the contact surface of particles, which is significantly larger for ultra fine particles per mass unit. This increases the potential for toxic or reactive elements on the surface of particles to interact with lung cells, causing inflammation and disease. It also illustrates the complexity of explaining particle toxicity. It is not yet clear whether size alone or the combination size with chemical characteristics of the particle is determining the toxicity. It is evident that some components on the surface of particles like adsorbed PAHs, may contribute to lung cancer when deposited in the lower regions of the lungs. But it is not always clear: diesel exhaust particles for example are able to enhance lung injury, both due to the carbonaceous core only (i.e. removing metals or organics did not affect this, Yanagisawa, 2003) and through the complete particle (i.e. by washing leachates or organics extractions the reactivity of the original material did not alter; Pan, 2004). Both size and relative abundance of metals and organics in the different size fractions of PM seem to explain some of the effects of particles. Component specific toxicity can only be determined when restricting to one size mode. Otherwise chemical and size characteristics cannot be disentangled (Schlesinger, 2005).

But composition of particles is even more complex. Several metals like iron, vanadium, nickel and copper are associated with DNA damage, oxidative stress and inflammation. Several studies contribute to the evidence that metals in residual oil fly ash and from steel industrial sources are toxic (Schlesinger et al., 2005). One notable result is the study of Ghio and Devlin (2003) who instilled to volunteers particles from Utah Valley, from a period before during and after a strike in a steel mill. Inflammation was associated with the samples from the periods when the steel mill was in operation. Further analysis showed that the samples that were causing more inflammation had higher amounts of copper, zinc, lead, and nickel. This theory of particle toxicity emphasizes the ability of metals on fine particulates to induce toxic oxygen radical species and subsequent inflammatory response in the lung.

Particles serve as the transportation vehicle for toxic gases that can adsorb onto them (like aldehydes). Some of the organic compounds that are adsorbed onto particles are mutagenic, or can induce irritation or allergic reactions. Carbon nuclei can promote fibrosis in the lung tissue. Organic carbon components and elemental carbon are suspected of contributing to oxidative stress in the lungs, but information is limited because organic matter is not very well characterized and might be

very different in different locations. Most information from toxicology stems from diesel exhaust particles, that contain a large fraction of organics. Mills and colleagues (2005) demonstrate that inhalation of diesel exhaust at levels of approximately 300µg/m³ can damage vascular function in humans, providing a plausible mechanism that links air pollution to aetherothrombosis and acute heart attack. Important evidence comes also from the Dublin intervention study (Clancy et al., 2002), where black smoke is the strongest indicator for reduced respiratory and cardiovascular mortality due to restrictions on coal combustion. But in two studies funded by the health effects institute Frampton et al. (2005) and Gong et al. (2003) didn't find any significant effect of controlled exposure to resp. ultra fine carbon particles and concentrated air particles (CAP) on healthy and asthmatic subjects.

Particles might even contain viruses, bacteria and endotoxines, giving rise to acute respiratory effects. The same applies for (parts of) pollen, spores from fungi who are allergenic. These latter are dependant on season, but up to now little systematic information is available in Europe.

It is mentioned before that from a toxicological point of view ammonium salts (sulfates, nitrates) aren't toxic at low (ambient) concentrations (Schlesinger and Cassee, 2003). Sulphuric acid however is capable of causing respiratory problems, certainly with asthma patients, but at rather high concentrations. The acidity of particles due to the presence of sulphuric acid or in nitric acid can change the solubility of metals. Studies taking into account the acidity of ambient air show early effects (of perhaps chronic conditions) associated with the presence of acid vapour (Gauderman, 2005).

A lot of the studies point to either diesel particles (in toxicological studies) or to traffic as source of hazardous pollution. Although in epidemiological studies the evidence is indirect, using proximity to traffic as indicator of exposure, the co-varying presence of gases like CO, or the modifying effect of NO2 on mortality risks, there is on balance reason to give particles from traffic a higher weight in the impact assessment. At the same time NO2 and CO should then be excluded from the impact model, because of the probable overestimation of the impact. Because of the proximity of traffic sources to humans these sources are easier to detect. Other industrial or residential sources contribute to the general ambient air and are more homogenous across large regions, and are therefore more difficult detect in epidemiological studies where spatial exposure contrast is an important criterion to detect significant associations. This doesn't mean that these other sources are

not harmful to human health. In time series for example, where exposure contrast is over time rather than over space. Intervention studies like in Utah valley clearly link industrial PM with health effects. It is nevertheless not possible to compare both sources with respect to their relative toxicity, only their relative contribution to the exposure is being measured. In most cases dilution of industrial sources through regional dispersion makes the contribution of industrial sources less important than traffic, when emissions of both these sectors are comparable.

But it is not yet completely clear how these component specific toxicities add up to the total toxic effect of particles. Röösli et al (2003) demonstrate that the known carcinogenic components of PM, considering their mass contribution and the reported unit risk factors do not add up to the full lung cancer effect of PM as measured by the long-term cohort studies. Sulphates and SO_2 in the ACS study show up as being associated with mortality. Whereas sulphates are seen as a component of $PM_{2.5}$, the association of gaseous SO_2 with mortality seems to act independent and is fairly robust against recent sophisticated analyses that take into account a multitude of co-variables, confounders, and spatial auto-regression corrections.

It is concluded from this review that there isn't sufficient evidence to reliably weigh the contribution of different components of PM, in order to extend the model to evaluate and quantify impacts on health due to PM (equation 7). Some limited evidence is available to:

- give traffic PM higher weight than other PM;
- keep sulphates and perhaps nitrates into the impact estimate, with equal weight to particles from high stacks, because they represent the effect of the mix of particle and gas emissions that disperses from high industrial stacks;
- to give a weight to organic matter components of PM, if they are modelled, and if it is likely to contains carcinogenic material like (nitro-)PAHs;
- to take metal fractions and ultra fine carbon fractions into account separately if data and modelling capacity is available.

ANNEX 4

4 $PM_{2.5}$ exposure assessment

The variability within cities, what does it do to effect estimates for a city?

There is a possibility that variability between measurement sites within a metropolitan area is larger than the variability between cities, and that this will lead to underestimation of the real risk in cohort studies. Newer analyses using intra-urban interpolated concentration maps demonstrate this. In brief the reasoning behind this is:

1. Spatial uniformity is not always true within a city. The review by Wilson and colleagues (2005) gives a methodology to make a thorough study and good classification in this respect.
2. Pope et al. (1995, and 2002) and the reanalysis (Krewski et al., 2000) use central site ambient monitoring stations as the exposure metric. Probably some of these metropolitan areas in the ACS study have a uniform monitoring system, and some have a heterogeneous set of measurements, showing higher variability of $PM_{2.5}$ within a city than between cities.
3. This leads in the case of heterogeneous exposure situations to misclassification of exposure, where people in the "high polluted metropolitan areas" actually live in clean neighbourhoods and vice versa, and this is not picked up by the analysis of the ACS study (Pope et al. 1995 and Krewski et al., 2000), nor in the follow-up (Pope et al., 2002).
4. Exposure classification because of this intra-urban variability of $PM_{2.5}$ leads to underestimation of the risk. Therefore the new spatial analysis of air pollution and mortality in Los Angeles, taking into account this intra-urban variability, gives higher risk estimates (Jerrett et al., 2005). The potential of exposure misclassification is reduced.

