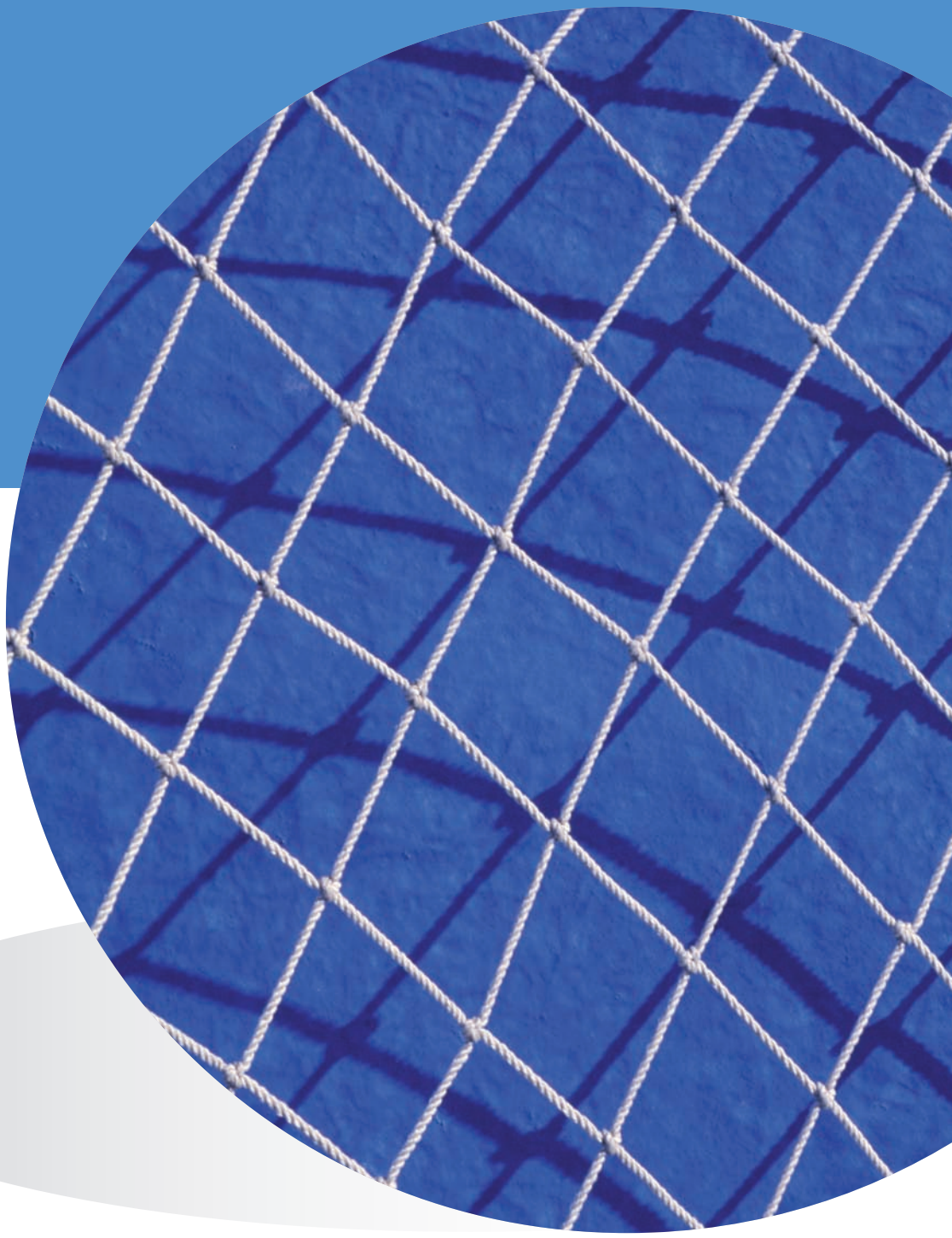
Otto Hänninen
Anne Knol
(Eds.)

# European Perspectives on Environmental Burden of Disease

## Estimates for Nine Stressors in Six European Countries

REPORT



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b Project coordination
c Preparation of Appendix B on ICT model
d Data management
e Uncertainty analysis
f Preparation of the first draft of the Methods chapter
g Preparation of the first draft of the Introduction chapter

*Coordinators of stressor work*

1 radon
2 ozone and particulate matter
3 lead
4 dioxins
5 benzene and formaldehyde
6 second hand smoke
7 transportation noise

# Executive summary

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The highest environment-related health benefits can be expected from policies that efficiently target environmental exposures having high contributions to the burden of disease (BoD) in the population. Such benefits are demonstrated for example by the smoking bans in public places that have shown significant population health improvements in many European countries. However, the health impacts of environmental stressors range from relatively mild psychological effects like annoyance to effects on morbidity such as asthma, cardiovascular diseases, cancer and premature mortality. This diversity of health effects challenges the comparison of the impacts of alternative policies.

The objectives of the multinational European EBoDE-project (Environmental Burden of Disease in the European region) included updating previous environmental burden of disease (EBD) assessments, identifying stressors relevant for the European region, testing a harmonized EBD methodology in the participating countries, and developing and making available the methodology for other countries. The project has assessed the environmental burden of disease related to nine selected stressors across six countries: Belgium, Finland, France, Germany, Italy and the Netherlands. The assessed stressors were: benzene, dioxins (including furans and dioxin-like PCBs), non-smokers exposure to second-hand smoke, formaldehyde, lead, transportation noise (including road, rail and air traffic), ozone, particulate matter ($PM_{2.5}$) and radon. The stressors were selected based on their public health relevance, potential for high individual risks, public concern and/or large economical impacts.

The environmental burden of disease is expressed in Disability Adjusted Life Years (DALYs), which are a summary measure of population health combining mortality and morbidity. Calculations were based on the most recent scientific evidence concerning population exposure and health effects, national exposure data and WHO burden of disease data.

Even though the most recent scientific knowledge and data were used, many uncertainties and controversies remain. Results give only a crude ranking of environmental health impacts and need to be interpreted with caution. The results suggest that 3–7% of the burden of disease[1] in the participating six countries is associated with the selected nine environmental stressors. Particulate matter (PM) is estimated to be associated with the highest disease burden (6 000 to 10 000 DALYs per million people[2]), followed by second-hand smoke, traffic noise and radon.

Burden of disease estimates quantify the attributable health impacts of environmental exposures. However, due to background exposures from natural sources and practical limitations in removing anthropogenic pollution, the total attributable burden of disease cannot be directly interpreted as reduction potential. EBD estimates can be used to identify areas of high disease burden for more detailed analysis of the reduction potential by targeted policies. Quantitative methods like EBD and health impact assessment should be used to inform policy makers about the health benefits of specific policy measures.

Keywords: Environmental burden of disease, disability adjusted life years (DALY), benzene, dioxins, second hand smoke, formaldehyde, lead, transportation noise, ozone, particulate matter ($PM_{2.5}$), radon, Belgium, Finland, France, Germany, Italy, The Netherlands

1 Discounted and age-weighed according to the standard procedure (WHO, 2010a).
2 Non-discounted and non-age-weighed values.

# Contents

# Abbreviations

| | |
|---|---|
| ALRI | Acute lower respiratory infection |
| AM | Arithmetic mean |
| BOD | Burden of disease |
| B-Pb | Blood lead concentration |
| COPD | Chronic obstructive pulmonary disease; group of related pulmonary diseases, most common form chronic bronchitis |
| DALY | Disability adjusted life year; burden of disease metric that combines years of life lost and years lived with disabilities (DALY=YLL+YLD) |
| dB(A) | Decibel (A-frequency weighing). Measure of noise level |
| DW | Disability weight, parameter used in defining magnitude associated with years lived with disability |
| EBD | Environmental burden of disease; BoD associated with defined environmental causes |
| END | European Noise Directive |
| ERF | Exposure-response function |
| GBD | Global Burden of Disease (WHO assessment) |
| GM | Geometric mean |
| GSD | Geometric standard deviation |
| HSD | High sleep disturbance; health endpoint associated with environmental noise exposures |
| HTD | Hypertensive disease |
| ICT | Impact calculation tool, probabilistic modelling tool run in Analytica environment |
| IHD | Ischaemic heart disease |
| IQ | Intelligence quotient |
| $L_{day}$ | Weighted noise level for daytime |
| $L_{den}$ | Weighted noise level for day-evening-night |
| LE | Life expectancy |
| $L_{night}$ | Weighted noise level for nighttime |
| LRS | Lower respiratory symptoms |
| MI | Myocardial infarction |
| MMR | Mild mental retardation |
| MRAD | Minor restricted activity days; health outcome defined in CAFE study for ozone |
| OR | Odds ratio |
| PAF | Population attributable fraction |
| PCB | Polychlorinated biphenyls, a chemical group of toxic compounds |
| $PM_{10}$ | Particulate matter with aerodynamic particle diameter smaller than 10 μm |
| $PM_{2.5}$ | Particulate matter with aerodynamic particle diameter smaller than 2.5 μm |
| RAD | Restricted activity days; health outcome defined in CAFE study for particulate matter |
| RR | Relative risk expressed either for the prevailing exposure level (in cohort studies) or normalized for a selected unit exposure, e.g. per 10 μg $m^{-3}$ |
| SD | Standard deviation |
| SHS | Second hand smoke; sometimes called also environmental tobacco smoke (ETS) |
| TEQ | Toxicity equivalent; a metric expressing the toxicity of a mixture of dioxins and PCBs as an equivalent amount of the most toxic compound, TCDD (original set of toxicity equivalency factors by WHO 1998, updated in 2005) |
| UR | Unit risk; population risk per an individual and unit exposure |
| YLD | Years lived with disability, a component of disease burden measured as DALYs |
| YLL | Years of life lost, a component of disease burden measured as DALYs |

# 1 Overview of the EBoDE-project

## 1.1 Introduction

Exposures to many environmental stressors are known to endanger human health. Negative impacts on health can range from mild psychological effects (e.g. noise annoyance), to effects on morbidity (such as asthma caused by exposure to air pollution), and to increased mortality (such as lung cancer provoked by radon exposure). Properly targeted and followed-up environmental health policies, such as the coal burning ban in Dublin (1990) and the smoking ban in public places in Rome (2005) have demonstrated significant and immediate population level reductions in deaths and diseases. In order to develop effective policy measures, quantitative information about the extent of health impacts of different environmental stressors is needed.

As demonstrated by the examples above, health effects of environmental factors often vary considerably with regard to their severity, duration and magnitude. This makes it difficult to compare different (environmental) health effects and to set priorities in health policies or research programs. Public health policies generally aim to allocate resources effectively for maximum health benefits while avoiding undue interference with other societal functions and human activities. In order to develop such policies, it is necessary to know what 'maximum health benefits' are. Decades ago, such decisions tended to be made based on mortality statistics: which (environmental) factor causes most deaths? However, nowadays, most people get relatively old, and priority has shifted from quantity to quality of life. This has lead to the need to incorporate morbidity effects into public health decisions, and therefore to find a way of comparing dissimilar health effects.

Such comparison and prioritisation of environmental health effects is made possible by expressing the diverging health effects in one unit: the environmental burden of disease (EBD). Environmental burden of disease figures express both mortality and morbidity effects in a population in one number. They quantify and summarize (environmental) health effects and can be used for:

- Comparative evaluation of environmental burden of disease ("how bad is it?")
- Evaluation of the effectiveness of environmental policies (largest reduction of disease burden)
- Estimation of the accumulation of exposures to environmental factors (for example in urban areas)
- Communication of health risks

An example of an integrated health measure that can be used to express the environmental burden of disease is the DALY (Disability Adjusted Life Years). DALYs combine information on quality and quantity of life. They give an indication of the (potential) number of healthy life years lost in a population due to premature mortality or morbidity, the latter being weighted for the severity of the disorder. The concept was first introduced by Murray and Lopez (1996) as part of the Global Burden of Disease study, which was launched by the World Bank. Since then, the World Health Organization (WHO) has endorsed the procedure, and the DALY approach has been used in various studies on a global, national and regional level.

WHO collects a vast set of data on the global burden of disease. The first study quantified the health effects of more than 100 diseases for eight regions of the world in 1990 (Murray and Lopez, 1996). It generated comprehensive and internally consistent estimates of mortality and morbidity by age, gender and region. In a former WHO study, it was shown that almost a quarter of all disease worldwide was caused by environmental exposure (Prüss-Üstün and Corvalán, 2006). In industrial sub-regions this estimate was about 16% (15–18%). These fractions, however, are dependent on the conclusiveness of the included environmental factors and health effects. The WHO programme on quantifying environmental health impacts has addressed more than a dozen stressors (http://www.who.int/quantifying_ehimpacts/publications/en/). In order to support further applications of the environmental burden of disease (EBD)

assessments, a methodological guidance has been published by WHO (Prüss-Üstün et al., 2003) and was followed here too.

In Europe, national environmental burden of disease (EBD) assessments are on-going in several countries. The work by RIVM was one of the first systematic European works in this area that utilized disability-adjusted life years (DALY) as a measure to compare the burden of different health outcomes related to the exposure of the population to environmental stressors (Hollander et al., 1999). The results highlighted that (i) a number of environmental stressors may cause chronic or acute diseases or death, (ii) a few top ranking stressors cause over 90% of the national EBD, and (iii) these top ranking stressors are not necessarily those that have drawn the most concern, regulatory action and/or preventive investment.

## 1.2 Objectives

The EBoDE-project was set up in order to guide environmental health policy making in the six participating countries (Belgium, Finland, France, Germany, Italy and the Netherlands) and potentially beyond. From a policy perspective, these insights from the EBoDE-project can be useful to evaluate past policies and to gain insight in setting the policy priorities for the future. We have calculated the total EBD associated with the nine environmental stressors. The total EBD is not identical to the avoidable burden of disease, because some exposures are not realistically reducible to zero (e.g. fine particles). Also, our estimates do not take into account the costs of reducing the EBD. Thus, the results are only one input into the full process of developing cost-effective policies to achieve better environmental health.

The objectives of the project were to update the available previous assessments, to focus on stressors relevant for the European region, to provide harmonized EBD assessments for participating countries, and to develop and make available the methodologies for further development and other countries.

The specific objectives are to:

- Provide harmonized environmental burden of disease (EBD) estimates for selected environmental stressors in the participating six countries;
- Test the methodologies in a harmonized way across the countries.
- Assess the comparability of the quantifications and ranking of the EBD
    - between countries
    - within countries
    - between environmental stressors;
- Qualitative assessments of variation and uncertainty in the input parameters and results.

Environmental burden of disease estimates have been calculated for:

- nine environmental stressors: benzene, dioxins (including furans and dioxin-like PCBs), second-hand smoke, formaldehyde, lead, noise, ozone, particulate matter (PM) and radon;
- six European countries: Belgium, Finland, France, Germany, Italy and the Netherlands;
- the year 2004 (and some trend estimates for the year 2010).

As outlined above, the EBoDE study was carried out in order to test the environmental burden of disease methodology in various countries. The results of the studies are intended to allow comparison of the disease burden between different environmental stressors and between countries. Consequently, the study does not to identify the 'reduction potential'. Our estimates should therefore not be interpreted as the 'avoidable burden of disease': most risks cannot realistically be completely removed by any policy measures. For some exposures, however, the numbers may nonetheless be interpretable as reduction potential, eg for dioxins, formaldehyde, benzene, etc, as these exposures could potentially be completely eliminated.

## 1.3 Outline of this report

This report describes the methods, data and results of the EBoDE-project. Chapter 2 presents the methodology. The environmental stressors are introduced in Chapter 3, which also presents the data used (selected health endpoints, exposure data, exposure response functions). In Chapter 4, the results are presented and discussed. Chapter 5 gives information about uncertainties in the approach, and provides some alternative calculations using different input values. In Chapter 6 conclusions are drawn. The report ends with the references and two appendices: Appendix A presents country-specific results and Appendix B some considerations for using a life-table approach in EBD modelling.

# 2 Methods: Environmental burden of disease calculation

This chapter provides information about methods to calculate the environmental burden of disease, and the assumptions and choices that need to be made. Table 2-1 provides an overview of these baseline assumptions underlying the calculations as performed in the EBoDE project. The remainder of this chapter describes the specific models used for calculating the EBD and explains the different parameters and data used.

TABLE 2-1. Baseline facts and assumptions underlying environmental burden of disease calculations as carried out in the EBoDE project.

| Parameter of assumptions | Choice made | Motivation | Remarks |
|---|---|---|---|
| Year | 2004 | Most recent year with relatively good data availability | Exposure trends were evaluated till 2010 for a qualitative policy analysis |
| Environmental stressors | Benzene, dioxins (including furans and dioxin-like PCBs), second-hand smoke, formaldehyde, lead, noise, ozone, particulate matter (PM) and radon | Based on selection criteria | See section 3.1 |
| Countries | Belgium, Finland, France, Germany, Italy and the Netherlands | Integration of national projects | EBoDE working group and methodology is open for other countries |
| Age weighing & discounting | Main results without discounting and age-weighing; alternative results with discounting (3%) and age-weighing (standard) | Ethical reasons. Supplementary discounted and age-weighed results presented for comparability with WHO estimates | See section 2.3 |
| Standard Life Expectancy | 80 years for men and 82.5 for women | Comparability with WHO estimates | See for exceptions section 2.2. |
| Lag time | Calculations carried out with and without lag times | For certain diseases there is a relatively long lag between exposure and the effect. When using discounting, the lag should be accounted for | Lag times are based on author judgement and serve as rough estimates (see section 3.11) |
| Uncertainty analyses | Qualitative and partly quantitative | It is essential to assess whether the substantial inherent uncertainties affect the order of magnitude of the results or the ranking of stressors | Data availability and limited resources allowed only for qualitative approach. Additional quantitative analyses are recommended as a part of follow-up research (see section 2.4 and Chapter 5) |
| Multi-causality | Not adjusted | Lack of knowledge/ data | See section 2.2 |
| Co-morbidity | Not adjusted | Lack of knowledge/ data | See section 2.2 |

# 2.1 Basic calculation of the environmental burden of disease

The DALY measures health gaps (i.e. years of life lost due to death or disability) as opposed to health expectancies. It measures the difference between a current situation and an ideal or alternative situation. The DALY combines the time lived with disability and the time lost due to premature mortality in one measure:

DALY = YLL + YLD

*where:*
YLL = Years of Life Lost due to premature mortality.
YLD = Years Lost due to Disability.

Years of Life Lost (YLL) in a case of individual death is calculated as the difference between the standard life expectancy at the age of death and the actual age at death. When population data is tabulated for age categories, YLL can be calculated as:

$L = LE(age_{death}, gender) - age_{death}$

The basic formula for calculating the population-wide YLL is:

YLL = N x L

Methods for calculating YLL are further described in section 2.2.

*where:*
LE(age,gender) = life-expectancy at age of death, accounting for gender
$age_{death}$ = age at death

N = number of deaths in a given age category
L = remaining years to standard life expectancy at age of death (in years).

To estimate the Years Lost due to Disability (YLD), the number of disability cases is multiplied by the average duration of the disease and a disability weight (see further discussion below). The basic formula is:

YLD = n x DW x L

*where:*
YLD = Years Lost due to Disability
n = number of incident cases.
DW = disability weight.
L = average duration of disability (years)

The formulas above describe undiscounted, non-weighted DALYs. DALYs are sometimes attributed a discounting rate, and weightings according to age, in which case the formulas become more complex. These so-called social preferences are discussed in section 2.3.