Air pollution exposure in the cohort studies is an ecologic variable that is measured at the group level (per city) rather than on the individual level. Epidemiologists have spent considerable effort in thinking about the impact of errors in epidemiological studies on the strength and significance of the association. With respect to non-differential[3] errors in epidemiological studies Armstrong (1998) concludes that

- Classic random errors in continuous exposure variables (like concentrations) result in an attenuation of the true relative risk with a factor that is proportional to the

[3] Non-differential error does not depend on the health outcome. In case survival or life expectancy is the outcome, as in the ACS semi-individual cohort study, both subjects who have died during follow-up as those who survived were equally likely to be misclassified. Prospective studies have the advantage of measuring exposure prior to the outcome, making differential error less likely (White et al .1998).

correlation between independent measurements of the concentrations (e.g. the concentration measured at several stations within a city).

- Berkson error (e.g. when using exposure at an ambient monitoring site as a single exposure measure for a group) gives little or no bias in the risk estimate, but widens the confidence interval and hence lowers the power of a study to detect significant associations.

In case the errors are differential, it is unclear whether the bias is upward of downward. Künzli and Tager (1997) presented indirect evidence for the fact that underestimation of the effect estimates is likely to occur in semi-individual studies. Note also that these theoretical considerations try to evaluate the true risk in relation to the observed risk. The use of the approximate estimate, e.g. when trying to predict the effect of air pollution reduction, is not influenced by error and is still appropriate. In other words, the established relationship between ambient air particle concentrations and mortality remains fit for purpose, even with measurement error.

EPA (2004) states that the correlation between measurements is good enough to use the average long term concentration as a proxy. Moreover it is this proxy that is being used in standard setting and in predicting the risk after implementing clean air measures. In that case the proxy of exposure is appropriate. EPA's summary report of the results of the PM research programme (EPA, 2004) concludes that there is a longitudinal correlation (i.e. in time) with the averaged personal exposure over a large group of people within a community, based on a series of exposure studies since 1997. The PM research program of US-EPA also concludes that ambient $PM_{2.5}$ is a good predictor of personal exposure to PM2.5, inter-personal differences being explained through network characteristics and I/O characteristics (and of course indoor sources). What's more, it is now understood that there is a weak correlation between personal $PM_{2.5}$ concentrations and the personal exposure to gaseous pollutants, which makes the gaseous pollutants unlikely confounders for the relation between $PM_{2.5}$ ambient and individual health effects. Finally, there seems to be a strong correlation between personal $PM_{2.5}$ concentrations and ambient concentrations of the gaseous pollutants, making them appropriate surrogates for the effects of $PM_{2.5}$ on health.

Other authors also describe a strong correlation between $PM_{2.5}$ and PM_{10} ambient concentrations measured at different sites within a community. For example Özkaynak and Spengler (in Wilson and Spengler (1996)), report correlations ranging from 0.9 to 1 in Philadelphia for $PM_{2.5}$, and higher than 0.7 for PM_{10} in Chicago and LA, when distances are shorter than 32 km between sites. In Riverside California, central site PM_{10} was also correlated with the measurements of PM_{10} outside

the residences of 178 volunteers. Also Landis et al. (2001) found a good correlation between $PM_{2.5}$ and sulphur measured at the community level, outdoor near residences and personal, over longer time periods. This illustrates that spatial correlation of measurements within a city is possible, but whether this ambient concentration is also representative for personal exposure for a large group of people in the long-term is difficult to prove. The fact that there is good temporal agreement between measurement sites within the same city, and the longitudinal correlation with the individual exposure to particles, does not implicate that there is a long-term average spatial homogeneity between these sites. This aspect is well documented by Wilson and colleagues (2005) and by Pinto et al. (2004). Although the spatial correlation of measurement sites within the same city or metropolitan area is good in many locations in the US, basically because of the high contribution of secondary $PM_{2.5}$, which is a regional pollutant that is more homogenous across cities, there are notable exceptions (Pinto et al., 2004). For 27 urban areas Pinto and colleagues showed that although temporal correlations between daily values of $PM_{2.5}$ measured at different sites were high, the 90-th percentile of the absolute daily concentration difference between these sites were larger than 10 $\mu g/m^3$ for one or more site pairs in 17 of these cities. On the other hand, annual mean values of $PM_{2.5}$ in these 27 urban areas, did not differ more than 6 $\mu g/m^3$ from site to site (except for Los Angeles). Caution is warranted because of this uncertainty in exposure. This is also concluded by (Nerriere et al., 2005), and from the study of Brunekreef where the authors state that "…*for epidemiological studies of long-term exposure to air pollution, the implication of the large within-person variance component is that it is difficult to assess differences in long-term average exposure between subjects with a small number of repetitions per subject.*"

The reanalysis team failed to address this aspect fully, because it requires a thorough investigation of the relation between individual long term exposure and the measured concentrations from fixed-site monitors (see Krewski et al., 2000 p229). Individual measurements however are unavailable and costly. They used the variation between monitors within a city as a first indication of exposure measurement error, and concluded that in case of high measurement error the relative risks might even double. The influence of indoor–outdoor relationships and the error in instrumentation used to measure the concentrations was not addressed. The latter seems to be of minor importance (Krewski et al. 2000 p229). This has also been reviewed by the US-EPA to decide whether false negative findings in epidemiological studies of the coarse fraction of PM_{10} (PM_{10}-2.5) can occur due to this kind of measurement error. It is concluded that it is highly unlikely that the effect of coarse PM_{10} is transferred to a surrogate (like $PM_{2.5}$). Therefore, measurement error is not important. The issue of indoor PM is more important. If exposure to indoor PM (from specific sources, uncorrelated with

ambient PM) is acting independent, and on average equally high in different cities, than the indoor component will not confound the estimate of mortality due to outdoor PM very much. Dominici et al. (2000) examined a database consisting of five different PM exposure studies and concluded that nonambient PM exposure can be treated as relatively constant from city to city although the data show greater variability than a similar analysis of data reported by Ott (2000). Although it is quite reasonable to assume that indoor exposure to particles from indoor sources is not correlated with the exposure to ambient particles, this assumption might even be relaxed. Abrahamowicz et al (2004) simulate the effect of omitting or aggregating covariates in semi-individual studies like the ACS study. It is concluded that aggregating or grouping covariate measures will systematically result in and underestimation of the true effect.