## 2.1.1 Disability weights (DW)

Disability weights are used to make different health effects with varying degrees of severity comparable. They are weight factors that reflect the severity of the disease on a scale from 0 (perfect health) to 1 (equivalent to death). These factors have been determined in expert panels using standardized surveys.

## 2.1.2 Duration estimates (L)

Besides disability weights, estimates of DALYs for morbidity (Years Lost due to Disability – YLD) also take into account the duration of the disease derived from health statistics, registries, expert judgments, etc. In some cases, prevalence data are used in burden of disease calculations instead of incidence data. In that case, the durationof disease is set to 1 year in the calculation, assuming a steady-state situation in which prevalence equals incidence times duration. For mortality, the duration estimate equals the YLL (Years of Life Lost, see section 2.2).

### 2.1.3 Models for estimating the environmental burden of disease

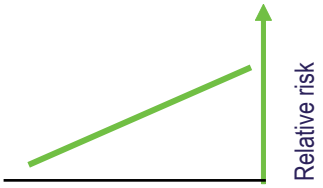
The general methodology for the environmental burden of disease calculations as carried out in EBoDE follows the Comparative Risk Assessment Approach (Ezzati et al., 2002; Prüss-Üstün et al. 2003). In general, information about population exposure, an exposure response function and (in some cases) background incidence data are needed in order to estimate the environmental burden of disease (EBD).

In EBoDE, three different methods for deriving the EBD are used. They are presented in Figures 2-1a and 2-1b (methods 1A, 2A and 2B). Model 1B is not used in EBoDE, but complements the other three methods. The methods differ in how they derive the population attributable fraction (using a unit risk (UR) or a relative risk (RR) – see Textbox 1), and in whether burden of disease figures are derived from the WHO database or estimated using disability weights (DW) and duration factors (L).

- Model 1A is the primary model used in EBoDE. Exposure data and a relative risk derived from epidemiological data are used to derive the population attributable fraction (PAF). This fraction is applied to the burden of disease figures as given in the WHO global burden of disease database.
- In model 2A, the PAF is derived indirectly. The unit risk and exposure information are used to estimate the attributable incidence (AI). The PAF is indirectly estimated from dividing the total incidence by this AI. Subsequently, the PAF is applied to the WHO burden of disease data for both YLL and YLD.
- In model 2B, the AI is derived similarly as in model 2A. However, for the factors for which this approach has been used, no appropriate burden of disease data were available from WHO and the EBD was calculated by multiplying the estimated number of attributable cases with WHO disability weights (DW) and corresponding estimates of duration (L).
- Model 1B, which was not used in EBoDE, could be used when the PAF is derived using a RR risk, and the EBD is calculated using disability weights (DW) and estimates of duration (L).

The conceptual basis of the different methods is the same. Which exact method is chosen for a specific calculation mainly depends on the available data. In principle, these different means should come to the same end. If one would use all different approaches for the same calculation, they would ideally result in the same number. However, in reality it is hardly ever possible to perform all these different calculations because of unavailability of data. Even if possible, the different methods will rarely result in the same number. This stresses the importance of interpreting burden of disease figures as crude ranges and not as absolute infallible numbers. More information about uncertainty is presented in section 2.4 and in Chapter 5.

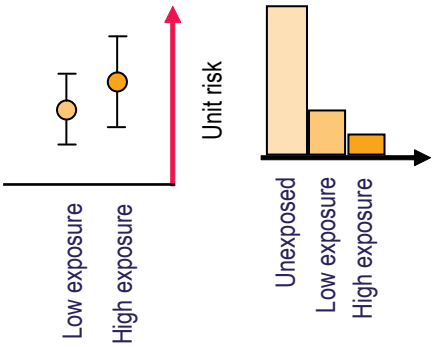
**Textbox 1: Relative Risk, Unit Risk and Odds Ratio**




Exposure response functions can take many forms, dependent on the type of relation between the exposure and the health effect, and the type of study used to derive it. In EBoDE, we use relative risks (RR), unit risks (UR) and some more complex functions (not further discussed here).

The RR* is defined as the risk of developing a disease (the event) relative to exposure, expressed as the ratio of the probability of the event occurring in the exposed group versus a non-exposed group

$$RR = \frac{P\text{exposed}}{P\text{non-exposed}}$$




The UR gives the absolute number of cases that are to be expected at a certain exposure, and can be derived for effects which are independent of the background disease rate. The number of cases is directly estimated based on exposure (number of people exposed) multiplied by the unit risk estimate.

Odds ratio is the ratio of the odds of a disease occurring in the exposed group to the odds of it occurring in the non-exposed group. OR is typically estimated in epidemiological studies, where the study groups are selected by their exposure status and thus the ratio of exposed does not directly reflect the corresponding fraction in the general population. However, when the disease prevalence in the non-exposed population is relatively low (<10 %), OR can be used as an estimate of the relative risk (RR).

* The definition for RR provided here is valid for the prevailing exposure levels. In contrast, most of the RR values used in the calculations are expressed per selected unit exposure, e.g. 10 μg $m^{-3}$.

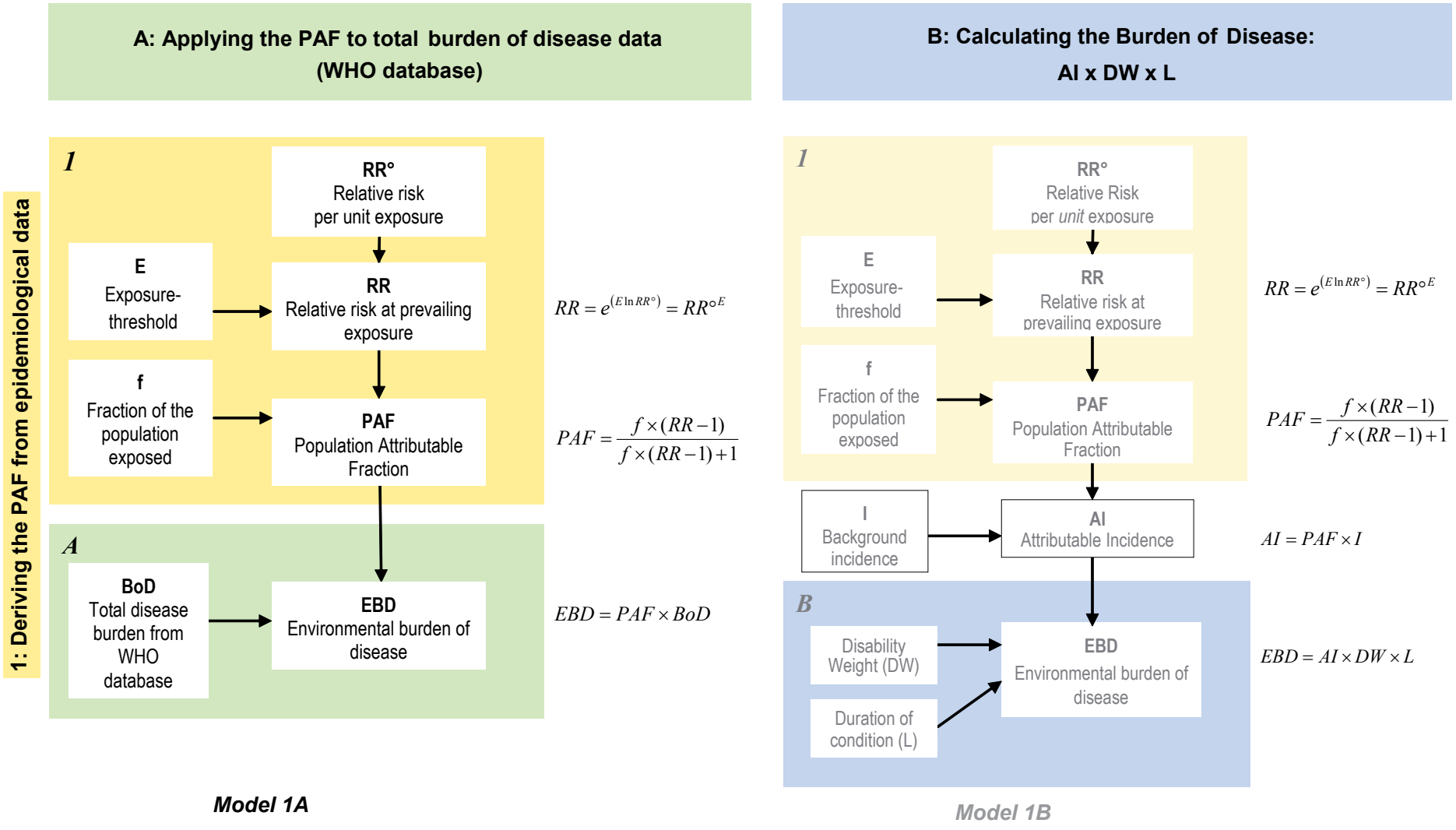



FIGURE 2-1a. Relative risk models to estimate the environmental burden of disease. Model 1b (greyed) is not used in EBoDE.

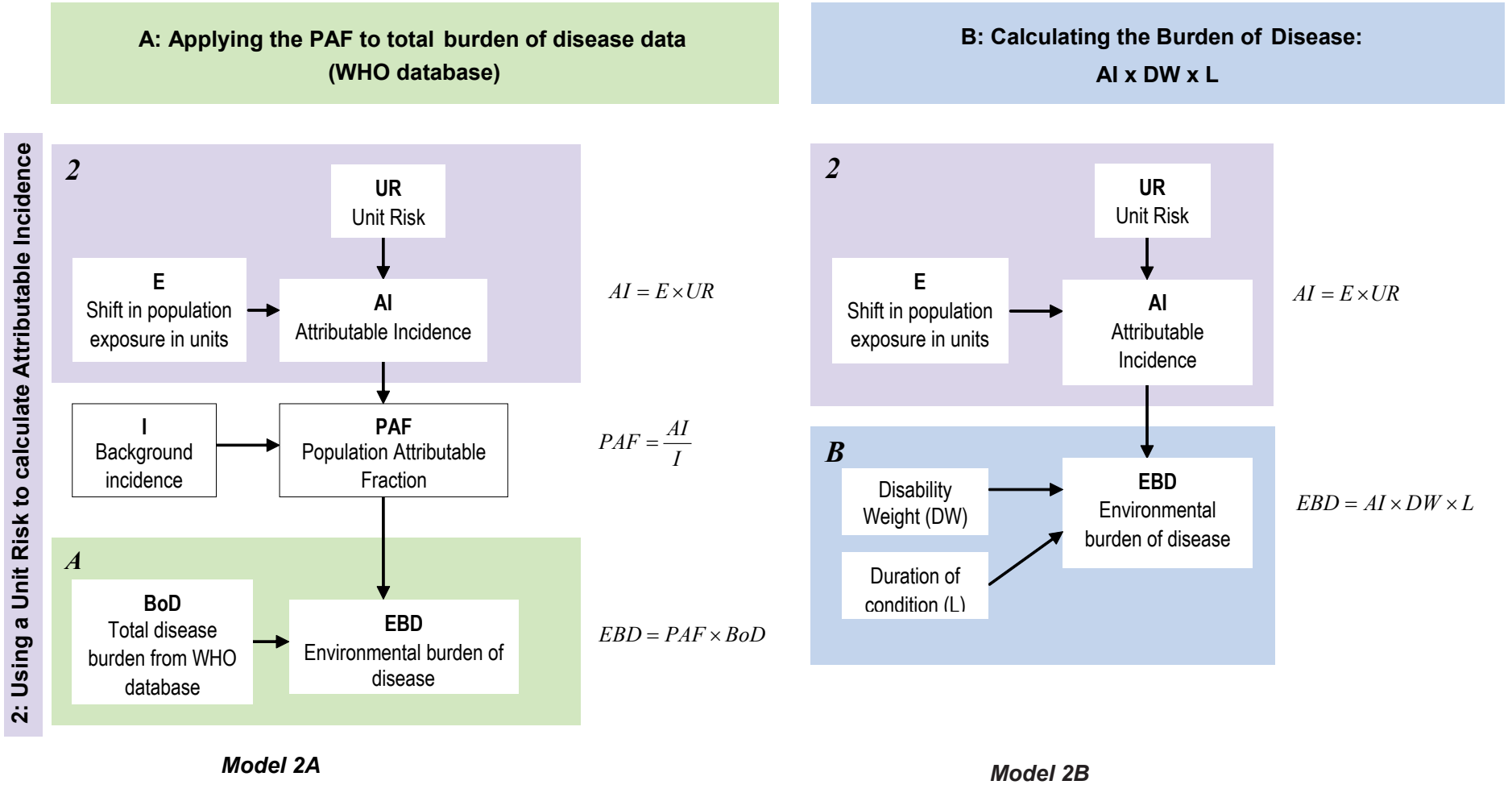
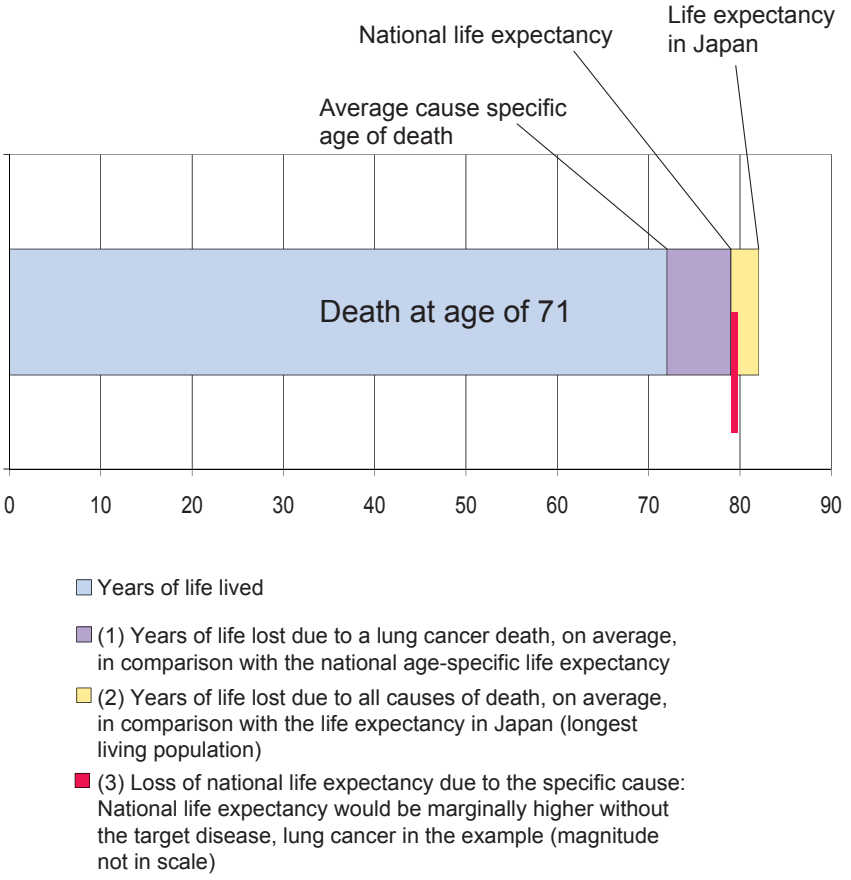



FIGURE 2-1b. Unit risk models to estimate the environmental burden of disease.

## 2.2 Years of Life Lost, co-morbidity and multi-causality

DALY calculations for mortality outcomes include an estimate of the Years of Life Lost, i.e. the number of years a person would have continued to live, had this person not died due to the environmental exposure. In the WHO Global Burden of Disease programme and in the current work a standard life expectancy, defined as a population with highest known life-expectancy, is used. This ensures that all populations globally are treated equally when addressing the disease burden.

However, in the current context where the focus is set on specific environmental causes of burden of disease, it can be argued that if all environmental causes of burden of disease were removed, the population in question still would not reach this optimal life expectancy. This is due to the fact that all factors affecting the population health (including also genetic factors, lifestyle, the health care system, etc.) contribute to the national life expectancy. Thus it could be argued that a national life expectancy should be used in estimating the impacts of given single factors. This difference in approaches is highlighted in Figure 2-2. In the EBoDE project we have chosen to use the standard life expectancy in order to allow for better comparison between countries.Figure 2-2. Estimating the years of life lost due to lung cancer.




### 2.2.1 Co-morbidity

Co-morbidity can play a role in estimating DALYs for morbidity. In the case of co-morbidity, people are not only affected by the disease under scrutiny, but are also weakened by other conditions. In industrialised countries, older people often have more than one disease. Severity weights do not take account of these co-morbid conditions (Gold et al., 2001). The disease burden is disease-specific and not individual specific, so adding up the severity weights for all diseases in a person could result in a weight of more than one, representing a state worse than death (Anand and Hanson, 1997; Schneider, 2001). Effects of co-morbidity can be relevant when looking at one person with several diseases. However, when estimating the burden of disease for a complete population, the effect of co-morbidity is not very influential.

### 2.2.2 Multi-causality

Multi-causality means that people have a single disease, but that this disease is caused by multiple factors. People may for example have lung cancer due to a combined effect of radon and smoking. If the exposure to radon would be removed, part of the lung cancer cases could be prevented. However, partly the same cases could in principle be prevented by quitting smoking. Because of this effect, which is called multi-causality, the estimated attributable fractions of these two separate causes for lung cancer are potentially additive to more than 100%. Therefore, there may be overlap in the estimated disease burdens of these two factors and they can not be summated without correction for this overlap. In case of mortality, this concept is also referred to as 'competing causes of death'.

Correcting for multi-causality is most important when a significant number of cases are indeed caused by several of the addressed risk factors. It is however difficult to estimate exact effect of multi-causality, as the underlying epidemiology is lacking in many instances. In this project, multi-causality may affect the health impacts related to the joint exposure to outdoor air pollution and second-hand smoke, or to second-hand smoke and radon. The potential effect of multi-causality and overlapping health endpoints is not corrected for in our estimates of the burden of disease, but is discussed in the respective chapters. The stressor-specific figures should be interpreted as the burden of disease that could theoretically be prevented if the specific risk factor was removed.

## 2.3 Discounting, age-weighing and lag times

The environmental burden of disease estimated in EBoDE as DALYs are expressed in three alternative metrics. The differentiating weighing factors are described shortly in this chapter. The three metrics we used are:

- non-discounted, non age-weighted DALYs (i.e. health impacts occurring later in the future are counted with similar weight as immediate effects; health effects are weighted the same at all ages; no lag times are included)
- discounted and age-weighted DALYs (i.e. future health impacts are brought to present value assuming a constant discount rate of 3%; health impacts in older and younger people are age-weighted using the standard WHO procedure; no lag times are included)
- discounted and age-weighted DALYs with lag (i.e. future health impacts are brought to present value assuming a constant discount rate of 3%; health impacts in older and younger people are age-weighted and the delay from current exposure to the manifestation of the associated disease, e.g. cancer (lag) is included in the discounting procedure).

### 2.3.1 Discounting

When DALYs are discounted, future years of healthy life are valued less than present years. Discounting is based on the fact that people generally seem to prefer a healthy year of life immediately over a year of life lived in the future. In its Global Burden of Disease study, WHO applies a 3% time discount rate to years of life lost in the future. This means that a year of healthy life gained 10 years from now is worth 24% less than a year gained now. The use of discount rates can also be debated. Applying discounting to burden of disease figures is not favourable for children and future generations (Anand and Hanson, 1997; Arnesen and Nord, 1999) and preventive measures are devalued, as they cost money now while benefits will become apparent later (Schneider, 2001).

### 2.3.2 Age weighing

In the GBD study as done by WHO, a year of healthy life lived at younger and older ages is weighted lower than for other ages (see Figure 2-3). The motivation for such age-weighing is a number of studies that have indicated a social preference to value a year lived by a young adult more highly than a year lived by a young child, or lived at older ages. The social value of middle-age groups is considered to be greater,

due to responsibility for their dependants, than the value of younger or older people. However, the use of age weights is highly controversial. Some critics state that it is unethical to value the lives of children and elderly less than other lives (Arnesen and Nord, 1999; Anand and Hanson, 1997; Schneider, 2001).

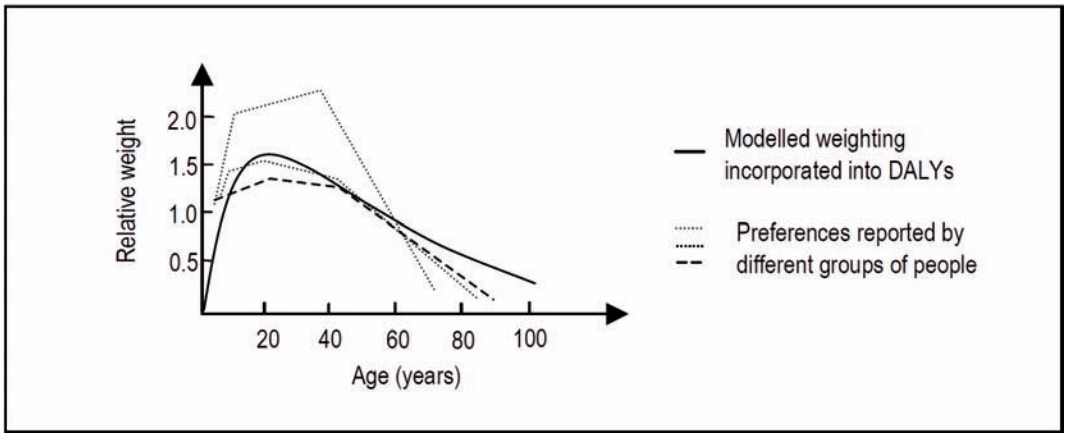



FIGURE 2-3. Relative value of a year of life lived, by age: reported preferences and modelling. A 3% discount rate is used by WHO. (Adapted from Figure 3.1 in the WHO document. http://www.who.int/quantifying_ehimpacts/publications/en/9241546204chap3.pdf)

### 2.3.3 Discounting and age-weighing in EBoDE

The choices made for discounting, age weighing and the severity weights chosen can lead to large differences in DALYs. Alternative but still realistic assumptions for all these parameters can lead to chances up to a factor of four (Arnesen & Kapiriri, 2004). In recognition of the non-favourable aspects of age-weighing and discounting as outlined above, our main results are presented without any discounting or age-weighing. However, in order to make EBoDE results comparable to other WHO burden of disease estimates, we have also calculated our results using age-weighing and discounting (3%).

### 2.3.4 Lag times

Certain health impacts like cancer and chronic diseases develop slowly and the outbreak of the disease occurs years or decades later than the exposure associated with it. These lag-times are not commonly included in burden of disease calculations even though when discounting is used, any delays in the impact will affect also the discounted present value of the impact.

To complement the standard discounting approach in the EBoDE project, we have tested the effect of lag-times on discounted DALYs. Only the effect of increased discounting due to lag-times is included (so changing population dynamics over time are not taken into account). The lag times used in EBoDE are presented in the Data chapter (section 3.11).

## 2.4 Uncertainty analysis

Many factors contribute to the uncertainty in burden of disease estimates. Besides uncertainties caused by differences or inconsistencies in the methodological approaches and assumptions discussed above, also the basic data on population exposures contain uncertainties. In addition, our knowledge about environmental health impacts is incomplete, and a variety of assumptions need to be made, including assumptions about causality and exposure-response relationships. For the formal discussion of these factors we will use the framework presented by Knol, 2009, and distinguish between context uncertainty (i.e. the boundaries of the assessment, the definitions used, the selected stressors and health endpoints, etc), model structure uncertainty (i.e. for example uncertainty about causality) and parameter and input data uncertainty (i.e. confidence intervals of exposure response functions or inconsistent health statistics).

In EBoDE, qualitative and partly quantitative methods were used to estimate the impact of various sources of uncertainty in the estimates. The following sources of uncertainties were taken into account:

*Context uncertainty:*
- Selection of exposure metric
  - approximating particulate matter exposures with $PM_{2.5}$ or $PM_{10}$
  - indoor versus outdoor concentrations versus personal exposure versus doses
- Selection of health endpoints
  - Dioxins: total cancers versus more specific health endpoints
  - Lead: hypertension versus cardiovascular endpoints
  - Road transport noise: Myocardial Infarction versus Ischemic Heart Disease
- Estimation of policies and trends from 2004 to 2010

*Model structure uncertainty:*
- Comparison between alternative model approaches
  - Unit risk and Relative Risk models for Radon
  - Formaldehyde: models for asthma and cancer using different thresholds
  - Probabilistic versus deterministic modelling (ICT vs. Excel, see section 2.5 and Appendix B)

*Input data and parameter uncertainty:*
- Statistical confidence intervals for the exposure-response model parameters
- Deficiencies in the representativity of the noise exposure data
- Qualitative analysis of population representativity of exposure data
- Temporal representativity of the exposure data (e.g. lead estimation from data from 1990's; SHS trend model)
- Comparison of $PM_{2.5}$ and ozone models with earlier CAFE estimates (exposure data updated using new estimation methods)
- Estimation of noise $L_{night}$ levels from $L_{den}$ levels (see section 3.7): comparison for countries with both variables reported

The impact of the uncertainties on the results is discussed in Chapter 5, which also presents a table with the most important sources of uncertainty for each stressor. More thorough uncertainty and sensitivity analysis is recommended for the potential follow-up work using for example probabilistic modelling, such as the Impact Calculation Tool (see the Appendix B).

## 2.5 Software

Model calculations were completed in a number of Excel models based on deterministic point value estimates of the various input parameters. Variability and uncertainty calculations were conducted using 95% confidence intervals of the exposure-response relationships. Exposure distribution estimates (for formaldehyde and lead) were conducted using Risk 4.0 (Palisade Corp., NY) simulation and probabilistic distribution calculations using the Excel worksheet functions for normal distributions.

The Excel sheets used in the EBoDE project do not contain distributional uncertainty or sensitivity analyses. In addition, they are not using life tables in order to model changes in population demographics that may affect health effects in the future. In Appendix B we discuss the use of a probabilistic model called Impact Calculation Tool (ICT), developed in collaboration by THL and RIVM, which includes many of the above mentioned features. It was not yet fully developed when EBoDE calculations were carried out, but can be used for future health impact assessment studies. Some test calculations were performed and the comparisons did not indicate changes in the main conclusions and recommendations. Use of life-

tables would require more elaborate definitions of the causal associations, e.g. the duration from exposure to outbreak of the disease (the lag-time) that were modelled here with simpler methods.

For selected stressors, EBoDE results were compared with results obtained using the ICT, thereby comparing a probabilistic life-table approach with deterministic point value calculations. The two approaches are compared in Appendix B.

# 3 Selected exposures and health effects

This chapter presents, for each stressor, the health effects included in our analyses, exposure response functions that were used and the exposure data. Table 3-19 presents an overview of the selected environmental stressors, health endpoints, exposure-response functions and methods used. An overview of exposure data used for each stressor is given in Table 3-20.

## 3.1 Selection criteria

### 3.1.1 Environmental stressors

We aimed to study the burden of disease in the general population associated with stressors in the physical environment. Occupational hazards and risks associated with lifestyles (e.g. alcohol use, active smoking, nutrition), as well as infectious diseases, were excluded from the assessment.

Four criteria were defined for selection of environmental stressors to be included in the study:
- Public health impact;
- High individual risk;
- High political or public concern;
- Economic significance.

In addition, the selection was affected by the feasibility of the calculation. Therefore, we also considered:
- availability of exposure data
- availability of evidence-based exposure response function(s)
- availability of baseline health statistics.

Discussion among environmental health experts as represented in the EBoDE working group selected the environmental stressors based on these selection criteria. A first list of stressors was divided into two parts:
- a high priority list of stressors, which either scored high on many of the criteria and/or which were relatively easy to calculate
- a medium priority list of stressors.

| High priority list of environmental stressors | Medium priority list of environmental stressors: |
|---|---|
| Benzene<br>Dioxins (including furans and dioxin like PCBs)<br>Second-hand smoke (SHS)<br>Formaldehyde<br>Lead<br>Transport noise<br>Ozone<br>Particulate matter<br>Radon | 1,2-Dichloroethane<br>Accidents - domestic<br>Accidents - traffic<br>Acrylamide<br>Arsenic<br>Chlorination by-products<br>Carbon monoxide (CO)<br>Damp housing<br>Foodborn epidemics<br>Indoor insecticides<br>Methyl mercury<br>UV radiation<br>Waterborne epidemics |

The pilot project, which is described in this report, only included the stressors on the high priority list. These stressors will be shortly introduced in paragraphs 3.2 to 3.10. This list represents only a limited number of environmental stressors, and therefore the results of this study cannot be interpreted as estimates of the complete environmental portion of the total burden of disease.

The stressors on the medium priority list are candidates for addressing in subsequent studies.

### 3.1.2 Health outcomes

For every environmental factor, a set of health endpoints had to be selected which are causally linked to the exposure of interest. Only health effects that are included in the International Statistical Classification of Diseases and Related Health Problems (ICD) were selected. Therefore, wellbeing effects and for example 'noise annoyance' were not included.

Within that definition of health, the health endpoints were selected based on the following criteria:

- "sufficient" evidence for a causal relationship between exposure to the environmental stressor and the health effect
- "sufficient" evidence that the health effect is substantive enough to have an impact on the burden of disease estimate
- sufficient data to carry out the calculations (burden of disease data, exposure-response functions).

For some stressors, the exclusion of health endpoints with insufficient evidence may have led to underestimation of the results, for example for lead and dioxins. On the other hand for dioxins the selection of total cancer as the modelled health endpoint and assuming all cancer cases lethal may lead to overestimation (see also chapters about the individual stressors and the discussion on uncertainty in Chapter 5).

The health endpoints considered in this project and the corresponding exposure-response functions are summarized in Table 3-19 in section 3.12.

### 3.1.3 Exposure-response functions

For each combination of environmental stressors and health endpoints, exposure-response functions were selected from:

- International recent meta-analyses or WHO guidelines
- If not available: individual high quality studies

### 3.1.4 Exposure data

Exposure data were as much as possible collected from international harmonized and validated sources. If such data were not available, national data sources were used. In such cases, national data needed to characterize the population exposures in a representative and comparable manner, accounting for potential differences in the urban and rural exposures, different age groups, gender and other relevant sub-groups.

International exposure data were used for SHS, transport noise, ozone, PM and radon. National data were used for benzene, dioxins, formaldehyde and lead, with complementary information from (non-comprehensive) international data sources used when available (AirBase ambient data for benzene; several international multicenter studies for indoor concentrations of benzene and formaldehyde covering some of the participating countries, and WHO Mother's milk database for dioxins). The sources of exposure data are summarized in Table 3-20 in section 3.12. Exposure data for the target year 2004 are presented in Table 3-21 in the same paragraph.

The exposure trends for the year 2010 were estimated using existing data and author judgment to facilitate the evaluation existing policies in the light of the impact estimates for 2004. For several stressors (e.g. lead, dioxins) not enough data were available to make sensible trend estimates. For other stressors (PM, ozone, benzene), temporal and/or spatial variability was so large that reliable evaluations of the trends on the basis of these data were not possible. In these cases, expert judgment was used to estimate trends and corresponding confidence intervals. The estimated trends are summarized in Table 4-4. Due to the large uncertainties no national trend estimates were created.

# 3.2 Benzene

## 3.2.1 About benzene

Benzene is an organic chemical compound that was added to gasoline in the past. The use of benzene as an additive in gasoline is now limited, but it is still used by industry in the production of for example drugs and plastics. In addition, cigarette smoke contains some benzene.

Inhalation is the major route of human exposure to benzene. However, exposure may also occur through oral absorption or by dermal exposure (primarily in workplace settings). Exposure to benzene-contaminated water can cause inhalation and dermal absorption in the general population (e.g. when having a shower), but this does not occur often (US Department of Health, 2007).

The genotoxicity of benzene has been extensively studied. Benzene is a known carcinogen for which no safe level of exposure can be recommended. The most significant adverse effects from prolonged exposure to benzene are haematotoxicity, genotoxicity and carcinogenicity (IARC group 1 carcinogen) (IARC 1982, 1987). Chronic benzene exposure can result in bone marrow depression expressed as leukopenia, anaemia and/or thrombocytopenia, which can in turn lead to pancytopenia and aplastic anaemia (WHO, 2000b). Increased mortality from leukaemia has repeatedly been demonstrated in workers occupationally exposed (Arp et al 1983, IARC 1982, Decouflé et al 1983, Bond et al 1986, McCraw, 1985, Yin 1987, Paxton et al. 1994a, b). There are also studies that using proxies of benzene exposure indicate an increased risk of leukaemia in children, but conclusions are not definitive (Weng et al, 2009, Brosselin et al, 2009, Whitworth et al 2008, Gunier et al 2008, Steffen et al, 2004, Crosignani et al, 2004, Pearson et al, 2000, Nordlinder et al, 1997).

Benzene was selected in the EBoDE project because it may pose high individual risks and is still of global concern. Even though policies in Europe have already greatly reduced environmental benzene exposure, it is still identified as a concern (e.g. the INDEX project identified benzene as high priority stressor (Koistinen et al., 2008, Kotzias et al., 2005); European air quality directive 2008/50/EC; setting of WHO guidelines for indoor air quality (WHO, 2010b)).

## 3.2.2 Selected health endpoints and exposure-response functions

Benzene effects were estimated for leukaemia, including morbidity and mortality. Other proposed health endpoints were not included, because they only occur at high exposure levels, typical of occupational settings. We used the exposure response function as recommended by the WHO Air Quality Guidelines (WHO, 2000b) (see Table 3-19 in section 3.12). WHO uses the 1984 risk calculation of Crump (1984), in which the geometric mean of the range of estimates of the excess lifetime risk of leukaemia at an air concentration of 1 µg/m3 is estimated to be $6 \times 10^{-6}$ (unit risk). This estimate falls within the range of the risk estimate that is used by the US EPA ($2.2 \times 10^{-6}$ to $7.8 \times 10^{-6}$ per µg $m^{-3}$). This unit risk is applied to the whole population, including children. Specific estimates that have been supplied for children could not be used, because the underlying studies often use proxies of exposure (petrol station density, traffic density, etc.) instead of actual benzene exposure levels.

The estimated number of leukaemia cases were used to calculate the population attributable fraction using method 2A.

## 3.2.3 Exposure data

Benzene exposures are best described by residential indoor air levels (µg $m^{-3}$). Besides being affected by benzene levels in outdoor air, indoor levels may be raised especially by indoor smoking and potentially the storage and use of fuels e.g. in case of attached garages and storage rooms.

Benzene is a regulated ambient pollutant and therefore outdoor monitoring is required by the European Union. Benzene measurements are included in the AirBase database (European Environment Agency, AirBase, 2009).

Benzene exposure is estimated from national indoor levels, supplemented with outdoor levels. Different national data demonstrate that benzene exposure concentrations vary from 0.9 µg $m^{-3}$ in the

Netherlands to 2.9 μg $m^{-3}$ in Italy. The data used in this project are summarized in Table 3-21 in section 3.12.

The confidence levels of the exposure data cannot be directly compared, because the measurements are based on different time periods. Data from the Netherlands and France reflect a 1 week average exposure, while Italian and Finnish data are based on 2 day measurements.

Sources of uncertainty in exposure data include differences in sampling selection. In France, data reflect a large number of dwellings, while in other countries data are limited to a smaller number of monitored houses. In addition, the presence or absence of tobacco smoke in indoor environments is not always reported, making comparison more difficult. This at least partly explains the higher levels in Finland, where benzene from smoking was included. In Italy, levels are likely to be higher because of the large number of two-stroke engines used there, which emit a lot of benzene.

TABLE 3-1. Characteristics of benzene indoor concentration measurements.

| Country | Including benzene from smoking | Sample size | Time period of measurements |
|---|---|---|---|
| Belgium | Yes | 85 houses and 25 day-care centres | |
| Finland | Yes | random; 20 adults | 2 day average |
| France | Yes | 567 residences | 1 week average |
| Germany | Yes | 1790 subjects | |
| Italy | Yes | 50 subjects | 2 day average |
| Netherlands | Yes | 1240 dwellings | 1 week average |

## 3.3 Dioxins (including furans and dioxin-like PCBs)

### 3.3.1 About dioxins (including furans and dioxin-like PCBs)

Dioxins (including furans and dioxin-like PCBs) are a group of polychlorinated organic compounds with the same toxic mechanism. They are by-products of various industrial processes and combustion activities and are considered to be highly toxic.

Dioxins and dioxin-like PCBs are quantified by toxic equivalents (TEQs) representing the total toxicity compared to the most toxic compound, 2,3,7,8-Tetrachlorodibenzodioxin (TCDD). The power of toxicity is calculated with Toxic Equivalent Factors (TEFs), which allow the toxic potentials of each compound to be added up, in order to derive the TEQ of the mixture. Acute toxicity, leading for example to chlorakne or alteration of liver function, is only expected at very high doses. Long-term exposure to dioxins has been linked to effects on the immune system, the nervous system, the endocrine system and reproductive functions and is also known to cause tooth and bone defects, diabetes as well as several types of cancer (USEPA, 2003). The association between dioxins and cancer has been most consistent for non-Hodgkin's lymphoma. IARC classified TCDD (2,3,7,8-Tetrachlorodibenzo-p-dioxin), as a "known human carcinogen" (IARC, 1997). All other dioxin-like compounds are classified as "likely to be carcinogenic to humans".

This group of chemicals is selected in EBoDE because of their high toxicity and potential troubling exposures through e.g. mothers milk.

### 3.3.2 Selected health endpoints and exposure-response functions

In EBoDE, we have quantified the effect of exposure to dioxins and dioxin-like PCBs on cancer (all cancer types, mortality only). The non-fatal and non-cancer effects were not suited for health impact assessments due to difficulties in estimating the exposure-response relationships and the other input parameters

necessary for estimating DALYs. Therefore, our estimates may underestimate the true dioxin-related burden of disease.

Leino et al. (2008) assumed a linear exposure-response relationship for excess cancers associated with dioxin intake. They estimated the health risk for toxicity equivalent intake assuming additivity of the toxicity of the different types of dioxins and all cancer cases to be lethal.

The EBoDE calculations use the Leino et al. (2008) approach, but the results have been corrected with an updated cancer slope factor $1\times10^{-3}$ per pg/kg/d of dioxin intake of the U.S. Environmental Protection Agency (USEPA, 2003; NAS, 2006). The assumption that all cancers are lethal may lead to overestimation of the impacts.