This also means that grouping the exposure variable will result in underestimating the effect estimate. But it is nevertheless interesting to develop better risk estimates that take into account variability across a population living in the same study area. Jerrett et al. (2005) demonstrate in a spatial analysis of mortality among ACS participants in LA that the mortality risk is indeed increased, even with a factor 2 to 3, when implementing a more detailed exposure assessment through 'intelligent' interpolation of air pollution monitors in the area. With this information, it is clear that, although there might be substantial variability within cities, the inter-urban comparison of risks will likely underestimate the true effect and not overestimate or generate false positive results.

With this in mind, it is justified and probably even on the safe (i.e. lower boundary) site to use average ambient concentrations of $PM_{2.5}$ to evaluate the long term risks and effects of exposure to ambient air pollution. Moreover it is the ambient concentration that is affected by policies that reduce emissions, and from that point of view unnecessary to go into detail on the exact (total) exposure of people.

the variability of PM2.5 across individuals (either in the same city or between cities). When does it affect estimates of mortality when comparing different cities?

The cohort study of Pope et al. (2002) uses $PM_{2.5}$ concentrations, measured in central site monitoring stations in metropolitan areas. The exposure assessment of the population utilises the average concentration over these monitoring stations within one area. This community average exposure index is likely to misclassify part of the population under study. Some people will be more

exposed to $PM_{2.5}$ due to local sources, and therefore the health burden might be underestimated when the average measured concentration is used. It is also plausible that within-city variability of the $PM_{2.5}$ concentrations is large. The use of the average concentration over monitoring stations within a city is then not a good representation of the distribution of the populations' real exposure When comparing two cities on the basis of their average annual concentrations, this intra-urban variability then causes uncertainty.

If this variability exists and is large, and if this is also reflected in a variability of exposure to personal PM, people will be misclassified. If furthermore this variability occurs in many cities, and when variability within cities is larger than the variability between cities, exposure will be biased and effect estimates biased to lower or higher values. In cases where a lot of cities are compared (like the ACS study) this is less likely to occur.

ANNEX 5

5 INDOOR EXPOSURE TO PM FROM COMBUSTION SOURCES, ADDITIONAL DATA

5.1 Introduction

To assess the potential contribution of indoor exposure to the total exposure to particulate matter, a case study is developed based on a modelling approach (chapter 9). This case study illustrates the fact that indoor sources are of importance to some people, constituting a large part of their daily average exposure, but that this contribution of indoor sources is quite small on a population average basis (including people without the indoor source in question) compared to the general exposure to ambient PM both outdoor and penetrating indoor. This annex contains additional data and scenarios. To derive at these modelled results we have made an inventory of emission data from literature (5.2 and 5.4), from woodstove and woodstove use in Europe (5.3). Given the fact that data on house characteristics, stack efficiencies and woodstove use is limited and also very variable across the EU we have followed a scenario approach, testing different values for the different parameters (5.5).

5.2 Emission data for combustion sources indoor

There exists an extensive literature on indoor exposure to air pollution. The influence of combustion sources on the indoor concentration is either derived from comparing a situation with or without the source, or directly measured in controlled circumstances in the absence of other sources. Average concentrations of NO_2 over longer averaging periods are in the order of 20 to 100 µg/m³ (Kotzias et al., 2005). Short term peak values can be higher than 1000 µg/m³ (1hour) (Kotzias et al., 2005). Dedicated measurements of PAHs due to airtight and non airtight stoves give insight into the effectiveness of closed stoves. Airtight stoves generated 2 to 5 ng/m³, comparable to outdoor concentrations. Non-airtight stoves' concentrations were as high as 100 to 350 ng/m³ (Orme et al., 2002). The same difference was measured for TSP: 24 to 70 µg/m³ for airtight stoves, higher than outdoors, but much lower than non-airtight stoves (150 to 500 µg/m³). A significant effect of smoking and kerosene heating is also noted by Leaderer et al. (1994), where $PM_{2.5}$ concentrations in 394 homes during a 7-day measurement ranged from 14.1 to 61.4 µg/µ³ indoor compared to 16 to 19 µg/m³ outdoor. Typical source emission rates are scarce in literature. In (Orme et al., 2002)

values for kerosene and as heaters are compared to cigarette smoking emission rates, but only for VOC (Table 5.1).

Woodstove PM emission rates to the indoor environment depend on the efficiency of the chimney, the spilling of ashes, smouldering of the fire,… Modelling of the PM emission rate to the indoor environment is further worked out in 5.5.

Table 5.1:Typical emission rates for sources of VOCs in residences

Source	Condition	Emission factor	Assumed amount	Emission rate(mg/h)
Combustion sources				
Unvented gas burner		85-144 mg/h	1 burner	100
Unvented gas space heater (HCHO)	radiant	0.001 mg/kJ	20,000 kJ/h	20
Unvented kerosene space heater	convective/ radiant	0.007 mg/kJ	6100 kJ/h	45
Unvented kerosene heater	radiant	0.064 mg/kJ	9400 kJ/h	600
Cigarette smoking	1 smoker	10 mg/cig	2 cig/h	20

5.3 EU woodstove inventory

In order to assess the magnitude of the woodstove PM related health problem, it is essential to have knowledge about how widespread and frequently woodstove use prevails in the EU.

To our knowledge, accurate numbers of woodstove users and their woodstove use frequency for the EU (or at national levels) do not exist or are based on a rather small number of households (e.g. in Flanders based on a survey of 1000 households). Therefore, these numbers are derived from national wood consumption data for the residential sector. According to the yearly energy statistics report of the European Commission (2006 Edition report), 130 000 ton wood [4] (and wood-waste) was consumed for energy purpose in 2004 by the EU-15 and 156 000 ton wood by the EU-25 in 2004. Of the total amount of renewable energy sources excluding electricity, which consists on average by 93 % of wood and wood-waste, on average 63 % was consumed by households and services in 2004, thus 80 000 ton wood was consumed by households and services in 2004 by the EU-15 and 102 000 ton by the EU-25. Though, wood consumption per household varies a lot between EU countries (Table 5.2).