The health endpoints considered in this project for dioxins and the corresponding exposure-response functions are summarized in Table 3-19 in section 3.12. YLD estimates in the table are based on the attributable fraction derived from the ERF using method 2A (see Figure 2-1), which is applied to the total YLD for all cancers as represented in the WHO database.

### 3.3.3 Exposure data

Dioxins and dioxin-like PCBs are persistent and bio-accumulating. The main exposure route for these chemicals is animal fat in nutrition, which accounts for about 90% of all exposure. Other routes, such as inhalation, play a minor role.

In order to estimate health effects related to dioxin exposure, daily intake data were needed. This intake depends on eating habits, age, gender, body weight and food consumption. Often, breast feeding contributes to the highest intake of dioxins for humans in their life. Dioxins have a long half life. Therefore the development of health effects in humans depends not only on the daily intake, but also on the body burden accumulated over years. On average, the daily intake of dioxins and dioxin-like PCBs decreases, while the body burden increases with age.

The cancer slope factor is expressed for daily intake of adults. There are different ways to measure the daily intake, each with different limitations. Table 3-2 describes some different measurement methods and provides short information about their use and limitations.

TABLE 3-2. Different ways to measure daily intake of dioxins and dioxin-like PCBs.

| | Type of measurement | Type of use | Specific limitations and uncertainties |
|---|---|---|---|
| A | Survey (questionnaire) on food consumption | Information on food consumption and about the content of dioxins in representative food samples allow modelling of daily intake | Results are modelled for an average population - food contamination and eating habits can differ on a large scale |
| B | Total diet studies | The total diet in a population group over a certain time period and dioxin in this food or representative food samples are measured. | Results are only relevant for the investigated groups and not necessarily representative for the whole population, sampling period influence the results. |
| C | Human biomonitoring Investigation of human milk or blood levels | Analyses of samples can show the body burden. Experimental scaling is used to convert observed biomonitoring results (blood) into daily intakes. | D-R function is based on daily intake. Human milk or blood samples are not widely available. Different fat content of the bodies influences the results. |

In addition, in all these studies different compounds can be measured:

(i) Only dioxins and furans;
(ii) dioxins, furans; and dioxin-like PCBs
(iii) dioxins, furans and dioxin-like PCBs as well as all other dioxin-like compounds detected as dioxin-like activity, expressed as TEQ in Bioassays (e.g. CALLUX).

In the EBoDE project, we have used national exposure data because there is no international comparable data source available. The different countries have used different methods to derive the daily intake values.

Table 3-3 provides a summary of the data and sources for dioxin. The specific data used in this project are summarized in Table 3-21 in section 3.12.

For the EBoDE project daily intake data are expressed as Toxic Equivalent (TEQ), estimated using the Toxic Equivalent Factors (TEFs) as provided by WHO (Van den Berg et al. 1998). Even though later TEFs exist (Van den Berg et al., 2006; http://www.who.int/ipcs/assessment/tef_update/en/), we used the results of the 1998 review, because most available data have been calculated using these TEFs.

TABLE 3-3.: Summary of the sources of dioxin data. Explanation for A, B C see Table 3-2.

| Countries | Population groups | Source | Sampling years | Compounds measured | Dioxin intake 2004 pg/kg bw/d |
|---|---|---|---|---|---|
| Belgium (A) | female 18-44 y<br>adults 50-65 y<br>adults | Bilau 2008<br>Bilau 2008<br>Calculated mean | 2002–2006 | Calux-all dioxin-like compounds[1] | 2.1<br>1.7<br>1.9 (mean) |
| Finland (A) | all | Kiviranta et al 2005 | 2002 | Dioxins+PCB | 1.5 |
| France (C) | 30–65 y | Fréry et al. 2006 | 2004 | Dioxins+PCB | 2.3[2] |
| Germany (A) | adults | Umweltbundesamt 2005 | 2003 | Dioxins+PCB | 2.0 |
| Italy (A) | 13–94 y | Fattore et al 2006 | 1997–2003 | Dioxins+PCB | 2.3[3] |
| Netherlands (A+B) | adults | De Mul 2008 | 2004 | Dioxins+PCB | 1.0[4] |

[1] Belgium – Dioxin and all dioxin-like compounds are measured with Bioassay, only the sum of all dioxin-like compounds is given; the daily intake was calculated as mean of the 2 adult groups.
[2] France – daily intake calculated based on blood concentration of 27.7 WHO-TEQ pg/g blood fat.
[3] Italy – daily intake were calculated using, for most dioxin and DL-PCB concentration data, a database available from the European Commission (Gallani et al., 2004).
[4] Netherlands – Values in the study were calculated using TEFs from 2005. For comparability, we have adjusted the values as presented by Mul et al (2008) by adapting the results to TEF 1998 adding 10%.

We have only used data on the daily intake of adults. We have chosen to do so, because the daily intake differs substantially between different age groups. The highest intakes are calculated for breastfed babies (about 50 to 100 WHO-TEQ pg/kg bw/d). Children have a higher intake than adults because of the different proportion between body weight and food intake and their different food habits (children take more milk and dairy products). Since there are only very few data for children available, we have limited ourselves to adults.

Due to the differences in measurement approach, it is difficult to compare dioxin intake numbers between countries. As a form of quality assurance, we have compared our daily intake estimates of dioxins and dioxin-like PCBs to international data on dioxins and PCBs in mother's milk (milk data from 2001–2003) as provided by WHO in the ENHIS-database (WHO, 2007a) and from Malisch and Leeuwen (2003). In principle, the ratio between the estimated daily intakes and the levels of mother's milk should be roughly similar between countries. The ratios are presented in Table 3-4. As can be seen from this table, the ratios are relatively similar across the countries, except in the Netherlands, where the intake level seems to be somewhat lower than in the other countries in comparison with the mother's milk levels. We have not corrected for this difference in the EBoDE calculations, as the causes for the difference are yet unknown.

TABLE 3-4. Comparison of dioxins and PCBs human milk (WHO, 2007a) and the estimated daily intakes (country-specific results – see Table 3-3.

| Country[a)] | Human milk ng TEQ/kg fat | Daily intake pg TEQ/kg bw/d | Factor milk/intake |
|---|---|---|---|
| Belgium | 29.5 | 1.9 | 16 |
| Finland | 15.3 | 1.5 | 10 |
| Germany | 26.2 | 2.0 | 13 |
| Italy | 29.0 | 2.3 | 13 |
| Netherlands | 29.8 | 1.0 | 30 |

[a)] France was not included in the WHO-milk study.

## 3.4 Second-hand smoke

### 3.4.1 About second-hand smoke

Second-hand smoke (SHS; also called environmental tobacco smoke or passive smoking) is a known human carcinogen (IARC, 2004). Exposure to SHS has been shown to cause lung cancer, IHD (ischemic heart disease) sudden infant death syndrome, asthma, lower respiratory infections in young children, low birth weight, reduced pulmonary function among children, acute otitis media, and acute irritant symptoms (WHO, 1999; Californian EPA 2005; US Surgeon General 2006; IARC 2004, Jaakkola et al. 2003). Most evidence for SHS-related impacts is fairly consistent.

SHS has been selected in our study because of its high public health impact, public concern and political interest. Policy measures to (further) reduce SHS exposure have been implemented in the recent past (e.g. the smoking ban) and further policy actions may be taken in the future.

### 3.4.2 Selected health endpoints and exposure-response functions

Out of the large number of health endpoints that SHS is associated with, we selected mortality and morbidity due to lung cancer and ischemic heart disease (IHD), morbidity due to onset of asthma (both in children and in adults), lower respiratory infections and acute otitis media. For the other health endpoints mentioned above, strong evidence is available, but the necessary disease statistics were lacking.

For the SHS-related burden of disease calculations, we have followed the recent WHO methods on the global estimation of disease burden from SHS (Öberg et al. 2010). A summary of outcomes with their respective evidence levels is provided in Table 3-5. The exposure response functions are presented in Table 3-19.

The selected exposure-response values are not gender-specific (e.g. exposure to male or female smoking spouse; exposure to paternal or maternal smoking). Instead, we used the mean relative risk for exposure to adults' smoking. This choice was made in order to limit the sensitivity to gender-specific changes in smoking habits over time and across countries, and because not all exposure data were provided separately for men and women.

The selected outcomes are being applied only to non-smokers, i.e. to the non-smoking disease burden. To that effect, the disease burden due to active smoking has been deduced from the total disease burden, by country (based on total disease burden and active smoking disease burden by country provided by WHO; update 2002 based on Ezzati et al. (2004)).

TABLE 3-5. Summary of recent reviews of health effects of second hand smoke (Adapted from: Öberg et al. 2010).

| Health endpoint | Description | Conclusion regarding the level of evidence (in 3 reports) | | |
|---|---|---|---|---|
| | | WHO (1999) | Californian EPA (2005) | U.S:. Surgeon General (2006) |
| **Outcomes in children** | | | | |
| Acute lower respiratory infection (ALRI) | Incidence of acute lower respiratory illnesses and hospitalisations | *** | *** | *** |
| Otitis media (middle ear infection) | Incidence of otitis media | *** | *** | *** |
| Asthma onset | Incidence of new cases | n | *** | ** |
| **Outcomes in adults** | | | | |
| Asthma induction | Adult-onset incident asthma | *** | ** | n |
| Lung cancer | Incidence | *** | *** | *** |
| Ischemic heart disease (IHD) | Incidence of any ischemic heart disease | *** | *** | n |

* = The evidence of causality is concluded to be "inconclusive", "little", "unclear" or "inadequate".
** = The evidence of causality is concluded to be "suggestive", "some" or "may contribute".
*** = The evidence of causality is concluded to be "sufficient" or "supportive".
n = Not evaluated in the report.

### 3.4.3 Exposure data

Exposures to SHS and background risks vary by gender. Therefore, the data collection should account for differences in the exposures by gender. Some health effects are specific for children, so exposure data also had to be collected separately for children. Overall, the following exposure data are required for estimating the health impacts from SHS:

1. Percentage of children exposed to SHS (i.e. regularly exposed), OR percentage of children having at least one smoking parent
2. Percentage of non-smoking men exposed to SHS
3. Percentage of non-smoking women exposed to SHS

For exposure data collection, we used data from national and international surveys as for example the Survey on Tobacco by the Gallup Organization for the European Commission (EC, 2009) or the European Community Respiratory Health Survey (Janson et al. 2006). The fieldwork for this study was conducted in December 2008 and over 26,500 randomly-selected citizens aged 15 years and over were interviewed in the 27 EU Member States and in Norway. The exposures for the six countries included in EBoDE are presented in Table 3-6. The "upper estimate" is used as the most realistic estimate, as this exposure description matches best the exposure definition used in epidemiological studies from which we derived our exposure-response functions. The lower estimates are provided in Table 3-6 for future sensitivity analysis. Table 3-21 in section 3.12 provides a summary of these data.

TABLE 3-6. Summary of European SHS exposure data for children and non-smoking adults.

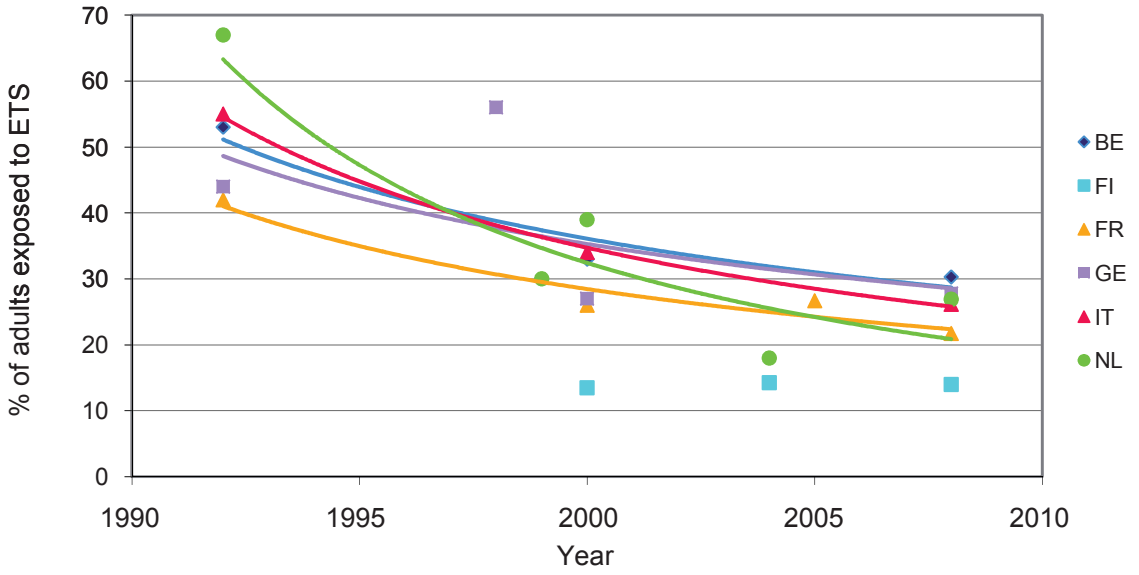
| | Children | | Adults | | | |
|---|---|---|---|---|---|---|
| | [%] | Data year, reference | men [ %] | women [%] | total [%] | Data year, reference |
| Belgium[a)] | - | - | 59<br>34<br>- | 48<br>32<br>- | 53<br>33<br>25/30[b)] | 1990–1994, ECHRS I[1]<br>2002, ECRHS II[1]<br>2008, Eurobarometer[2c)] |
| Finland | 7 | 1996, Lund[3] | 14<br>-<br>- | 13<br>-<br>- | -<br>15<br>6/14[b)] | 2002, Jousilahti[4]<br>2004, NPHI[5]<br>2008, Eurobarometer[2d)] |
| France | 23/33[b)] | 2005, INPES[6] | 38<br>23<br>-<br>- | 46<br>30<br>-<br>- | 42<br>26<br>13/21[b)]<br>13/22[b)] | 1990–1994, ECHRS I[1]<br>2002, ECRHS II[1]<br>2005, INPES[6b)]<br>2008, Eurobarometer[2] |
| Germany | 24 | 2003–2006, GerES IV[7] | 48<br>51<br>28<br>- | 42<br>60<br>26<br>- | 44<br>-<br>27<br>20/28[b)] | 1990–1994, ECHRS I[1]<br>1998, BGS[8]<br>2002, ECRHS II[1]<br>2008, Eurobarometer[2] |
| Italy | 50 | 2001, ICONA[9] | 62<br>37<br>- | 49<br>30<br>- | 55<br>34<br>22/26[b)] | 1990–1994, ECHRS I[1]<br>2002, ECRHS II[1]<br>2008, Eurobarometer[2] |
| Netherlands | 20/36[b)] | 2000–2005, RIVM[10e)] | 68<br>-<br>45<br>-<br>- | 67<br>-<br>33<br>-<br>- | 67<br>30<br>39<br>18/40[b)]<br>18/27[b)] | 1990–1994, ECHRS I[1]<br>1998–2001, RIVM[10]<br>2002, ECRHS II[1]<br>2004–2007, RIVM[10]<br>2008, Eurobarometer[2] |

NA: Adequate data not available
NB: Additional national data are available for some countries, however, these did not match the description of regular exposure.
Definitions used for lower and upper estimates:
[a)] For Belgium, no data for children was found; estimate is calculated using mean of other countries.
References: [1] Janson et al. 2006; [2] EC 2009; [3] Lund et al. 1998; [4] Jousilahti and Helakorpi 2002; [5] Finnish National Public Health Institute, 2004; [6] Institut National de Prévention et d'Education pour la Santé (INPES) 2005; [7] Conrad et al. 2008; [8] Schulze and Lampert 2006; [9] Tominz et al. 2005; [10] van Gelder et al. 2008.
[b)] Lower/upper estimates; INPES: Lower estimate based on "regular" exposure; upper estimate based on exposure "from time to time"; Eurobarometer: Lower estimate based on daily exposure of more than one hour exposure at work and home exposure; upper estimate based on daily exposure of also less than one hour at work and home exposure. RIVM: ranges based on values provided by various studies.
[c)] Exposure at home and at work supposed to be distributed equally.
[d)] Finnish national data (NPHI) also provide survey results, but total exposure to SHS for non-smokers are more difficult to interpret. Therefore only the Eurobarometer data were taken into account here.
[e)] The RIVM report contains data from various studies (e.g. Doetinchem, STIVORO, PIAMA)

Available exposure data (Table 3-6) range across several years, and have been assessed with slightly differing definitions of exposures. In order to estimate exposure data for the target year (2004), exposures have been modelled on the basis of the survey data listed in Table 3-6 as follows:

- Modelling was performed with total adult data, and men/women and children data were assumed to vary according to the same trends.
- Power functions showed the highest correlations in most countries, and were therefore applied in all countries. No trend was apparent for Finland, therefore only the mean was applied.

Resulting trends are displayed in Figure 3-1, and estimated exposure data for 2004 in Table 3-7.




* A trend line for Finland was not considered sufficiently reliable due to paucity of data showing no apparent trend.
NB: Points correspond to the survey data presented in Table 3-6.

FIGURE 3-2. Observed SHS exposure levels (markers) (% of non-smokers) for adults and corresponding modelled trends (lines) in the participating countries.

TABLE 3-7. Modelled exposure to SHS, in children and non-smoking adults in 2004.

| Year 2004 | Children | | Adults (total) | | Women | | Men | |
|---|---|---|---|---|---|---|---|---|
| | Lower* [%] | Upper* [%] | Lower [%] | Upper [%] | Lower [%] | Upper [%] | Lower [%] | Upper [%] |
| Belgium | NA | NA | 28 | 32 | 27 | 31 | 29 | 33 |
| Finland | 4 | NA | 14 | 14 | 14 | 14 | 14 | 14 |
| France | 23 | 33 | 17 | 25 | 20 | 29 | 15 | 22 |
| Germany | 24 | NA | 26 | 31 | 25 | 30 | 27 | 33 |
| Italy | 40 | NA | 26 | 30 | 23 | 26 | 29 | 32 |
| Netherlands | 20 | 36 | 22 | 30 | 19 | 25 | 26 | 34 |

* Lower and upper estimates correspond to different computations of survey data. For example, the upper estimate corresponds to the inclusion of shorter durations of exposure from certain surveys.

## 3.5 Formaldehyde

### 3.5.1 About formaldehyde

Formaldehyde is a high-production volume chemical widely used in building materials, industrial processes and wide range of products. Formaldehyde is widely present both indoors and outdoors, but it reaches high levels mostly indoors. It is used in the production of several building materials and household products, or it can be a by-product of combustion. The high volatility of the compound can lead to high formaldehyde levels in indoor spaces.

Predominant acute symptoms of formaldehyde exposure in humans are irritation of the eyes, nose and throat and aggravation of asthma symptoms (WHO, 2000a). A number of studies point to formaldehyde as an important indoor irritant associated with respiratory illness. A relationship between asthma-like symptoms and indoor concentrations of formaldehyde has been reported, as well as between exposure to

formaldehyde emitted from indoor paint and asthma. Repeated exposures are not associated with more severe effects or lowering of the threshold concentration. Consequently, short-term concentrations are predictive of the effects also after long-term exposure.

Exposure to formaldehyde has also been associated with development of cancer. Convincing evidence exists of high concentrations of formaldehyde being capable of inducing nasal cancer in rats and possibly in mice and genotoxic effects in a variety of in vitro and in vivo systems. Sinonasal cancer in humans has also been associated with high formaldehyde exposures in occupational industrial settings (ranging from 2 to 6 mg $m^{-3}$) (WHO, 2000a). Based on this, IARC has recently classified formaldehyde as carcinogen group 1 (IARC, 2006a).

Formaldehyde was included in EBoDE due to its high toxicological potential, economic significance and related political concern.

### 3.5.2 Selected health endpoints and exposure-response functions

In the EBoDE study, only the development of asthma in toddlers has been included. Sinonasal cancer was not included, because the WHO Air Quality Guidelines working group (WHO, 2000a) as well as recent update of the reviews for the development of WHO Guidelines for indoor air quality (WHO, 2010b) concluded that there is no epidemiological or toxicological evidence that formaldehyde would be associated with sinonasal cancer at levels below 1 mg/m3. The WHO Guidelines for Indoor Air Quality use eye irritation as the main health end-point associated with formaldehyde; however, due to difficulties in estimating a burden of disease from irritation this endpoint was not included in our calculations.

Association with asthma is suggested by the systematic review by McGwin et al., 2010, even though evidence has not been consistent across all the studies (e.g. Krzyzanowski et al, 1990). We selected childhood asthma as the endpoint for formaldehyde, but due to the inconsistencies in the scientific evidence the estimates calculated here should be considered preliminary and to be confirmed by future research. In order to estimate formaldehyde-related asthma, we used the exposure-response function as reported by Rumchev et al. (2002). They studied a cohort of 88 children in Perth, Australia. For every 10 μg $m^{-3}$ increase in formaldehyde exposure in bedrooms, they found an increase of 3% in the risk of having asthma (OR=1.03, 95% CI 1.02–1.04). Based on a reanalysis of their data over reported exposure categories and rescaling for 1 μg $m^{-3}$, the relative risk used in our calculation is 1.0167 (see also Table 3-19 in section 3.12). Asthma effects were calculated for children (<3 years). A similar association may potentially exist for older children and adults, but due to the lack of evidence such relationship was not modelled. This may lead to underestimation of the true formaldehyde-related burden of disease.

A threshold level for effects was applied. The original study by Rumchev reported elevated risks starting from exposures of 60 μg $m^{-3}$. When their data were plotted in order to derive the relative risk, the threshold could be even as low as 40 μg $m^{-3}$. However, the Rumchev study was criticized for confounding factors. WHO (2000a, 2010b) indicated that the lowest concentration that has been associated with nose and throat irritation in exposed workers after short-term exposure is 0.1 mg $m^{-3}$, although some individuals can sense the presence of formaldehyde at lower concentrations. To prevent significant sensory irritation in the general population, an air quality guideline value of 0.1 mg $m^{-3}$ as a 30-minute average was recommended as the WHO Guideline (WHO, 2010b). This is the threshold value that we used in our calculations. Since this is an order of magnitude lower than the presumed threshold for cytotoxic damage to the nasal mucosa, there is a negligible risk of upper respiratory tract cancer in humans below this threshold. As part of the uncertainty analysis, we compared alternative threshold models for cancer (threshold levels of 40, 60 and 100 μg $m^{-3}$) and asthma, see section 5.2.

### 3.5.3 Exposure data

Inhalation is the dominant pathway for formaldehyde exposure in humans. The relevant exposure metric is the residential indoor air level (μg/$m^{-3}$). As indicated above, both of the exposure-response models used apply a threshold level (100 μg $m^{-3}$). Therefore, it is necessary to assess the fraction of the population being exposed to levels higher than this threshold level. A probabilistic simulation model was used to calculate the fraction of the population exceeding the threshold using mean and standard deviation data and assuming

lognormal distributions. No international exposure data sources were identified for formaldehyde, so data have been collected from heterogeneous national sources.

For Belgium, Germany and the Netherlands, only mean exposures were available, without information about the variability. For these three countries, the exposure distributions were based on the data from the other countries (estimated coefficient of variation: 0.6).

TABLE 3-8. Population distributions of residential formaldehyde concentrations.

| Country | mean µg $m^{-3}$ | sd µg $m^{-3}$ | References |
|---|---|---|---|
| Belgium | 24.0 | 14.4[1] | Swaans et al,. 2008 |
| Finland | 41.6 | 22.4 | Jurvelin et al, 2001 |
| France | 23.0 | 14.0 | OQAI, 2006 |
| Germany | 26.0 | 15.6[1] | Umweltbundesamt, 2008 |
| Italy | 16.0 | 8.0 | Lovreglio et al, 2009 |
| Netherlands | 13.0 | 7.8[1] | Dongen,van & Vos, 2008 |

[1] Mean coefficient of variation of the countries with data on variability used for estimation.

Exposure data for formaldehyde are presented in Table 3-21 in section 3.12.

The mean formaldehyde indoor concentrations vary from 13 µg $m^{-3}$ in the Netherlands to about 42 µg $m^{-3}$ in Finland. In Finland formaldehyde exposure levels are higher than in many other developed countries due to the construction materials used and the relatively tightly sealed building envelopes. As shown in Figure 3-3, approximately 42% of population is exposed to levels above 40 µg $m^{-3}$ and 2 % above 100 µg $m^{-3}$.

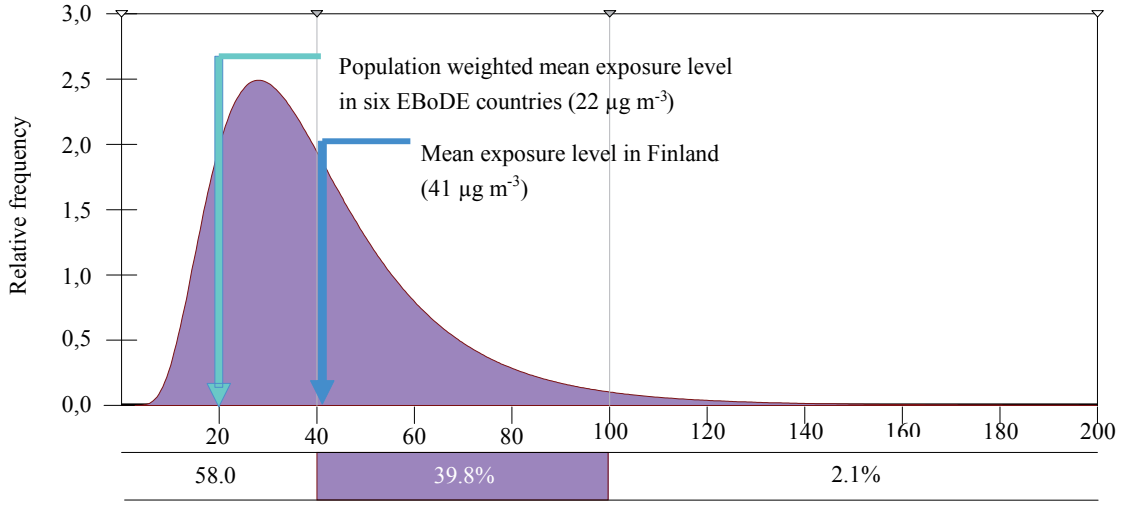



FIGURE 3-3. Estimated formaldehyde exposure distribution in Finland.

Data comparability is compromised for formaldehyde by the differences in population sampling. In France, Germany and the Netherlands, data measurements are representative for country-wide exposure. However, in other countries, measurements have only been carried out in a few cities or were based on a smaller subset of houses.

# 3.6 Lead

## 3.6.1 About lead

Lead is present in the environment due to former application of lead in gasoline, leaded drinking water pipes, and use of lead in paints and other housing materials. Exposures to lead originate from various sources including air, drinking water, food stuff as well as surfaces and consumer products.

Lead is one of the most studied environmental pollutants and has been associated with a large number of health implications (WHO, 2007b). Exposure to lead may cause, amongst other things, kidney damage, miscarriages, effects of the nervous system, declined fertility, alterations in growth and endocrine function, and behavioural disruptions (Hauser et al. 2008; Lanphear et al., 2005; Selevan et al. 2003). Lead is a known neurotoxic pollutant affecting the development of the central nervous system of children and consequently their intelligence. Effects on attention, behaviour disorders and hearing-threshold changes have been described as particularly important (Needleman 1990, WHO/IPCS 1995). Lead exposures have also been shown to be associated with increased blood pressure and risk of hypertension in (female) adults (Nash et al. 2003). Correlations with low lead levels have been reported for the attention deficit hyperactivity disorder (ADHD) (Braun et al., 2006). In addition, there is evidence showing that lead may cause cancer. Lead has been loosely linked with cancers of the lung and stomach. IARC (2006b) rated lead and inorganic lead compounds as probably carcinogenic to humans (Group 2A). Current studies suggest that there is no "safe" level of lead exposure.

Most of the health endpoints are significant at much higher exposure levels that are found in European population today. Exposure to lead has significantly decreased for many countries in the last two decades, especially since the phasing out of leaded gasoline and the replacement of leaded water pipes. For example, Figure 3-3 shows the reduction of internal exposure to lead in humans in German students between the 1980s and now (German Environmental Specimen Bank [Umweltprobenbank des Bundes], data available online at www.umweltprobenbank.de). Indeed, lead has been the success story in environmental policies, but the follow-up in exposure data in the general population is poor.

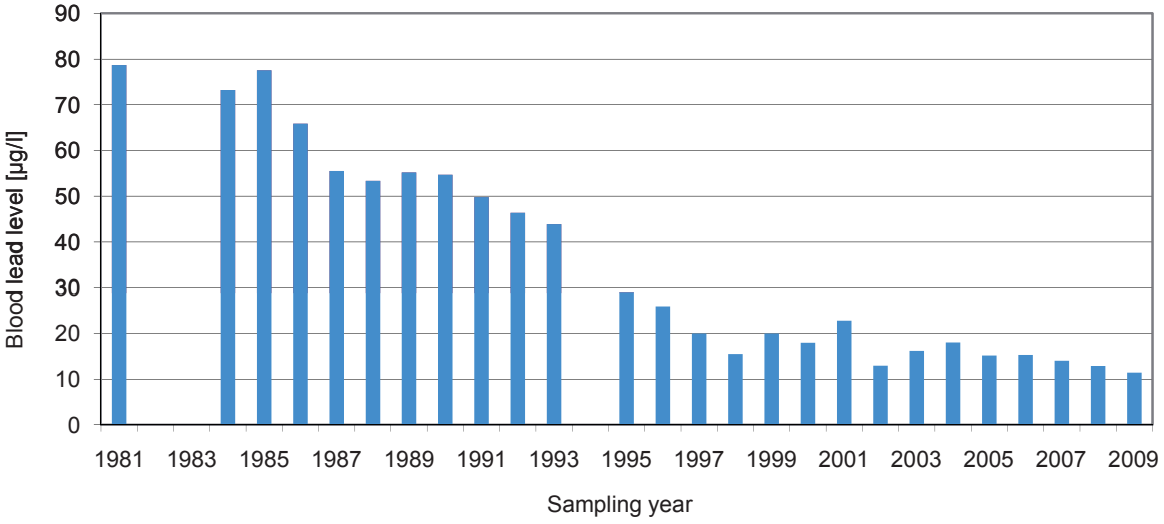



FIGURE 3-4. Blood-Pb in German Students (1981–2009, geometric mean in µg/l, sampling location: city of Münster) (data available at www.umweltprobenbank.de).

## 3.6.2 Selected health endpoints and exposure-response functions

The EBoDE project focuses on two endpoints that have been shown to be relevant at current exposure levels: mild mental retardation (due to IQ loss) and hypertensive disease (due to rise in systolic blood pressure). For the other health endpoints, i. a., no empirically sound exposure-response-relationships are

available. Therefore, our results may underestimate the actual EBD of lead exposure in Europe. The extent of this underestimation cannot be quantified sufficiently.

The hypothesis of an effect threshold was rejected in several studies (Téllez-Rojo et al. 2006, Binns et al. 2007, Chiodo et al. 2004, Kordas et al. 2006). There is strong evidence for an association between B-Pb (blood lead) and negative effects on neuropsychological parameters at levels lower than 100 µg/l (Walkowiak et al., 1998; Canfield et al., 2004; Carta et al., 2005). Therefore, extending the dose-response curve to the range below 100 µg/l is possible. Lanphear et al. (2005) proposed a log-linear model for this curve.

Findings on lead's effects on the central nervous system in the low-dose range are available from longitudinal and cross-sectional studies (Lanphear et al., 2005). These studies showed B-Pb and decrease in IQ points with B-Pb in children. The WHO model for IQ loss was recently updated to consider B-Pb levels above 24 µg/l. It has to be taken into account, however, that no threshold for mental retardation has been confirmed, yet. The exposure/response-function (ERF) in the WHO model is:

$$IQloss = \frac{(B_{Pb} - 24)}{20}$$ (Lanphear et al., 2005; see also Table 3-19 in section 3.12).

The population distribution of IQ is as defined as N(100;15). When the IQ falls below a diagnostic threshold, IQ loss is defined as mild mental retardation, which is the health endpoint used in this study. This threshold is set at 70 IQ points. We calculate the number of cases of mild mental retardation by estimating how many individuals in the target age group (children 0-4 years) exceed the diagnostic thresholds due to the lead exposure.

Several longitudinal studies have examined associations of blood pressure change or hypertension incidence in relation to lead concentration in blood or bone. Glenn et al. (2006) concluded that systolic blood pressure is associated both with acute changes in the blood lead level as well as with long-term cumulative exposure. Blood lead levels can increase in women over the menopause, as lead is released from bone. This may increase women's risk of high blood pressure.

The current WHO model for increased systolic blood pressure in adults aged 20–79 years assumes a linear relationship between 50-200 µg/l (increase of 1.25 mmHg for males and 0.8 mmHg for females per increase of 50 µg/l B-Pb). Above 200 µg/l, an increase of 3.75 mmHg for males and 2.4 mmHg for females per increase of 50 µg/l B-Pb is assumed. The model does not account for aggravating effects of increased blood lead levels during the menopause.

The ERF for mean increase in the systolic blood (mmHg) in the WHO model is (B-Pb >50 µg/l) (Fewtrell et al, 2003):

$$\Delta mmHg = \frac{(B_{Pb} - 50)}{40}$$

The calculation of the numbers of cases of hypertensive disease is similar to the calculations for mild mental retardation. The population distribution of systolic blood pressure is defined as N(135, 15). When exposure exceeds the diagnostic threshold, of 140 mmHg, the increase in blood pressure is defined as hypertensive disease. We calculate how many individuals in the target age group (>15 year olds) exceed the diagnostic threshold due to the lead exposure.

### 3.6.3 Exposure data

It is not easy to estimate lead exposure levels, because population exposure measurements are not regularly conducted, and because of the decreasing trends in lead concentrations which are not fully known. The most reliable way to account for all different possible exposure routes is to measure the body burden of lead. The commonly used exposure metric for such measurement is the blood lead level (B-Pb, whole blood, µg/l).

For the application of the WHO model for IQ loss, distributions of B-Pb (defined by percentiles) are necessary, stratified by specific age groups. This means that data are needed about different fractions of the population that are exposed to certain categories of B-Pb levels. No coherent international data sources

were identified for lead. Hence, data from individual studies conducted in all participating countries were used. The year in which these studies were conducted differs between countries and in some cases the limited temporal coverage prohibited trend estimation. In these cases the most recent data have been used. It is clear that the limited temporal representativity of the lead exposure data poses a significant source of uncertainty. Due to well established lowering trends for lead this is expected to cause mainly unknown overestimation of exposures and effects.

The data are presented in Table 3-9 below and summarized in Table 3-21 in section 3.12. As shown in Table 3-9, lead data have been measured in different age groups in the different countries. Data from the German Environmental Survey (GerES) show that age is an important influencing factor for B-Pb levels in humans. As there is virtually no evidence for a significant reduction in B-Pb levels since the year 2000, the difference in age groups is assumed to be one of the most important sources of uncertainties when comparing the different countries. Unfortunately, B-Pb data are not sufficient to correct the country data for age.

TABLE 3-9. Lead data (µg/l) for different countries, measured in different age groups and years, used in the lognormal simulation to yield the required distributional parameters.

| Country | Estimates (2004) | | | Age group | Year |
|---|---|---|---|---|---|
| | AM | GM | SD | | |
| Belgium | 22 | | 16 | 14–15 | 2000–06 |
| Finland | 16 | | 11 | Adults | 2004 |
| France | | 26 | 18 | 18–74 | 2006–07 |
| Germany | 22 | | 16 | 20–29 | 2004 |
| Italy | 39 | | 24 | 18–64 | 2000 |
| Netherlands | | 19 | 11 | 1–6 | 2005 |

AM: Arithmetic Mean; GM: Geometrical Mean; SD: Standard Deviation (estimated using coefficient of variation).

As indicated above, both of the exposure-response models used apply a threshold level (50 µg $l^{-1}$ and 24 µg $l^{-1}$). Therefore, it is necessary to assess the fraction of the population being exposed to levels higher than these threshold levels. A probabilistic simulation model was used to calculate the fraction of the population exceeding the threshold using mean and standard deviation data and assuming lognormal distributions. Standard deviations were estimated for the simulation using a coefficient of variation estimated from the Finnish data.

TABLE 3-10. Population distributions of blood lead levels used in the simulation of threshold exeedances assuming log-normal distribution.

| | Country | BE | FI | FR | DE | IT | NL |
|---|---|---|---|---|---|---|---|
| Adults | mean | 22.0 | 16.0 | 25.0 | 22.0 | 39.0 | 19.0 |
| | SD | 15.6 | 11.4 | 17.8 | 15.6 | 27.7 | 13.5 |
| | CV | 0.71 | 0.71 | 0.71 | 0.71 | 0.71 | 0.71 |
| Children | mean | 22.0 | 16.0 | 25.0 | 22.0 | 39.0 | 19.0 |
| | SD | 15.6 | 11.4 | 17.8 | 15.6 | 27.7 | 13.5 |
| | CV | 0.71 | 0.71 | 0.71 | 0.71 | 0.71 | 0.71 |

SD: Standard deviation, CV: coefficient of variation.

# 3.7 Transport noise

## 3.7.1 About transport noise

Noise from road, rail, and air traffic affects a great number of people. Exposure to transport noise may cause sleep disturbance as well as annoyance, potentially leading to high blood pressure and increased incidence of myocardial infarction (WHO, 2000b; Miedema & Vos 2007; Babisch 2006, 2008). Transport noise exposure as a part of total environmental noise has also been linked to effects on cognition. Transport noise is selected in this study due to its ubiquity and high public health impact. In addition, due to the economic significance of transport, noise levels despite technological progress keep on increasing over time.

## 3.7.2 Selected health endpoints and exposure-response functions

As health end-points, high sleep disturbance and Ischemic Heart Disease (IHD) were included (Miedema & Vos, 2007; Babisch 2006, 2008). Hypertension and related heart disease due to aircraft noise was not considered because no clear review could be identified. Nevertheless, since causal relationships are very likely and have been reported recently, this health effect may be considered in the near future (Babisch & Kamp, 2009). For railway noise no significant associations with hypertension and IHD could be identified (Barregard et al., 2009).