[4] energy units expressed as ton wood recalculated from toe (tonne of oil equivalent) in the EU Energy report and heating value of 12 MJ/kg wood and 42.5 MJ/kg fuel oil

Table 5.2 Wood consumption in the EU (source: the EU energy statistics report of the EC, 2006)

data 2004	total wood consumption	total wood consumption households & services	wood consumption per household [a]	% households 'typical wood users' [b]	# persons in typical wood users households
	x 1000 ton wood/year		kg wood /household/ year	%	x 1000 persons
EU-25	155897	101508	531	14%	64965
EU-15	128699	79564	496	13%	50921
Belgium	1544	674	162	4%	449
Czech Republic	2436	1745	427	11%	1163
Denmark	2223	1899	774	21%	1114
Germany	16051	14233	362	10%	7970
Estonia	1722	1274	2452	65%	883
Greece	3210	2854	672	18%	1979
Spain	11984	6983	478	13%	5400
France	31143	26189	1014	27%	16761
Ireland	644	155	92	2%	99
Italy	5348	4644	209	6%	3220
Cyprus	4	130	532	14%	104
Latvia	3686	2997	3619	96%	2238
Lithuania	2013	1653	1391	37%	1278
Luxemburg	53	49	272	7%	33
Hungary	2167	2250	578	15%	1560
Malta	-	-	-	-	-
the Netherlands	1096	812	115	3%	498
Austria	8400	6570	1937	52%	4205
Poland	13472	10789	876	23%	8919
Portugal	8680	4130	1104	29%	3084
Slovenia	1498	1136	1479	39%	787
Slovakia	921	14	8	0%	11
Finland	18505	4035	1700	45%	2367
Sweden	17798	3273	875	23%	2094
UK	1904	1203	46	1%	738
Bulgaria	2482	2261	783	21%	1628
Croatia	1323	1099	693	18%	821
Romania	10665	10021	1108	30%	6413
Turkey	19359	23086	784	21%	14775
Norway	3679	2372	1243	33%	1518

[a] all households were included, irrespective of residence type (flats, houses,…), and we assumed wood consumption by the services sector was negligible compared to the residential sector.

[b] the number of 'typical wood users' is calculated from both the number of households with wood as main energy (heating) source, and the number of households with wood as secondary energy source, accounting for their wood consumption relative to wood consumption of main wood heaters.

In a Flemish survey on energy consumption by household performed on 1000 households between 2001 and 2005, 1 - 2 % households used wood energy as main heating source (Flemish Community, 2005). In addition, 14-28 % (depending on year of the survey) used wood as secondary heating source. Irrespective of main or secondary heating source, 45 % of woodstove users heat near daily

with wood during wintertime, 31 % at least a few times per week, 11 % ± once a week and 12 % use their woodstove rather rarely. Belgian households with an operational woodstove/fireplace as main heating source, burn on average 1 stere[5] wood per month during the heating season or 8 steres per year. Assuming a average specific density of 620 kg/m³ for wood, this corresponds to annual consumption of 3.500 kg wood for a woodstove using household. A recent study in the Lombardy region of Italy confirms this estimate: average consumption of wood was 4.000 kg per family in 2004 (excluding non-users). For calculations of number 'typical wood using' households in Table 5.2, a default value of 3750 kg wood per year was used. We realize that this simple default value ignores variation between households, residential type and winter regimes across Europe. Nevertheless, if one assumes linearity between wood consumption and indoor PM and between indoor PM and health outcomes (without thresholds), the overall relation between wood consumption and health outcome is not violated. In other words, if these assumptions are fulfilled, the net health outcome of 4 households (in a total of 100 households) burning 3.750 kg wood/year would be equal that of 2 households (in a total of 100 households) with wood consumption of 7.500 kg wood/year.

From the EU energy report (2006), an average (i.e. including woodstove users and non-users) wood consumption of 160 kg wood/year/household in Belgium was derived (4.2 % typical wood energy user). Other countries with relative low wood energy consumption per household are the U.K., Italy, Ireland and the Netherlands.

From the annual wood consumption per household (Table 5.2) it was derived that that 4.2 % households in Belgium burn annually 8 steres. These numbers are in line with results of the Flemish energy consumption survey, i.e. 1-2 % households with wood as main energy source and 14-28 % with wood as secondary wood stove. This points out that a 'typical' secondary wood user consumes 6-fold less wood than people having wood as their main heating source. As mentioned above, number of households that consume wood as heating source (irrespective of main or secondary heating) were recalculated to numbers of 'typical' users with wood as main energy source.

The estimates of persons exposed to indoor PM related to wood smoke is here (column 6 in Table 5.2) based on a conservative assumption that all persons belonging to a household (i.e. on average 2.4 persons/household in the EU) stay in at home (and in the room where the wood stove is placed) during the wood stove use of that household. This assumption was made due to lack of data, and should be considered as a worse-case scenario.

[5] one stere is one cubic metre loosely packed chunks of wood, corresponding to 0,57 – 0.8 m³ wood volume, depending on chunk size.

time window for woodstove use

Thus, based on the EU energy report (2006), we estimated that an equivalent of 51.000.000 persons or 14 % in the EU-15 are exposed to indoor PM originating from yearly consumption of 3.750 kg wood in their households (Table 5.2). Given an average wood burning rate of 3 kg wood/h, this corresponds to 1.250 hours per year, or 14 % of the time. This value seems rather high, but, again, it should be considered as an equivalent to 'main energy' wood users. It was estimated that average 'secondary' wood users consume 6.5-fold less wood, and are thus exposed 2 % of their total time budget to operating wood stoves. Assuming that the ratio of households with wood as main energy source to the ones with wood as secondary energy source is not different between Belgium and the rest of the EU, it is estimated that 18.200.000 persons in the EU are exposed to operating woodstoves during 14 % of their time, and 196.710.000 persons are exposed to woodstoves during 2 % of their time. Again, this is a strong simplification of the continuum of woodstove use frequencies and wood consumptions.

In addition, the time budget of wood stove use is different from the time window of indoor wood smoke PM exposure. Obviously, the exposure lasts longer than the period of woodstove use. However, kinetic modelling of wood smoke derived indoor PM concentrations was not included in this study, and, therefore, time duration of woodstove use is here above used as a proxy for exposure time.

It should be stressed that the time window (14 % time) and the proportion of people exposed to indoor PM (14 % of the EU-15 inhabitants, corresponding to 14 % of their time) related to indoor PM from wood as main energy source is relatively small compared to the time window and number of persons that are affected by outdoor generated PM. Notwithstanding people spend less time outdoors than indoors, the outdoor generated PM also infiltrates indoors, and, in contrast to indoor PM, outdoor PM affects the whole population.

According to the WHO report " Indoor smoke from solid fuels: assessing the environmental burden of disease at national and local levels" (Desai et al, 2004), the number of EU inhabitants who are affected by wood smoke (and its related diseases) is negligible. However, our estimate of potentially exposed persons in Europe, based on the EU Energy Report (2006), appear to be higher than the WHO estimate. The difference between our estimates and the WHO estimates probably relies in the inclusion of households with wood as secondary energy source in our analysis. It is not clear from their report how the WHO handled this group, but they probably ignore their possible exposure.