Effects on cognition were also excluded, as these are difficult to quantify. In addition, severe annoyance, as annoyance was not included as annoyance does not fall weithin our definition of health and not considered a health effect by, amongst others, WHO. Some other studies (e.g. Knol & Staatsen, 2005) have applied a broader definition of health, in which annoyance was included as a health effect because it reduces quality of life. These studies show a substantial burden of disease due to transport noise related annoyance.

The formulas applied to estimate transport noise related high sleep disturbance (HSD) are as follows. The results directly give the number of people severely sleep disturbed at a certain decibel level for each noise source (Miedema et al., 2002, Miedema & Vos, 2007):

Road traffic noise: $\%HSD = 20.8 - 1.05L_{night} + 0.01486(L_{night})^2$
Railway traffic noise: $\%HSD = 11.3 - 0.55L_{night} + 0.00759\ (L_{night})^2$
Aircraft noise: $\%HSD = 18.147 - 0.956L_{night} + 0.01482(L_{night})^2$

$L_{night}$ is a measure of night-time noise, defined as the yearly average of night noise levels (23–7h) at the façade of houses. The formulas can be applied in the range of $L_{night}$ from 45 to 65 (max. 70) dB(A) (data are regularly available for >50dB(A)).

There is no exclusive causal mechanism postulated specifically to myocardial infarction (MI). Therefore, the OR for MI has been applied to all types of ischemic heart disease (Babisch, 2008) according to the following formula (Babisch 2006, 2008):

$OR = 1.63 - 0.000613*(L_{day,16h})^2 + 0.00000736*(L_{day,16h})^3$

$L_{day,\ 16h}$ is defined as the yearly average of day and evening noise levels (7-23h) and $L_{day}$ as the yearly average of day noise levels (7-19h) and $L_{evening}$ as the yearly average of evening noise levels (19–23h). The exposure-response functions are valid for $L_{day,\ 16h}$ noise levels ranging from 55 to approximately 80 dB(A).

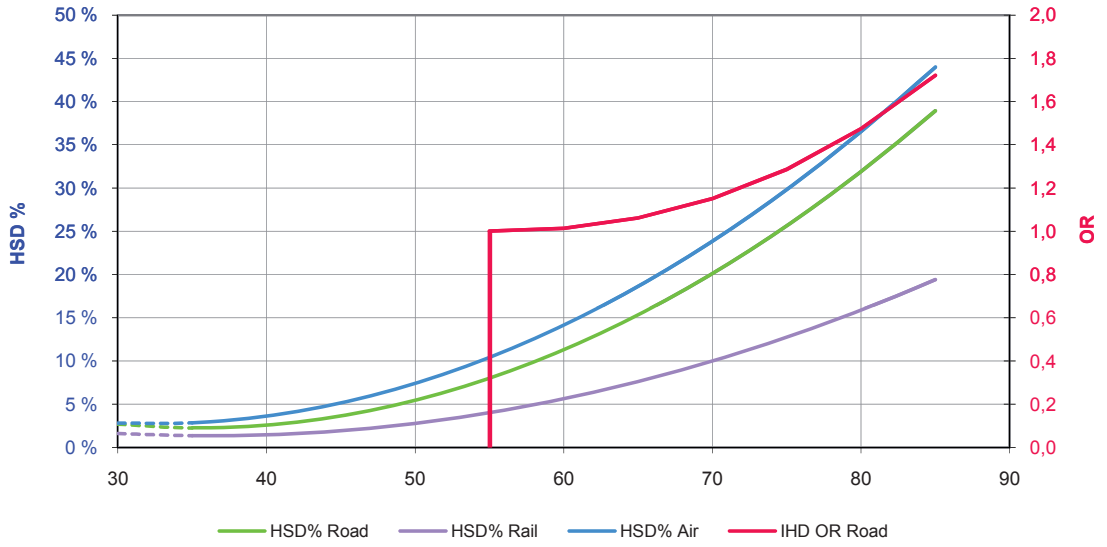



FIGURE 3-4. The exposure-response functions for high sleep disturbance (HSD; blue axis) and ischeamic heart disease (red axis) presented in graphical form for road, rail and air traffic noise. The OR for Myocardial Infarction, which we applied for all ischeamic heart disease (IHD), is modelled only for road traffic (red curve). (Dotted lines display E-R functions outside the range where they are considered valid).

### 3.7.3 Exposure data

The exposure metric used is the average 24h-noise level day-evening-night ($L_{den}$) and the average 8h-noise level during night-time ($L_{night}$) in dB(A), separated for road, aircraft and railway traffic. These indices have been suggested by the European Environmental Noise Directive (2002/49/EC, "END", (EU, 2002).

From the first phase of END-reporting, carried out in 2007/2008, noise exposure data per 5dB(A) categories are now available for most of the EU-countries. These data concern:

- agglomerations with more than 250 000 inhabitants (separately for road, aircraft and railway traffic);
- roads outside agglomerations with more than 6 000 000 vehicles/passages per year;
- railways outside agglomerations with more than 60 000 trains/passages per year;
- major airports with air traffic higher than 50 000 movements/flights per year (some separated for inside and outside agglomerations and total).

These data have been aggregated recently and still are to some extent being processed. So far, they only cover a relatively small percentage of the EU population. The data are presented in Table 3-11. The population coverage of the data for the countries that have so far been included depends substantially on their urbanity, on the administrative prerequisites (e.g. extent of cities) and on the location of country (central or in periphery) and resulting transit influences. In consequence, the comparability of the countries is limited. Additionally, data for Belgium until April 2010 only included Flanders. For France, only data for major agglomerations were included. Only exposure levels above $L_{night}$ 50dB ($L_{den}$ 55 dB) are reported in END data, so there are no data about exposure below those levels in the END database.

For ischemic heart disease, the exposure-response function was calculated for a 16h-daytime level ($L_{day}$). As a crude but practical tool for conversion, the following easy formula was used:

$L_{day}$,16h = $L_{den}$ – 2.5 (Babisch, 2008)

Unfortunately, END-reporting of many included countries until April 2010 was only complete for $L_{den}$ but incomplete considering data about $L_{night}$. Therefore adjustments had to be made for calculation of people affected by high/severe sleep disturbance. As a crude but practical tool for conversion, the following easy formula was used:

$L_{den} = L_{night} + 7.5$ (expert judgement after first data-pooling, most valid for road traffic noise in agglomerations)

There are large differences concerning these conversion factors, especially for conversion from $L_{den}$ to $L_{night}$, for which estimates range from +5 (very urban) up to +11 (including rural areas) (WG-AEN 2006). For comparability, 7.5 was chosen as a conservative factor, even though in some cases there might be an underestimation of real exposure.

END-reporting covers 5dB(A)-categories. For modelling purposes, the mid-values of the 5dB(A)-categories (50–54.9 → 52.5) were inserted in the non-linear polynomials as a feasible simplification.

TABLE 3-11. Baseline exposures for noise (Environmental Noise Directive reporting data, 2007/2008).

| **Road traffic, agglomerations** | Raw Lden [dB(A)] data from END-Reporting 1st stage 2007 | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Countries | Population in agglomerations | 55-59 dB | 60-64 dB | 65-69 dB | 70-74 dB | >75 dB | Population 2004 | Exposed >55 dB | Exposed population |
| Belgium (BE) | n/a | n/a | n/a | n/a | n/a | n/a | 10 359 676 | n/a | n/a |
| Finland (FI) | 559 716 | 87 249 | 88 660 | 46 648 | 14 860 | 73 | 5 231 166 | 237 490 | 4.5 % |
| France (FR) | 12 704 947 | 1 665 222 | 2 198 662 | 2 477 022 | 1 908 546 | 226 977 | 60 623 894 | 8 476 429 | 14.0 % |
| Germany (DE) | 13 499 029 | 1 219 623 | 828 255 | 652 079 | 336 339 | 48 910 | 82 627 588 | 3 085 206 | 3.7 % |
| Italy (IT) | 2 934 473 | 1 957 800 | 421 300 | 151 400 | 80 000 | 4 700 | 58 474 754 | 2 615 200 | 4.5 % |
| Netherlands (NL) | 5 002 655 | 827 900 | 673 300 | 344 100 | 44 500 | 1 000 | 16 263 535 | 1 890 800 | 11.6 % |
| Sum | 34 700 820 | 5 757 794 | 4 210 177 | 3 671 249 | 2 384 245 | 281 660 | 233 580 613 | 16 305 125 | 7.0 % |

| **Road traffic, outside agglom.[1]** | Raw Lden [dB(A)] data from END-Reporting 1st stage 2007 | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Countries | Road length km | 55-59 dB | 60-64 dB | 65-69 dB | 70-74 dB | >75 dB | Population 2004 | Exposed >55 dB | Exposed population |
| Belgium (BE) | 2 792 | 201 300 | 92 100 | 83 900 | 91 900 | 18 000 | 10 359 676 | 487 200 | 4.7 % |
| Finland (FI) | 647 | 590 | 246 | 81 | 13 | 1 | 5 231 166 | 931 | 0.02 % |
| France (FR) | 20 274 | 1 821 229 | 866 917 | 461 855 | 242 042 | 126 184 | 60 623 894 | 3 518 226 | 5.8 % |
| Germany (DE) | 17 056 | 1 858 365 | 840 909 | 490 458 | 239 627 | 41 814 | 82 627 588 | 3 471 173 | 4.2 % |
| Italy (IT) | 6 324 | 1 654 840 | 1 382 550 | 910 380 | 370 470 | 121 100 | 58 474 754 | 4 439 340 | 7.6 % |
| Netherlands (NL) | 3 496 | 128 600 | 43 600 | 13 200 | 1 900 | 100 | 16 263 535 | 187 400 | 1.2 % |
| Sum | 50 589 | 5 664 924 | 3 226 322 | 1 959 874 | 945 952 | 307 199 | 233 580 613 | 12 104 270 | 5.2 % |

| **Rail traffic, agglomerations** | Raw Lden [dB(A)] data from END-Reporting 1st stage 2007 | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Countries | Population in agglomerations | 55-59 dB | 60-64 dB | 65-69 dB | 70-74 dB | >75 dB | Population 2004 | Exposed >55 dB | Exposed population |
| Belgium (BE) | n/a | n/a | n/a | n/a | n/a | n/a | 10 359 676 | n/a | n/a |
| Finland (FI) | 559 716 | 27 513 | 25 390 | 16 669 | 207 | 0 | 5 231 166 | 69 779 | 1.3 % |
| France (FR) | 2 698 604 | 48 000 | 36 600 | 14 300 | 8 800 | 0 | 60 623 894 | 107 700 | 0.2 % |
| Germany (DE) | 10 752 155 | 194 617 | 112 457 | 71 768 | 18 422 | 1 919 | 82 627 588 | 399 183 | 0.5 % |
| Italy (IT) | n/a | n/a | n/a | n/a | n/a | n/a | 58 474 754 | n/a | n/a |
| Netherlands (NL) | 5 002 655 | 118 600 | 60 700 | 25 000 | 8 800 | 1 000 | 16 263 535 | 214 100 | 1.3 % |
| Sum | 19 013 130 | 388 730 | 235 147 | 127 737 | 36 229 | 2 919 | 233 580 613 | 790 762 | 0.3 % |

| **Rail traffic, outside agglom.[2]** | Raw Lden [dB(A)] data from END-Reporting 1st stage 2007 | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Countries | Rail length km | 55-59 dB | 60-64 dB | 65-69 dB | 70-74 dB | >75 dB | Population 2004 | Exposed >55 dB | Exposed population |
| Belgium (BE) | 461 | 33 300 | 19 700 | 16 100 | 13 400 | 3 900 | 10 359 676 | 86 400 | 0.8 % |
| Finland (FI) | 96 | 255 | 95 | 48 | 5 | 0 | 5 231 166 | 403 | 0.0 % |
| France (FR) | 1 781 | 624 242 | 419 956 | 250 289 | 139 459 | 105 232 | 60 623 894 | 1 539 178 | 2.5 % |
| Germany (DE) | 4 435 | 831 000 | 304 200 | 117 500 | 57 800 | 43 000 | 82 627 588 | 1 353 500 | 1.6 % |
| Italy (IT) | 438 | 89 900 | 61 900 | 37 300 | 33 000 | 24 800 | 58 474 754 | 246 900 | 0.4 % |
| Netherlands (NL) | 854 | 134 000 | 76 500 | 38 100 | 12 500 | 3 000 | 16 263 535 | 264 100 | 1.6 % |
| Sum | 8 065 | 1 712 697 | 882 351 | 459 337 | 256 164 | 179 932 | 233 580 613 | 3 490 481 | 1.5 % |

| **Air traffic, large airports** | Raw Lden [dB(A)] data from END-Reporting 1st stage 2007 | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Countries | Movements per year | 55-59 dB | 60-64 dB | 65-69 dB | 70-74 dB | >75 dB | Population 2004 | Exposed >55 dB | Exposed population |
| Belgium (BE) | 253 257 | 106 698 | 14 766 | 1 787 | 0 | 0 | 10 359 676 | 123 251 | 1.2 % |
| Finland (FI) | 173 000 | 57 200 | 1 700 | 100 | 0 | 0 | 5 231 166 | 59 000 | 1.1 % |
| France (FR) | 1 376 340 | 107 800 | 9 000 | 12 100 | 0 | 0 | 60 623 894 | 128 900 | 0.2 % |
| Germany (DE) | 1 862 273 | 479 500 | 178 000 | 25 700 | 1 700 | 0 | 82 627 588 | 684 900 | 0.8 % |
| Italy (IT) | 992 506 | 157 500 | 49 200 | 9 800 | 1 400 | 200 | 58 474 754 | 218 100 | 0.4 % |
| Netherlands (NL) | 440 153 | 61 600 | 6 100 | 1 200 | 100 | 0 | 16 263 535 | 69 000 | 0.4 % |
| Sum | 5 097 529 | 970 298 | 258 766 | 50 687 | 3 200 | 200 | 233 580 613 | 1 283 151 | 0.5 % |

[1] Population living close to roads with more than 6,000,000 vehicles/passages per year

[2] Population living close to rails with more than 60,000 trains/passages per year

# 3.8 Ozone

## 3.8.1 About ozone

Ozone in the lower atmosphere (or tropospheric ozone) is not emitted directly, but is formed in the atmosphere in photochemical reactions from anthropogenic and natural emissions of precursor components involving mostly volatile organic compounds (VOCs) and nitrogen oxides (mainly NO and $NO_2$). These substances react to form ozone under the influence of sunlight. Ozone is highly reactive and therefore other air pollutants also easily consume the ozone present in the air. Therefore, the highest ozone levels are typically found in background regions and levels in urban areas are generally lower than in the countryside.

Exposure to ozone can lead to a variety of respiratory health effects, such as coughing, throat irritation and reduced lung function. In addition, it can worsen bronchitis, emphysema, and asthma (WHO, 2006a). Ozone levels are increasing over time, and are cause for political concern.

## 3.8.2 Selected health endpoints and exposure-response functions

For ozone, as well as for PM (see section 3.9), we followed the health impact assessment approach as laid out in the Clean Air For Europe (CAFE) project and based on WHO European Centre for Environment and Health and CLTRAP Task Force on Health consultations. Health effects that are taken into consideration include total non-violent mortality, minor restricted activity days (MRADs), and cough and lower respiratory symptoms (LRS) in children aged 5–14 years. The choice of these endpoints was guided by Cost Benefit Analysis as carried out in the CAFE project (Hurley et al, 2005, WHO 2008). The health endpoints considered and the corresponding exposure-response functions are summarized in Table 3-19 in section 3.12.

## 3.8.3 Exposure data

The exposure metric used for ozone calculations is the sum of ozone maximum 8-h levels above 35 ppb, called SOMO35 (WHO, 2008). SOMO35 (expressed in $\mu g\ m^{-3} \times$ hours) is the sum of the maximum daily 8-hour concentrations that are exceeding 35 ppb (70 $\mu g\ m^{-3}$) for each day in the calendar year, i.e. e.g. a daily level of 100 $\mu g\ m^{-3}$ would contribute 30 to the SOMO35 calculation. Regardless of the name referring to the ppb unit of measurement, the values are expressed as mass concentrations ($\mu g\ m^{-3}$).

For ozone (as well as for PM, see section 3.9), exposures were estimated by the European Topic Centre on Air and Climate Change (ETC/ACC) using AirBase data and air quality maps (SOMO35) (de Leeuw & Horalek, 2009). The European Environment Agency (EEA) has recently published an evaluation of new monitoring-based methods to estimate population weighted spatial distributions of ambient PM and ozone levels (EEA, 2009). These methods are based on interpolated maps using 10×10 km spatial resolution and using observed concentrations from national monitoring networks as primary data source. These are combined with regional chemistry transport modelling (CTM) and other supplemental data sources to improve estimates in observation-sparse areas. Maps for rural and urban areas were created separately and were subsequently merged. This approach aims to provide an objective method for dealing with the differences found between the rural and urban interpolated concentration fields in most areas of Europe (EEA, 2009). It is different from the earlier Clean Air for Europe (CAFE) work, which relied on modelling as its primary source of information and uses monitoring only to calibrate the European Monitoring and Evaluation Programme (EMEP) chemical transportation model. The modelling approach is better suitable for prospective scenario analyses, while the monitoring based approach may be considered more reliable for retrospective analyses.

The air quality maps were prepared for 2005 with interpolation methodology using co-kriging of observed concentrations using additional spatial information (EMEP model results, meteorological data, altitude, population density map). The year 2005 instead of 2004 was chosen as the modelling year by EEA for practical purposes. Description of the maps is given by Horálek et al (2007) and de Leeuw and Horalek (2009). A brief introduction to AirBase and a description of the state of and recent trends in European air quality is presented by Mol et al (2009).

Population weighted ambient ozone concentrations were calculated using population data for year 2005. The population density map (resolution 10x10 km) is based on the detailed population map prepared by JRC (reference year 2002, see Horalek *et al.*, 2008 for further description of this dataset). The population density map for 2005 is made by scaling the 2002-reference map using the 2005/2002 ratio of national population numbers. Within a country the same age distribution is assumed in all grid cells.

Resulting population-weighted ozone exposure values for the participating countries are shown in Table 3-12 and are also summarized in Table 3-21 in section 3.12. The geographical distribution of the SOMO35 levels in Europe is shown in Figure 3-5.

TABLE 3-12.: National population weighted averages of ambient ozone levels (SOMO35) in 2005 for the six EBoDE countries (de Leeuw and Horalek, 2009).

| Country | SOMO35 (µg $m^{-3}$) |
|---|---|
| Belgium | 2 787 |
| Germany | 4 164 |
| Finland | 2 580 |
| France | 4 756 |
| Italy | 8 134 |
| Netherlands | 1 920 |

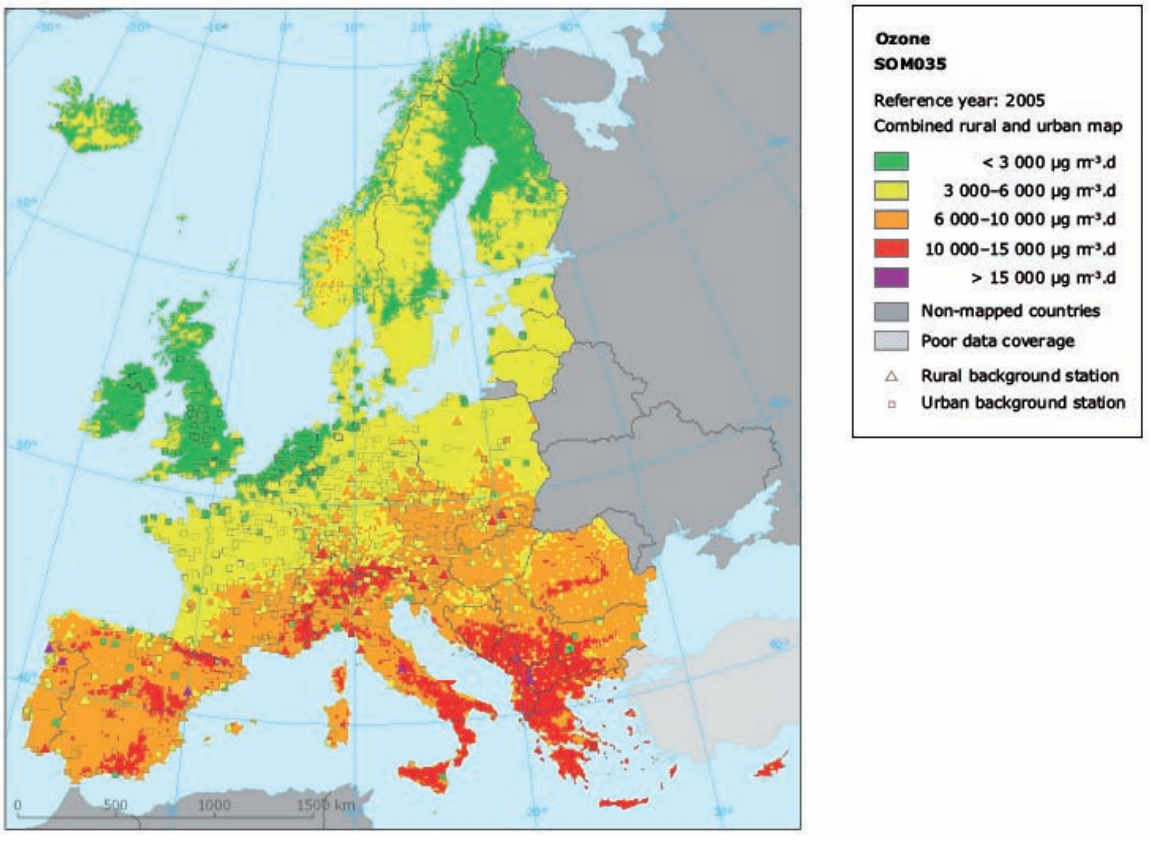



FIGURE 3-5. Ozone SOMO35-levels in Europe in 2005 (EEA, 2009).

# 3.9 Particulate matter

## 3.9.1 About particulate matter

Exposure to Particulate matter (PM) has been associated with both respiratory and cardiovascular effects and total non-violent mortality (Pope and Dockery, 2006, WHO, 2006a,b) and it is the most thoroughly internationally reviewed environmental pollutant during the last decade. PM was selected in EBoDE due to its high public health impact, economic significance (industry, transport) and political concern. Particulate matter is a complex mixture of components from natural and anthropogenic sources and is partly created in chemical and physical processes in the atmosphere from gaseous primary components like sulphur dioxide, nitrogen oxides, ammonia, and volatile organic compounds. The health implications of the particulate matter components have been extensively studied, but still the most convincing epidemiological evidence associates $PM_{2.5}$ mass concentrations with the health impacts (Pope & Dockery, 2006).

## 3.9.2 Selected health endpoints and exposure-response functions

For PM (and ozone) we followed the health impact assessment approach as laid out in the Clean Air For Europe (CAFE) project and based on WHO European Centre for Environment and Health and CLTRAP Task Force on Health consultations (Hurley *et al.* 2005, WHO, 2006a, b). $PM_{2.5}$ and $PM_{10}$ both serve as indicators of a complex mixture of physically and chemically heterogeneous composition. In the EBoDE calculations, we calculated burden of disease related both to $PM_{10}$ and to $PM_{2.5}$ exposure. Due to the overlap between these two indicators, in the aggregate results only the results for $PM_{2.5}$ are included. For $PM_{2.5}$, we calculated the burden of disease for cardiopulmonary mortality, lung cancer mortality, total non-violent mortality, chronic bronchitis and restricted activity days (RAD; defined by Hurley *et al.*, 2005). Due to the overlap between the different mortality endpoints, we included only cause specific mortality in the aggregate results. For $PM_{10}$, lower respiratory symptoms (LRS) and new cases of chronic bronchitis were included.

For mortality, we used the relative risks as provided by Pope (Pope et al., 2002; WHO, 2006a,b). For morbidity, relative risks are based on the thorough review made for the CAFE estimates by Hurley et al. (2005) and WHO (2006b). The health endpoints and corresponding exposure-response functions are summarized in Table 3-19 in section 3.12.

## 3.9.3 Exposure data

Annual population weighed mean ambient concentrations of $PM_{2.5}$ and $PM_{10}$ were estimated, similarly to the values for ozone, by the European Topic Centre on Air and Climate Change (ETC/ACC) using geographical modelling. Population density data were based on JRC data. For further details, see the ozone section. Exposure values are presented in Table 3-13 and summarized in Table 3-21 in section 3.12.

The calculations involve no reference concentration for estimating the PM effects, so all PM-related morbidity and mortality are included in the burden of disease estimates. This is in contrast to, for example, the CAFE calculations, in which only the impacts of European anthropogenic emissions were estimated. The EBoDE calculations include the contribution to PM from natural sources and sources outside Europe.

TABLE 3-13. National population weighted averages of ambient PM levels in 2005 for the target countries (de Leeuw and Horalek, 2009).

| Country | Concentrations | |
|---|---|---|
| | $PM_{10}$ ($\mu g\ m^{-3}$) | $PM_{2.5}$ ($\mu g\ m^{-3}$) |
| Belgium | 28.9 | 18.7 |
| Finland | 13.3 | 9.1 |
| France | 19.1 | 12.3 |
| Germany | 22.1 | 16.0 |
| Italy | 32.7 | 19.6 |
| Netherlands | 29.1 | 18.7 |

# 3.10 Radon

## 3.10.1 About radon

Radon is a short-lived radioactive gas that occurs naturally in soils and rocks. It is generated by the radioactive decay of uranium. Indoor radon concentrations differ based on the characteristics of the geological substrates beneath houses and the use of different building materials.

Exposure to radon can lead to lung cancer. Studies to estimate the risk of lung cancer associated with residential radon exposure have been conducted in many European countries (Lagarde *et al.* 1997, Bochicchio, 2005, 2008; Darby *et al.*, 2005, 2006). Radon is classified by IARC as carcinogenic to humans (type 1, 1988) with genotoxic action. No safe level of exposure can be determined (WHO, 2000a). Besides lung cancer radon is not known to cause other health effects.

Radon has a synergistic effect with smoking. Epidemiological evidence suggests that the risk of simultaneous exposure to both tobacco smoke and radon is more than additive but that it may be less than multiplicative.

## 3.10.2 Selected health endpoints and exposure-response functions

Radon effects are usually presented as additional cases of lung cancer at a certain exposure (i.e. unit risk model). In order to account for the interaction with smoking, however, a relative risk model seems more appropriate. We therefore calculated results using both a unit risk model and a relative risk model (method 1A and 2A). The RR method (1A) is used in the final aggregate results. The radon UR model (UR=6.6E-07 $(Bq\ m^{-3})^{-1}$, Darby *et al.*, 2005) is used for comparison of UR and RR modelling approaches in Chapter 5.

The relative risk model, as suggested by the meta-analysis of Darby et al. (2005), assumes the lung cancer risk from radon to be linearly proportional to the radon exposure, but also to the background lung cancer rate caused by tobacco smoking (and, to a lesser extent, by exposure to second-hand smoke, ambient air particulate matter and possibly some occupational exposures) (see Table 3-19 in section 3.12 for the RR values).

## 3.10.3 Exposure data

The soil uranium contents and respectively the residential radon concentrations vary significantly between the countries. Yet the differences within the countries are still far greater, and the indoor radon concentrations in individual buildings are essentially impossible to predict. Long-term average indoor radon concentrations, however, are relatively easy to measure and are therefore better known and comparable between the countries than those of any other indoor air contaminant.

EBoDE uses the national residential radon exposure estimates as collected by the EU RadonMapping project (http://radonmapping.jrc.ec.europa.eu; country reports available from http://radonmapping.jrc.ec.europa.eu/index.php?id=37&no_cache=1&dlpath=National_Summary_Reports, accessed 11 June 2009). and the UNSCEAR 2000 Report, as presented in Table 3-14 and summarized in Table 3-21 in section 3.12. No further national data collection was conducted, but some additional international data sources were identified, notably from the WHO Radon project (IRP, 2010).

TABLE 3-14. Radon concentrations in dwellings determined in indoor surveys (compiled from National Summary Reports at http://radonmapping.jrc.ec.europa.eu/ and UNSCEAR, 2000). The respective cancer risks are estimated from background lung cancer rates using both absolute and relative risk models.

| Country | AM ($Bq\ m^{-3}$) | GM ($Bq\ m^{-3}$) | GSD | % (of people exposed) › ≥200 $Bq\ m^{-3}$ | % (of people exposed) › ≥400 $Bq\ m^{-3}$ | Max ($Bq\ m^{-3}$) |
|---|---|---|---|---|---|---|
| Belgium | 69 | 76 | 2.0 | | 0.5 | 4 500 |
| Finland | 120 | 84 | 2.1 | 12.3 | 3.6 | 33 000 |
| France | 89 | 53 | 2.7 | 8.5 | 2.0 | 4 964 |
| Germany | 50 | 40 | 1.9 | 3.0 | 1.0 | 10 000 |
| Italy | 70 | 52 | 2.0 | 4.1 | 0.9 | 1 036 |
| The Netherlands | 30 | 25 | 1.6 | 0.3 | 0.0 | 382 |

AM: Arithmetic Mean; Bq: Becquerel; GM: Geometric Mean; GSD: Geometric Standard Deviation.

## 3.11 Burden of disease, health and population data

For this project, the harmonized health statistics database as held by the World Health Organization was used. This database provides health data specific for each country, health endpoint as defined by the Environmental Burden of Disease -programme, age group and gender. We used data (deaths and DALYs) for the year 2004 (WHO, 2009b; more detailed data available on request)(World Health Organization. *The global burden of disease: 2004 update*. Geneva: World Health Organization; 2009. Available at: http://www.who.int/healthinfo/global_burden_disease/2004_report_update/en/index.html).

The data were obtained in discounted/age-weighted and undiscounted/un-age-weighted format.

Depending on the type of calculation (see section 2.1), different data were needed. For calculations according to methods 1A and 2A (Table 3-19 shows which methods were used for which calculation), the total YLL and YLD were needed per age group and country. The WHO database provides data for pre-defined age categories (e.g. 0–4; 5–14, etc). Age-specific values were derived assuming an equal distribution of people within the age categories in the WHO data. Table 3-15 shows a sample of the burden of disease as available from the WHO database, aggregated over all ages and for a selection of health endpoints only.

For calculations according to method 2B (see section 2.1 and Table 3-19), no background health data was applied; the incidences were calculated using a unit risk model and the burden of disease was estimated using the WHO disability weights and duration estimates. For these calculations (IQ loss and HTD from lead), no age-weighing was applied due to the lack of information on the age distribution of the effects; however, the impact of the simplification was estimated to be small and to affect only the discounted results. Table 3-16 shows the disability weights and durations that were used.

We have carried out preliminary calculations to investigate the potential effect of lag times on the discounted estimates. The lag times used per health endpoint are provided in Table 3-17.

Population data (number of people in 2004) were used to calculate numbers of DALYs per million people and are provided in Table 3-18.

TABLE 3-15. WHO burden of disease data (total undiscounted, not-age-weighted DALYs) in 2004 (sample of health endpoints; aggregated over all ages).

| Health endpoint | YLL/ YLD | Burden of disease data (WHO) – discounted (3%) and age-weighted | | | | | |
|---|---|---|---|---|---|---|---|
| | | Belgium | Finland | France | Germany | Italy | Netherlands |
| Total mortality (non-violent) | YLL | 1 137 042 | 520 755 | 5 904 337 | 9 261 877 | 5 780 589 | 1 585 775 |
| | YLD | | | | | | |
| Total morbidity | | 930 436 | 460 350 | 5 219 164 | 7 283 809 | 4 838 018 | 1 360 245 |
| Total cancer | YLL | 413 390 | 154 033 | 2 447 205 | 3 193 738 | 2 215 606 | 622 914 |
| | YLD | 34 945 | 12 796 | 198 478 | 254 086 | 172 230 | 50 271 |
| Leukaemia | YLL | 15 490 | 5 586 | 99 669 | 119 106 | 93 212 | 22 131 |
| | YLD | 487 | 167 | 3 222 | 3 845 | 2 859 | 689 |
| Lung cancers | YLL | 103 461 | 27 142 | 514 569 | 653 118 | 465 809 | 154 443 |
| | YLD | 2 125 | 583 | 9 473 | 12 740 | 10 014 | 3 031 |
| Otitis media | YLL | n/a | n/a | n/a | n/a | n/a | n/a |
| | YLD | 1 184 | 611 | 7 447 | 8 038 | 5 469 | 2 004 |
| Ischemic heart disease | YLL | 178 793 | 115 258 | 478 408 | 1 624 841 | 841 741 | 177 269 |
| | YLD | 21 764 | 13 676 | 56 462 | 188 782 | 98 850 | 22 340 |
| Cardiopulmonary disease | YLL | 385 102 | 203 063 | 1 222 063 | 3 067 603 | 1 861 658 | 425 443 |
| | YLD | 147 553 | 58 363 | 502 501 | 1 003 277 | 559 717 | 189 275 |
| Chronic bronchitis | YLL | 47 784 | 9 982 | 69 644 | 239 985 | 134 312 | 55 767 |
| | YLD | 66 091 | 14 949 | 125 272 | 404 043 | 151 689 | 83 814 |
| Asthma induction/ aggravation | YLL | 4 632 | 935 | 17 794 | 31 851 | 9 902 | 1 882 |
| | YLD | 13 818 | 9 264 | 99 867 | 100 872 | 60 658 | 31 209 |
| Lower respiratory infections | YLL | 30 003 | 13 331 | 97 589 | 168 799 | 78 505 | 44 596 |
| | YLD | 629 | 361 | 1 981 | 3 721 | 2 317 | 1 072 |
| Sum of all above | YLL | 2 313 816 | 2 313 816 | 2 313 816 | 2 313 816 | 2 313 816 | 2 313 816 |
| | YLD | 1 220 913 | 1 220 913 | 1 220 913 | 1 220 913 | 1 220 913 | 1 220 913 |
| | DALY | 3 534 729 | 3 534 729 | 3 534 729 | 3 534 729 | 3 534 729 | 3 534 729 |

TABLE 3-16. Input parameters for calculating the burden of disease (method 2B).

| Stressor | Health endpoint | Disability Weight | Duration (yrs) |
|---|---|---|---|
| Lead | Mild mental retardation | 0.36 | 77.6 |
| | Hypertensive disease | 0.2 | 3.6 |
| Road traffic noise | High sleep disturbance (HSD) | 0.07 [a)] | 1 |
| Railway noise | High sleep disturbance (HSD) | 0.07 [a)] | 1 |
| Aircraft noise | High sleep disturbance (HSD) | 0.07 [a)] | 1 |
| Ozone | Minor restricted activity days | 0.07 [b)] | 0.00274 (= 1 day) |
| | Cough days, children | 0.07 [b)] | 0.00274 (= 1 day) |
| | LRS days in children (excl cough) | 0.099 [c)] | 0.00274 (= 1 day) |
| $PM_{2.5}$ | Restricted activity days (RAD) | 0.099 [c)] | 0.00274 (= 1 day) |

[a)] Disability weight proposed by the WHO working group for noise impact assessment (confidence intervals 0.04–0.09).
[b)] Disability weight for pharyngitis.
[c)] Disability weight for lower respiratory infections (chronic sequelae).

TABLE 3-17: Crude estimated lag times (time between exposure to the onset of the disease) as used in the lag-time model (author estimates in years).

| Environmental stressor | Health endpoint | Crude estimated lag time (author judgement) years |
|---|---|---|
| Benzene | Leukaemia | 3 |
| Dioxins | Total cancer incidence | 10 |
| SHS | Tracheas, bronchus and lung cancers in non smokers | 30 |
| | Ischemic heart disease | 3 |
| | Asthma induction, adults (>21 yr) | 1 |
| | Asthma induction, children (<14 yr) | 1 |
| | Lower respiratory infections (<2 yr) | 0 |
| | Otitis media (<3yr) | 0 |
| Formaldehyde | Asthma aggravation (children) | 0 |
| Lead | IQ loss | 3 |
| | Hypertensive disease | 1 |
| Transport noise | High sleep disturbance (HSD) | 0 |
| | Ischemic heart disease (IHD) | 3 |
| Ozone | Total mortality (non-violent) | 1 |
| | Minor restricted activity days | 0 |
| | Cough days, children | 0 |
| | LRS days in children | 0 |
| $PM_{2.5}$ | Cardiopulmonary mortality * | 3 |
| | Lung cancer mortality * | 30 |
| | Total mortality (non-violent) * | 3 |
| | Chronic bronchitis | 1 |
| | Restricted activity days (RAD) | 0 |
| $PM_{10}$ | LRS symptoms days, children | 0 |
| | LRS symptom days, adults | 0 |
| Radon | Lung cancer | 30 |

* Overlapping end-points.

TABLE 3-18. Population sizes (in 2004; millions) in the defined target population groups (WHO, 2009b: Reference populations and live births).

| Populations (in millions) | Belgium | Finland | France | Germany | Italy | Netherlands | Total |
|---|---|---|---|---|---|---|---|
| All | 10.2 | 5.2 | 60.6 | 82.5 | 58.2 | 16.3 | 233.1 |
| Infants (<2 yr) | 0.229 | 0.113 | 1.530 | 1.452 | 1.090 | 0.399 | 4.8 |
| Toddlers (<3 yr) | 0.345 | 0.169 | 2.296 | 2.197 | 1.629 | 0.603 | 7.2 |
| Children (0-4 yr) | 0.58 | 0.28 | 3.83 | 3.69 | 2.71 | 1.02 | 12.1 |
| School children (5–14 yr) | 1.23 | 0.63 | 7.39 | 8.35 | 5.51 | 2.00 | 25.1 |
| Children (<14 yr) | 1.7 | 0.851 | 10.5 | 11.2 | 7.7 | 2.8 | 34.6 |
| Adults (>15 yr) | 8.4 | 4.3 | 49.4 | 70.5 | 50.0 | 13.3 | 195.8 |
| Adults (>15 yr) with chronic LRS * | 2.5 | 1.3 | 14.8 | 21.1 | 15.0 | 4.0 | 58.8 |
| Non-smoking adults (>15 yr) | 6.3 | 3.2 | 35.3 | 50.0 | 38.2 | 8.8 | 141.9 |
| Adults (>21 yr) | 7.7 | 3.9 | 44.7 | 64.7 | 46.4 | 12.1 | 179.6 |
| Adults (>27 yr) | 6.9 | 3.5 | 40.0 | 58.9 | 42.2 | 10.9 | 162.5 |
| Adults (>30 yr) | 6.5 | 3.3 | 37.8 | 56.1 | 39.8 | 10.3 | 153.8 |
| Adults (15-64 yr) | 6.7 | 3.5 | 39.5 | 55.4 | 38.7 | 11.0 | 154.7 |
| Working age (18–64 yr) | 6.3 | 3.3 | 37.1 | 52.5 | 37.0 | 10.4 | 146.7 |

* Adults with chronic respiratory symptoms estimated in CAFE to be 30% (Watkiss et al, 2005).