5.4 Model parameterization data

The model parameters used for modelling the impact of woodstove use on indoor PM concentrations using the model described by Equation 1 in paragraph 9.2 were retrieved from literature sources:

deposition or removal rate (k)

Ranges of deposition rates have been reported in Wallace (1996) and He et al. (2005) and compared with other studies. Allen et al. (2003) report additional results. The deposition rates for PM vary from about 0.2 to about 1 (h^{-1}) with peak values of 2.5 per hour in exceptional cases (Allen et al., 2003; He et al., 2005), under different air exchange rates. A relationship between deposition and air exchange rate is found for particles larger than 80 nm but smaller than 1 µm (He et al., 2005).

aeration rate (a)

Air exchange rates (a) reported vary between 0.1 and 0.6 h^{-1}, with some studies reporting rates higher than 1 in a limited number of cases (3 measurements in a series of 43 in Allen et al., 2003.). This is also confirmed in a Flemish study on energy conservation in residences (Senvivv, 1998), where the air exchange rate is deduced from measurements of the air tightness of buildings. Here we find an average air exchange rate of 0.4 h^{-1}, with a maximum of 6 h^{-1}. These considerations however do not include the influence of inhabitants on ventilation. It is generally agreed that activities like window opening, opening of doors etc… can increase air exchange rates with a factor 2 to 5 (Wallace, 1996; He et al., 2005). Rooms with open fireplaces have also typically large ventilation rates. There is to our knowledge very limited information or data in literature to disentangle natural ventilation due to building characteristics from ventilation through inhabitant activities. As a first lead there is the study of He et al. (2005) where deposition rates for particles where investigated under minimal (i.e. without opening windows and doors) and normal ventilation conditions. Air exchange rates for 14 houses have been measured but not reported individually

source strengths factors for cigarettes (S_{cig}), cooking (S_{cook}), wood stoves ($S_{wood\ stove}$) and other combustion sources ($S_{combustion}$)

The source strength for cigarettes (6680 µg PM10/cigarette) can be derived from an experiment of Invernizzi et al. (2006); Koutrakis et al. (1992) report a higher value, with a PM2.5 source strength of 12.7 mg/cigarette. The latter value was used in below reported calculations. Olson and Burke (2006) surveyed sources strengths of PM10 for cooking events. These authors

reported a mean value of 36 mg/minute (median: 12 mg/min) across all cooking events (n = 411), with highest mean sources strengths from burned food (470 mg/min), grilling (173 mg/min) and frying (60 mg/min). In that study, mean cooking durations were 11 min (median 7 min). One should keep in mind that fine particles produced during cooking can partly directly be removed through the kitchen fan before penetrating the bulk indoor environment (see also below for combustion sources). However, the person that is preparing the food can be fully exposed to this point source.

A starting point for estimating the source strength of woodstove and other fuel/appliances are reported PM emission factors. Emission factors for PM10 and PM2.5 for different fuels (wood, coal, natural gas, oil fuel) and appliances are listed in Table 5.3.

Table 5.3 PM emission factors for fuel combustion

fraction	fuel	appliance/application	emission factor	emission[b]	reference
			g PM /kg fuel	g PM/h	
PM10	wood	conventional wood stove	15,3	13,5	http://www.epa.gov/woodstoves/changeout.html
PM10	wood	non catalytic wood stove	9,8	8,7	http://www.epa.gov/woodstoves/changeout.html
PM10	wood	catalytic wood stove	10,2	9,0	http://www.epa.gov/woodstoves/changeout.html
PM10	wood	masonry heater (wood stove)	2,8	2,5	http://www.epa.gov/woodstoves/changeout.html
PM10	wood	domestic open fire places	1,8[a]	1,6	BUWAL, 2001, cited in www.iiasa.ac.at/rains
PM10	wood	domestic furnaces	1,8[a]	1,6	BUWAL, 2001, cited in www.iiasa.ac.at/rains
PM10	wood	domestic small boilers, wood pieces	0,6[a]	0,5	BUWAL, 2001, cited in www.iiasa.ac.at/rains
PM10	wood	small boilers, automatic loading	1,0[a]	0,9	BUWAL, 2001, cited in www.iiasa.ac.at/rains
PM10	wood	domestic heating	1,1[a]	1,0	TNO, 2001, cited in www.iiasa.ac.at/rains
PM10	wood	domestic heating	2,3[a]	2,0	TNO, 2001, cited in www.iiasa.ac.at/rains
PM10	wood (pine)	fireplace	2,8	2,5	Gullett et al., 2003
PM10	wood (oak)	woodstove	9,6	8,5	Gullett et al., 2003
PM10	wood (oak)	fireplace	5,6	4,9	Gullett et al., 2003
PM10	wood (artif. log)	fireplace	16,6	14,7	Gullett et al., 2003
PM10	wood pellets	pellet stove	2,1	1,9	http://www.epa.gov/woodstoves/changeout.html
PM10	wood/coal	coal & wood	10	8,9	Lee et al., 2005

fraction	fuel	appliance/application	emission factor	emission[b]	reference
PM10	coal	small furnaces	3,7[a]	1,2	BUWAL, 2001, cited in www.iiasa.ac.at/rains
PM10	coal	domestic boilers	3,0[a]	1,0	BUWAL, 2001, cited in www.iiasa.ac.at/rains
PM10	coal	industrial boilers	1,5[a]	0,5	BUWAL, 2001, cited in www.iiasa.ac.at/rains
PM10	natural gas	domestic furnaces	0,02[a]	0,005	BUWAL, 2001, cited in www.iiasa.ac.at/rains
PM10	natural gas	domestic boilers	0,01[a]	0,002	BUWAL, 2001, cited in www.iiasa.ac.at/rains
PM10	light fuel oil	domestic furnaces	0,04[a]	0,011	BUWAL, 2001, cited in www.iiasa.ac.at/rains
PM10	light fuel oil	domestic boilers	0,008[a]	0,002	BUWAL, 2001, cited in www.iiasa.ac.at/rains
PM10	light fuel oil	residential	1,3[a]	0,3	Berdowski et al 1997, cited in iiasa.ac.at
PM10	heavy fuel oil	residential	1,9[a]	0,5	UBA, 1989, cited in iiasa.ac.at
PM10	heavy fuel oil	residential	2,1[a]	0,5	Berdowski et al 1997, cited in iiasa.ac.at
PM2,5	heavy fuel oil	residential	1,3[a]	0,3	Berdowski et al 1997, cited in iiasa.ac.at
PM2.5	light fuel oil	residential	1,3[a]	0,3	Berdowski et al 1997, cited in iiasa.ac.at
PM2.5	wood	domestic heating	1,1[a]	1,0	TNO, 2001, cited in www.iiasa.ac.at/rains
PM2.5	wood	domestic heating	2,2[a]	1,9	TNO, 2001, cited in www.iiasa.ac.at/rains

[a] recalculated from emission factors expressed as kJ/PT units and lower heating values of 12 MJ/kg wood, 33.3 MJ/kg coal, 42.5 MJ/kg fuel oil and 38.1 MJ/kg natural gas (Bossel, 2003).

[b] hourly particulate matter emissions for a 125 m³ room (or house) with an average specific heating demand of 85 kJ/m³/h

Wood combustion for heating purposes produces up to > 10g PM10/kg wood, in contrast to more than 100-fold lower emission from natural gas (0.01-0.02 g PM10/kg). Given the larger heating value of natural gas than of wood, this contrast is even enhanced if emissions are expressed in terms of energy producing capacity.

Within the fuel category 'wood', PM emissions vary among appliance (fireplace, woodstove type) and wood type (oak, pine, pellets). From Table 5.3, an average emission factor of 8.0 g PM10/kg for woodstoves and 6.7 g PM10/kg for fireplaces was derived, corresponding to 7.1 g PM10/h for woodstoves and 5.9 g PM10/h for fireplaces for heating a 125 m³ room with a heating demand of 85 kJ/m³/h.

It is however not realistic to use these emission factors as indoor source strength factors. Doing so, this would imply a complete mixing of particulate matter from woodstove with the bulk indoor environment before removal via deposition and ventilation. The major part of PM10 produced in

the woodstove or fireplace is directly enforced outdoors through the chimney and does not contribute to indoor PM10. Under correct chimney dimensions and properly functioning of the chimney, only a very small fraction of PM10 produced at the woodstove enters the indoor environment. However, three conditions can lead to significant fluxes of PM to the indoor environment. Firstly, chimney draft is small at the start of the fire due to initial low chimney air temperature. Secondly, a smouldering fire also creates weak chimney draft. Opening the woodstove loading door during these conditions will spill smoke, and thus fine particles, in the room. In the third place, the chimney draft might be converted to a indoor flux in case if an underpressure is created in the room due to another air draft, e.g. kitchen fans or central vacuum cleaners. It is thus clear that indoor combustion source strengths are smaller than PM emission factors; however, the extent of removal through chimney is largely dependent on the individual situation of woodstove use. Malfunctioning by chimney obstruction or flow inversion due to underpressure leads to huge smoke spills indoors which will make the user to stop the fire/repair the chimney. There should be a distinction between these accident-based PM exposures and PM exposures related to good practice use of woodstoves; the latter involves PM emissions related to initial low chimney draft, smouldering fire and dispersion of ashes after fire extinction.

Basically, smoke withdrawing or smoke spill is related to the same principles of chimney draft for open fireplaces as for woodstoves. However, it is assumable that a slightly larger fraction of smoke/particles enters the room with open fireplaces, because there is no loading door or other type of barrier between the burning fire and room as is the case for a woodstove.

Direct removal of emitted PM through chimneys (for woodstoves and flued gasstoves) or kitchen fans can be taken into account in the model by the respective removel R factor; if direct removal is taken into account the factors S in Eqn. 1 are replaced by S^*, where

$$S^*_{cook} = S_{cook} \times R_{kitchen\ fan}$$

$$S^*_{wood\ stove} = S_{wood\ stove} \times R_{wood\ stove\ chimney}$$

$$S^*_{gas\ stove} = S_{gas\ stove} \times R_{gas\ stove\ chinmey}$$

5.5 Scenarios

The effect of model parameters, presence/absence of indoor sources is here illustrated with a few hypothetical scenarios (Table 5.5).

Source strength, emission factors (mean values) and room characteristics for home indoor environments were fixed among all scenarios and listed in Table 5.4. The time t (14.9 h/ 24h) is the average time people spend indoor home in the Netherlands (Kruize et al., 2000). This home indoor time pattern corresponds well with typical average hours spent indoors in other EU countries (13-17 hours, in Finland, France, Italy, Czech Republic; source: Expofact sheets, available at http://envi.uku.fi/expofacts2006). In addition, an *average* specific heating demand of 85 kJ/m³/h is used to calculate hourly fuel consumption for a 125 m³ volume. This corresponds to 0.9 kg wood/h and 0.27 kg natural gas/h. However, during winter time, when people use woodstoves, a higher energy demand corresponding to 3 kg wood/h is more realistic. The latter is included as scenario 4e and 4f in Table 5.5. In a first approach, air exchange rate of 1 is assumed and a 8-hours heating period out of 14.9 h indoor stay.

Table 5.4 default parameters used across all scenarios

V_{home}	125	m³
k	0,5	-
t	14,9	h
S_{cig}	12700	µg/cigarette
S_{cook}	2160000	µg/h
$EF_{combustion, woodstove}$	7118750	µg/h
$EF_{combustion, natural gas}$	5313	µg/h

It is mentioned that Equation 1 is here parameterized for the PM10 fraction, and not for the finer PM2.5 fraction. Some studies cited below report only PM2.5 and not PM10. It should be kept in mind that 50-90 % of PM10 exists of PM2.5, depending on its source and meteorological conditions (Rombout et al., 2000).

Predicted particulate matter (PM10) indoor air concentrations originating from indoor sources under different indoor air scenarios are given in Table 5.5. Total indoor concentrations (C_{indoor}) due to indoor sources (i.e. without the contribution of outdoor PM10 penetrating to the indoor environment) is split into contribution of cigarettes (C_{cig}), woodsmoke ($C_{woodsmoke}$) and natural gas (C_{gas}) boilers as heating system.

Table 5.5 Influence of heating with natural gas (1a-1b), cigarettes smoking (2a-2b), cooking (3a-3b), woodstove use (4a-4b), and a combination of these factors (5a) on indoor source concentrations of PM10. Direct removal of PM through chimney or kitchen fan is not accounted for, unless marked with*.

scen	N_{cig}	t_{cook}	$t_{woodstove}$	t_{gas}	C_{cig}	C_{cook}	$C_{woodsmoke}$	C_{gas}	C_{indoor}
	# cigarettes/h	h	h	h	µg/m³	µg/m³	µg/m³	µg/m³	µg/m³
1a	0	0	0	8	0	0	0	15	15

1b*	0	0	0	8	0	0	0	1	1
2a	0,067	0	0	8	5	0	0	0	5
2b	1	0	0	8	68	0	0	0	68
3a	0	0,36	0	8	0	278	0	0	278
3b*	0	0,36	0	8	0	7	0	0	7
4a	0	0	1	7	0	0	2548	0	2548
4b*	0	0	1	7	0	0	25	0	25
5a*	0,33	0,36	4**	4	6	4	38	0	47

* accounted for direct removal of PM through chimney or kitchen fan; R-values used in these examples: see text

** wood rate of 3 kg/hour (winter regime)

In scenario 1a, 8 hours per day heating with natural gas using a unflued gas stove is the single indoor PM10 source. This scenario is representative for room without cooking activities e.g. offices and bedrooms. Gas combustion contributes to 15 µg PM10/m³ if an unflued gas stove is used. This contribution drops to 1 µg PM10/m³ in case if gas stove/boiler is provided with a chimney (R= 0.95) (scenario 1b). Current modern devices nearly completely (R= 0.95-1) remove combustion products such as PM10. In the EXPOLIS study the duration of gas stove use arose as a significant determinant explaining 1.4 % variability in indoor air PM2.5 concentrations (Lai et al., 2006). Filling in a time duration of 8 hours gas stove use in the empirical model of Lai et al. (2006) that is based on measured indoor concentrations in 413 indoor dwellings 6 EU cities, a contribution of 4 µg PM2.5/m^3 from gas stove use is predicted. This concentration falls in the range of PM concentrations emitted by unflued (15 µg PM10/m³) to near perfectly flued gas stoves (1 µg PM10/m³), and suggests that, on average, nearly one third of PM emitted by gas stoves contributes to indoor PM levels. It is however worth mentioning that gas consumption rate during gas stove use might deviate largely (i) between different stoves/homes in the EXPOLIS study and (ii) from the assumed 0.27 kg natural gas/h.

In scenario 2a, the effect of cigarette smoking (one cigarettes in 14.9 h) is modelled, resulting in a predicted indoor environment concentration of 5 µg PM10/m³. One cigarette per hour augments C_{indoor} with 68 µg PM10/m³ (scenario 2b). In the EXPOLIS study, ETS revealed as the major indoor source of indoor PM2.5 (explaining 17.7 % variability in indoor PM2.5). The empirical model for Helsinki of Lai et al. (2006) predicts an increase of 10 µg PM2.5/m³ for the situation of 1 smoking person per house compared to only non-smokers (average consumption in Helsinki was 1 cigarette/day/home); the corresponding difference between 0 en 1 smokers in Milan was 3 µg PM2.5/m³ (average consumption in Milan was 4 cigarette/day/home). It is noted that among the 6 EU cities, the model for Helsinki predicted largest influence of smoking on C_{indoor} whereas the one

for Milan the lowest. The prediction for comparable cigarette consumption rate according to our model (1 cigarette per 14,9 h or 0.067/day) matches very well the ones of the empirical EXPOLIS model. Wallace et al. (2003) reported that the level of PM in inner-city homes was on average 37 µg PM/m³ higher in smoking households than in non-smoking households. In the former households, on average 9 cigarettes per day were smoked inside home.

It is noted that the current model assumes a steady-state model, which might be less appropriate for cigarette smoking than for more constant sources such as combustion for heating. Actual exposure to cigarette smoke PM10 will be higher immediately after cigarette lightening than the calculated average exposure.

Two average cooking events (t = 11 min; S = 36 mg PM10/min) without a kitchen fan (scenario 3a) have an up to 10-fold larger contribution (278 µg/m³) to indoor PM concentrations than cigarettes smoking or heating with gas. Cooking without kitchen fan is a common practice in less developed countries, for which indeed such high indoor PM concentrations have been reported (280 – 1560 µg PM3.5/m^3; 24-hours averages), especially when biomass cookstoves are used (Albalak et al., 2001). Kitchen fan performance strongly decreases $C_{cooking}$. Cooking with a well-functioning kitchen fan (R = 0.975) (scenario 3b), which is the most common practice in developed countries, only marginally affects indoor concentrations (7 µg/m³).

It is obvious from these examples that cooking is a larger source of indoor PM than heating with natural gas. Cooking with a well-functioning kitchen fan during 0.36 h (7 µg/m³) contributes more than 10-fold PM10 than that predicted for a well-functioning gas boiler (< 1 µg/m³). In contrast, PM indoor concentrations under these common cooking conditions are well below the ones of frequent cigarette smoking.

One hour woodstove use (rate 0.9 kg wood/h) without chimney draft (scenario 4a; $R_{chimney}$ = 0) is expected to raise the average indoor concentration with 2548 µg PM10/m³ during 14.9 h. As mentioned above, this is expected to happen only in accident-based situations; (even then, a woodstove user normally will extinguish the fire earlier than the postulated 1 hour wood stove use time). Near to complete chimney draft (R=0.99) is more realistic and predicts $C_{woodstove}$ of 25 µg/m³ for 1-hour burning at 0.9 kg/h (scenario 4b). The large influence of PM removal through chimney draft on indoor PM concentration originated from wood smoke is illustrated in Figure 5.1.

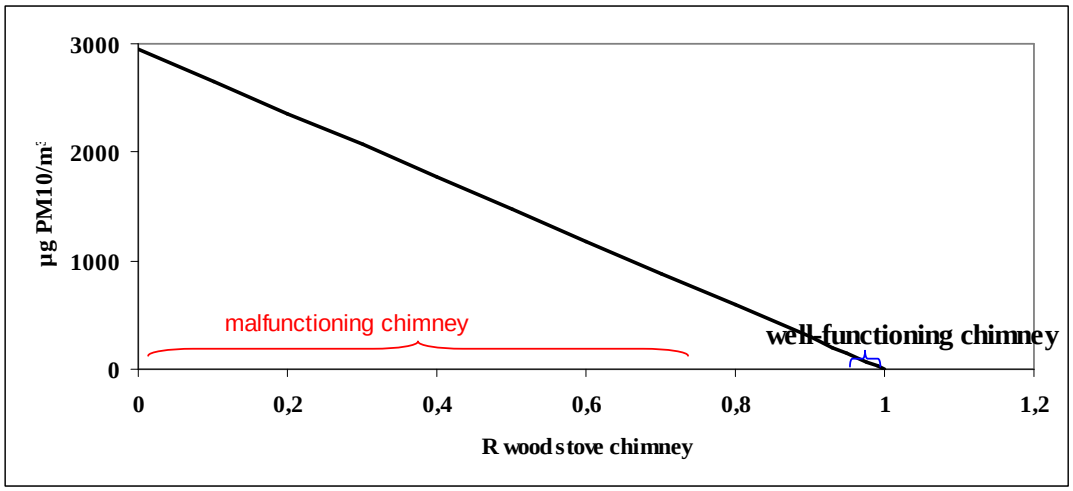

Figure 5.1: effect of direct removal of PM via the chimney on indoor PM concentrations (R= 0: no chimney; R=1: perfect chimney).

Four hours wood burning at 3 kg/h in a wood stove with chimney with R = 0.99 might lead to 354 µg PM10/m³. Chimney draft of R= 0.999 would lower C_{indoor} for the latter case (4h woodstove use at 3 kg/h) to 34 µg/m³. It is noted that the value of $R_{chimney}$=0.99 is speculative and actual $R_{chimney}$ values vary among the individual wood stove use. Coupled measurements of combustion rate, indoor concentrations, time of loading door opening, time of smouldering fire, …are necessary to obtain realistic ranges of $R_{chimney}$ values.

At the moment, no information was found whether R=0.99 or R=0.999 is closest to a realistic average. Nevertheless, referencing with measured concentrations in houses with vented airtight stoves (average 24-77 µg TSP/m³; Traynor et al., 1987) supports R values above 0.99, or even above R=0.999. Namely, indoor PM2.5 was only marginally and **not** significant (P = 0.278) elevated in Swedish homes with wood burning for heating purpose in wintertime (median C_{indoor} = 12 µg/m³; 24-h period) compared to reference houses without wood burning (median C_{indoor} = 9.5 µg/m³; 24-h period) (Molnár et al., 2005). In the study of Wallace et al. (2003) woodstove or fireplace use not a significant regressor in the indoor air PM model. Perhaps, the portion of households with woodstove or fireplace use was too low (1%) in the survey to reveal PM as significant for indoor PM.

Among all model predictions in Table 5.5, a default aeration rate of $1h^{-1}$ was used. The effect of variations in aeration rate on indoor PM concentrations in a house with 1-h burning woodstove is illustrated in Figure 5.2. Dwellings with woodstoves or fireplaces typically have larger aeration rates (e.g. a = $3h^{-1}$) than the default value of 1 used in model predictions in Table 5.5. Such an increase in aeration rate from 1 to 3 h^{-1} leads to halving of PM concentrations if other factors are

kept constant. Other conditions that also lead to high ventilation (a = 2 -5) are frequent door and window opening.

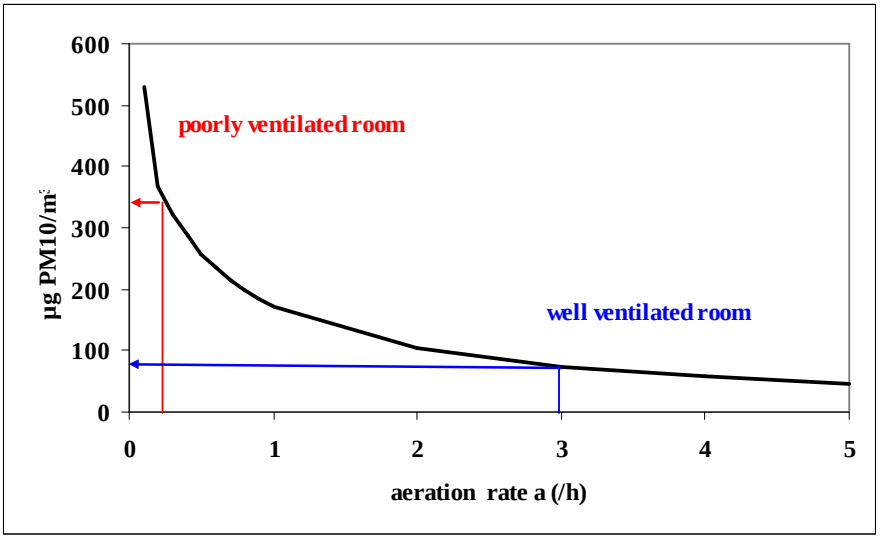

Figure 5.2: the effect of aeration rate on indoor PM concentrations in a room with a woodstove provided with a chimney (R= 0.975)

In scenarios 5, a combination of cigarettes smoke, cooking and woodstove use (3 kg/h) in a well ventilated house(a = 5/h) is made. Particulate matter from the woodstove is far the largest contributor to C_{indoor}, (>80%) even if a large $R_{chimney}$ (R= 0.99) is assumed. It is noted that a poorly ventilated house (a = 0.2/h) with cigarettes smokers (e.g. 1 cigarettes/hour), with vented gas heating and no wood burning may have larger C_{indoor} (145 µg/m³) than in under a scenario of a high wood stove use with a very good functioning chimney (R = 0.995) and very well aerated rooms (a = 5/h).

In conclusion, we can state that this relative simple indoor sources concentration model is a useful tool to predict indoor source generated PM concentrations if no measured concentrations are available. The examples above are an illustration, numerous other assumptions and parameter values are possible.

5.6 Indoor – outdoor relationships in literature

In homes without indoor sources (including human activity) indoor PM10 concentrations are typically 70 % of outdoor concentrations (Monn et al., 1997). The highest indoor/outdoor (I/O) concentration ratios, i.e. I/O = 2, were recorded for homes with smoking inhabitants. Occurrence of human activities and gas cooking resulted in I/O ratios of respectively 1.4 and 1.2 (Monn et al.,1997). Cao et al. (2005) found I/O ratios of 1.0, 1.5 and 1.0 for residential homes in Hong Kong near roadsides, in urban areas and rural areas respectively. Mean residential indoor concentrations

of ambient PM2.5 particles ranged from 7 (Helsinki) to 21 µg/m³ (Athens) in The EXPOLIS study. In the EXPOLIS study I/O PM2.5 concentration ratios vary from 0.90 (Athens) to 1.04 (Prague) (Götschi et al., 2002). All above I/O ratios refer to 24-h average concentrations variation in I/O ratios are much larger for smaller time resolution (Cao et al., 2005).

As stated in the introduction, it is in respect of energy externalities of utmost importance to separate the outdoor generated PM concentration from that of indoor generated PM. Homes with fuel combustion as the major source of indoor PM fraction are most suitable for this purpose (thus excluding ETS). The I/O ratio of a species that had no indoor sources can be used as a tracer for the attenuation factor for particles with the same size distribution. Sulphate is often used as a tracer for PM2.5 (Wilson et al., 2000; Wilson et al., 2006). Wilson et al. (2006) reported an average of 0.7 (90 % CI: 0.50-0.91) for I/O sulfate within 16 objects of the Vancouver Panel Study. This ratio matches very well the ratio obtained by Monn et al. (1997), which was obtained using another method.

5.7 Reported total personal (T), ambient (A) and non-ambient (N) exposure

According to Allen et al. (2004), ambient particles (*A*) accounted for 48 % (range: 21-80 %) of total personal exposure among sensitive populations in Seattle. This corresponds well with the ratio of *A* (8.1 µg PM2.5/m³; 90 % CI: 3.7-13.7 µg/m³) to *N* (10.5 µg/m³; CI: 0.5-29.7 µg/m³) in the study of Wilson et al. (2006). In the Research Triangle Park PM panel study, mean personal exposure among 38 participants amounted to 23.0 µg/m³ (Williams et al., 2003). Studies including smokers and non-smokers generally reveal that ETS influences *N* to the largest extent out of all indoor sources. Jedrychowski et al. (2005) could attribute 10 % of the variance in *T* among 407 Polish women to ETS. Only outdoor PM10 did explain more variance (31 %). Other indoor sources were of minor importance: 2 % variance was attributed to home heating by coal/wood stoves, another 2 % to other types of heating, and 1 % to proximity to industrial plant localization. Other investigated factors such as gas cooking and burning of candles were insignificant. In contrast, Sørensen et al. (2004) ascertained a 7.8 % increase in T per % increase in time exposed to candles burning. Environmental tobacco smoke caused an rise of 7.8 % in T per % increase in time exposed to ETS in that study.

In the study of Jedrychowski et al. (2005) on 407 Polish women, the personal exposure to PM2.5 was on average 18 µg/m³ higher if persons had coal/woodstove heating in their houses compared to no use of heating systems during air sampling. The corresponding elevation in PM2.5 due to electric/gas heating amounted to 10 µg PM2.5/m³ extra.