# 3.12 Data overview (tables)

TABLE 3-19. Summary of health endpoints, exposure units and exposure/response-relationships. Unless otherwise stated, both mortality and morbidity were estimated.

| Stressor | Health endpoint | Population | Exposure estimate | Unit of exposure | Type of ERF | Point estimate of ERF [a] | LCL (95%) | UCL (95%) | Reference(s) for ERF | Threshold | Calculation method [b] |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **Benzene** | Leukemia | All | Annual mean exposure | µg $m^{-3}$ | UR | $6.00 \times 10^{-6}$ | $2.20 \times 10^{-6}$ | $7.80 \times 10^{-6}$ | WHO, 2000a; IRIS 2003 | | 2A |
| **Dioxin** | Total cancer incidence | All | Daily intake of adults | pg/kg/d | UR | $1.00 \times 10^{-3}$ | $5.70 \times 10^{-4}$ | $5.10 \times 10^{-3}$ | NAS, 2004, IRIS, 2006, Leino 2008 | | 2A |
| **SHS** | Tracheas, bronchus and lung cancers [c] | Adult non-smokers | % of people exposed (= yes) | yes/no | RR | 1.21 | 1.13 | 1.30 | US S.G., 2006 | | 1A |
| | Ischemic heart disease | Adult non-smokers | | yes/no | RR | 1.27 | 1.19 | 1.36 | US S.G., 2006 | | 1A |
| | Asthma induction | Adult non-smokers | | yes/no | RR | 1.97 | 1.19 | 3.25 | Jaakkola et al., 2003 | | 1A |
| | Asthma induction | Children (<14 yr) | | parental y/n | RR | 1.32 | 1.24 | 1.41 | Cal-EPA, 2005 | | 1A |
| | Lower respiratory infections | Infants (<2 yr) | | parental y/n | RR | 1.55 | 1.42 | 1.69 | US S.G., 2006 | | 1A |
| | Otitis media | Toddlers (<3 yr) | | parental y/n | RR | 1.38 | 1.21 | 1.56 | Etzel et al., 1992; Cal-EPA 2005 | | 1A |
| **Formaldehyde** | Asthma aggravation (children) (morbidity only) | Toddlers (<3 yr) | Annual mean residential indoor concentration | µg $m^{-3}$ | RR | 1.017 | 1.004 | 1.025 | Rumchev et al., 2002 | 100 | 1A |
| **Lead** | IQ loss | Children (<5 yr) | Distribution of blood lead levels | µg/l | UR | 0.051 | 0.032 | 0.07 | Landphear et al., 2005 | 24 | 2B |
| | Mild mental retardation (morbidity only) | Children (<5 yr) | | µg/l | DS [d] | function | - | - | - | 24 | 2B |
| | Hypertensive diseases (morbidity only) | Adults/All | | µg/l | DS [d] | function | - | - | - | 50 | 2B |
| | Increased blood pressure | Adults/All | | µg/l | UR | $2.50 \times 10^{-2}$ | $1.70 \times 10^{-2}$ | $3.20 \times 10^{-2}$ | Fewtrell et al. 2003, Schwartz, 1995 | 50 | 2B |
| **Road traffic noise** | High sleep disturbance (HSD) (morbidity only) | All | Persons exposed to predefined exposure categories | Lnight (dB) | UR | function | function | function | Miedema et al., 2007 | | 2B |
| | Ischemic heart disease (IHD) | All | | Lday16h (dB) | OR | function | function | function | Babisch, 2006 | | 1A |
| **Railway noise** | High sleep disturbance (HSD) (morbidity only) | All | | Lnight (dB) | UR | function | function | function | Miedema et al., 2007 | | 2B |
| **Aircraft noise** | High sleep disturbance (HSD) (morbidity only) | All | | Lnight (dB) | UR | function | function | function | Miedema et al., 2007 | | 2B |

| Stressor | Health endpoint | Population | Exposure estimate | Unit of exposure | Type of ERF | Point estimate of ERF [a] | LCL (95%) | UCL (95%) | Reference(s) for ERF | Threshold | Calculation method [b] |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **Ozone** | Total mortality (non-violent) | Adults (>30 yr) | Population weighed ambient SOMO35 level | µg $m^{-3}$ | RR | 1.0003 | 1.0001 | 1.000 | WHO, 2006a | | 1A |
| | Minor restricted activity days (morbidity only) | Working age (18–64 yr) | | µg $m^{-3}$ | UR | 0.0115 | 0.0044 | 0.02 | Hurley et al., 2005, WHO 2006b | | 2B |
| | Cough days, children (morbidity only) | School children (5–14) | | µg $m^{-3}$ | UR | 0.093 | 0.019 | 0.22 | Hurley et al., 2005, WHO 2006b | | 2B |
| | LRS days in children (excl cough) (morbidity only) | School children (5–14) | | µg $m^{-3}$ | UR | 0.016 | -0.043 | 0.08 | Hurley et al., 2005, WHO 2006b | | 2B |
| **$PM_{2.5}$** | Cardiopulmonary disease | Adults (>30 yr) | Population weighted ambient level | µg $m^{-3}$ | RR | 1.0077 | 1.0020 | 1.0132 | Pope et al., 2002, WHO, 2006a | | 1A |
| | Lung cancer | Adults (>30 yr) | | µg $m^{-3}$ | RR | 1.012 | 1.004 | 1.020 | Pope et al., 2002, WHO, 2006a | | 1A |
| | Chronic bronchitis (new cases) | Adults (>27 yr) | | µg $m^{-3}$ | UR | $5.33 \times 10^{-5}$ | $1.70 \times 10^{-6}$ | $1.13 \times 10^{-4}$ | Hurley et al., 2005, WHO, 2006b | | 1A |
| | Restricted activity days (RAD) | 15–64 yr | | µg $m^{-3}$ | UR | 0.0902 | 0.0792 | 0.101 | Hurley et al., 2005, WHO, 2006b | | 2B |
| **Radon** | Lung cancer | All | Residential mean level | Bq $m^{-3}$ | RR | 1.0016 | 1.0005 | 1.0031 | Darby et al. 2005 | | 1A |

[a] These exposure response functions are all expressed per 1 unit of exposure.
[b] Different types of calculation methods were applied (see also paragraph 2.1):
1A: Deriving the PAF from epidemiological data; applying the PAF to total burden of disease data (WHO database)
2A: Indirectly calculating the PAF from the Unit Risk and background incidence; applying the PAF to total burden of disease data (WHO database)
2B: Using a Unit Risk to calculate Attributable Incidence; calculating the Burden of Disease: AI x DW x L
[c] The RR for spousal smoking is used as a proxy for any regular exposure (including at work).
[d] For lead, a shift in exposure distributions is linked to a unit risk approach. Further information provided in 3.6.

Function: No point estimate can be given, as the exposure response function is given by a more complex function.

AI = Attributable Incidence;
ARI = Acute respiratory infections;
Bq = Becquerel;
IHD = Ischemic heart disease;
Lday16h = noise level for day and evening;
LRS = Lower respiratory Symptoms;
PCB = Polychlorinated biphenyls,
PAF = Population Attributable Fraction;
PM = Particulate Matter;
RAD = Restricted activity days;
SHS = Second-Hand Smoke,
SOMO35 = sum of maximum 8-hour ozone levels over 35 ppb (70 µg/$m^3$);
UR = Unit Risk;
RR = Relative Risk;
yr = year; µg = microgram; mg = milligram; pg = picogram; kg = kilogram; d = day

TABLE 3-20. Summary of years and sources of exposure data.

| Stressor | Year(s) of original exposure data | Assumptions for trends estimation to 2004 | Exposure data source |
|---|---|---|---|
| Benzene | 2004 | National trend estimates when applicable | AirBase data for outdoor levels in 2004; national studies for indoors |
| Dioxins | 1997–2006 | No trend assumed | National data for intake |
| Second-hand smoke | 2008 | Available data fitted with power functions for trends | National and international survey data for exposures between 1990 and 2008 used for modelling 2004 data; |
| Formaldehyde | 1990–2005 | No trend assumed | National indoor concentration data |
| Lead | 1990–2005 | National trend estimates | National blood lead level data |
| Transport noise | 2007[1] | No trend assumed | EC Environmental Noise Directive data |
| Ozone | 2005 | No trend assumed | ECT/ACC spatial model based on AirBase observations and air quality maps |
| Particulate matter | 2005 | No trend assumed | |
| Radon | up-to 2005 | No trend assumed | RadonMapping project (http://radonmapping.jrc.ec.europa.eu) and the UNSCEAR 2000 Report |

[1] Target year of END data was set as 2007. The actual collected data contains subsets of data from various years.

TABLE 3-21. Summary of exposure values for benzene, dioxin and dioxin-likes, SHS, formaldehyde, lead and radon for 2004, ozone and PM for 2005.*

| Environmental stressor | Population group | Exposure metric | Unit | Belgium | Finland | France | Germany | Italy | Netherlands |
|---|---|---|---|---|---|---|---|---|---|
| Benzene | All | Average annual exposure concentration | µg $m^{-3}$ | 1.5 | 2.0 | 2.1 | 1.8 | 2.9 | 0.9 |
| Dioxins and dioxin-like PCBs | All | Average annual daily intake | pg TEQ/kg/d | 1.9 | 1.5 | 2.3 | 2.0 | 2.3 | 1.0 |
| SHS | All | Percentage of non-smokers | % | 74.9 | 74.2 | 71.5 | 71.0 | 76.5 | 66.6 |
| | Non-smokers | Percentage of non-smokers exposed | % | 32 | 14 | 25 | 31 | 30 | 30 |
| | Children | Percentage of non-smokers exposed | % | NA | 4 | 33 | 24 | 40 | 36 |
| Formaldehyde (threshold 100 µg $m^{-3}$) | All | Percentage of people exposed above 100 µg $m^{-3}$ | % | 0.20 | 2.30 | 0.20 | 0.30 | 0.05 | 0.05 |
| | All | Average indoor concentration above 100 µg $m^{-3}$ | µg $m^{-3}$ | 139 | 122 | 118 | 113 | 101 | 101 |
| Lead | All | Average blood concentration? | µg/l | 22 | 16 | 25 | 22 | 39 | 19 |
| | Adults | Percentage of adults exposed above 50 µg/l | % | 5.5 | 1.8 | 8.1 | 5.5 | 23.9 | 3.4 |
| | Adults | Average blood concentration for adults above 50 µg/l | µg/l | 68 | 68 | 68 | 67 | 76 | 65 |
| | Children 0–4 | Percentage of children exposed above 24 µg/l | % | 32.5 | 17.0 | 39.9 | 32.5 | 67.1 | 24.7 |
| | Children 0–4 | Average blood concentration for children above 24 µg/l | µg/l | 39 | 36 | 41 | 39 | 50 | 37 |
| Ozone | All | Annual sum of ozone maximum 8-h levels above 35 ppb (SOMO35) | µg $m^{-3}$ | 2 787 | 2 580 | 4 756 | 4 164 | 8 134 | 1 920 |
| $PM_{2.5}$ | All | Average annual ambient concentration | µg $m^{-3}$ | 18.7 | 9.1 | 12.3 | 16.0 | 19.6 | 18.7 |
| $PM_{10}$ | All | Average annual ambient concentration | µg $m^{-3}$ | 28.9 | 13.3 | 19.1 | 22.1 | 32.7 | 29.1 |
| Radon | All | Average annual indoor concentration | Bq $m^{-3}$ | 69 | 120 | 89 | 50 | 70 | 30 |

‘ Noise data are not included in this overview table, as they cannot be easily summarized due to categorial exposures. For noise data, please see paragraph 3.7.

# 4 Environmental burden of disease estimates

In the EBoDE project, we have calculated the environmental burden of disease for nine stressors in six countries, for the year 2004. The following paragraphs present and discuss the results of these calculations. The primary results are presented as undiscounted, un-age-weighted DALYs per million people. In addition to the results presented in this chapter, Appendix A presents the results per country.

Calculations were based on the most recent scientific evidence concerning population exposure-response functions, national exposure data, and WHO burden of disease data and methods for estimating disease burden where available. Even though the most recent scientific knowledge and data were used, many uncertainties and controversies remain (see Chapter 5). Results give only a crude ranking of environmental burden of disease associated with the stressors and need to be interpreted with caution.

## 4.1 Overall results

The results of the EBoDE project suggest that 3–7% of the standard WHO discounted age-weighted burden of disease in the participating countries is associated with exposure to the selected nine environmental stressors. The aggregate results for all stressors are shown in Figure 4-1, which also indicates the relative scientific strength of the evidence underlying the estimates. The quantitative uncertainty ranges provided in this figure are based on qualitative and semi quantitative evaluations of the uncertainties and author judgment.

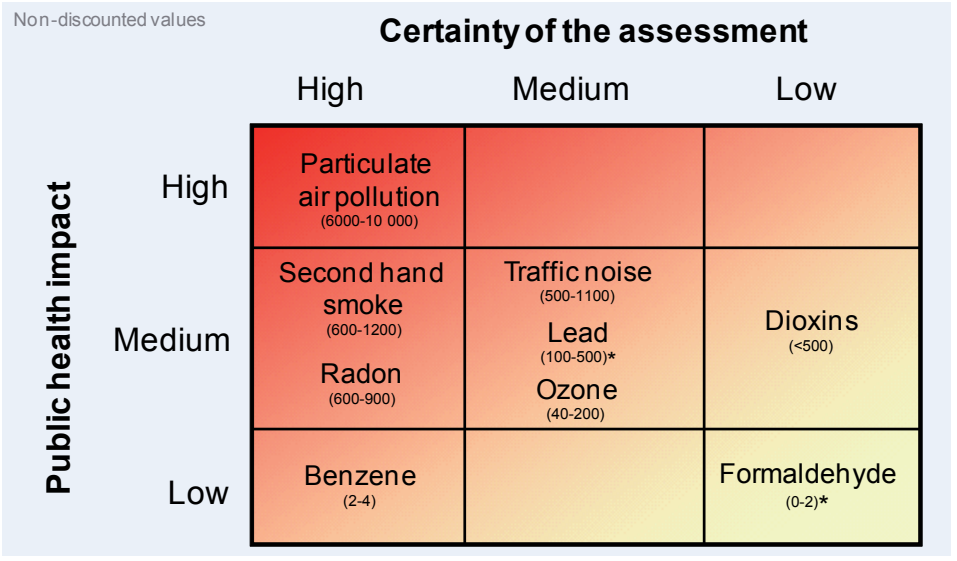



FIGURE 4-1. Relative public health impact of the selected environmental stressors in undiscounted un-ageweighted DALYs per population of a million in the participating countries. Numerical ranges reflect quantitative uncertainty in the average estimate. Variability between countries is in many cases much larger. (* =numerical model used in estimating threshold exceedances).

Figure 4-2 shows the burden of disease related to the nine stressors proportional to each other. As can be seen from this figure, particulate matter (using $PM_{2.5}$ as indicator) is estimated to be the leading factor associated with 6.000 to 10.000 non-discounted DALYs per million people. Overall, $PM_{2.5}$ is responsible for approximately two thirds of the environmental burden of disease related to the nine stressor evaluated in EBoDE. After PM, transportation noise, second hand smoke and radon contribute to the largest share of the environmental burden of disease. These four factors together are estimated to be responsible for over 90 % of the total studied environmental burden of disease.

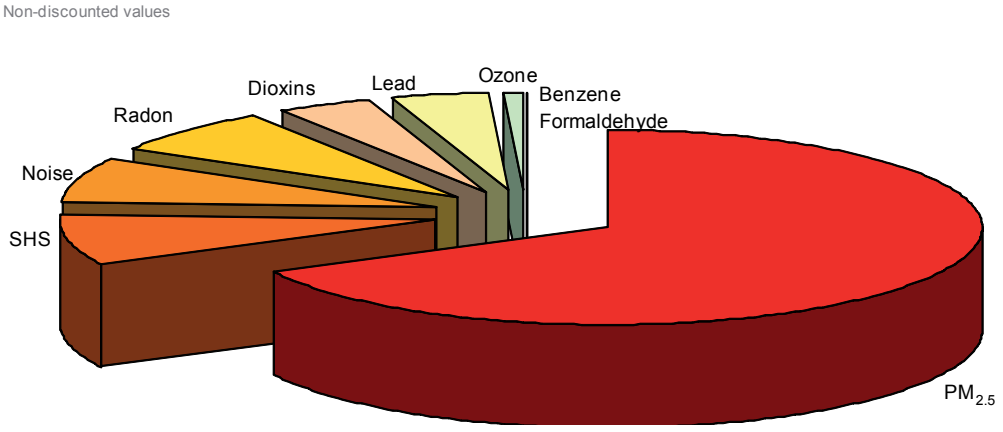



FIGURE 4-2. Relative contribution of the nine targeted stressors to the burden of disease (undiscounted, un-age-weighted DALYs) attributed to these stressors, average over the six participating countries.

The quantitative results of the environmental burden of disease calculations per stressor and health endpoint, averaged over the six countries, are presented in Table 4-1. The total results aggregated per stressor are shown in Table 4-2.

When we look at effects on mortality, the studied nine factors are estimated to be associated with approximately 1.6 million years of life lost (YLL, non-discounted, not age-weighted) in the participating countries in the 2004, or 6 900 YLL per a million inhabitants (Table 4-3). For most of the stressors, the impacts are dominated either by morbidity (formaldehyde, lead and traffic noise) or by mortality (benzene, dioxins, and radon). We realize that the selection of health endpoints and further assumptions are partly responsible for this effect. For dioxin, for example, all DALYs are due to mortality (i.e. YLL), as all cancers are assumed to be fatal in our estimates.

TABLE 4-1. Burden of disease in DALYs per million people for each stressor and endpoint, averaged over the participating countries.

| Stressor | Endpoint | Non-discounted | | Discounted with lag | | Difference |
|---|---|---|---|---|---|---|
| | | Total DALY | DALY per million | Total DALY | DALY per million | % |
| Benzene | Leukemia | 741 | 3 | 341 | 1 | -54 % |
| Dioxin | Total cancer incidence | 112 332 | 482 | 42 429 | 182 | -62 % |
| SHS | Lung cancers in non smokers | 19 381 | 83 | 3 989 | 17 | -79 % |
| | Ischaemic heart disease | 157 919 | 678 | 68 154 | 292 | -57 % |
| | Asthma induction, adults (>21 yr) | 30 363 | 130 | 23 892 | 103 | -21 % |
| | Asthma induction, children (<14 yr) | 10 481 | 45 | 9 988 | 43 | -5 % |
| | Lower respiratory infections (<2 yr) | 1 414 | 6 | 595 | 3 | -58 % |
| | Otitis media (<3 yr) | 654 | 3 | 270 | 1 | -59 % |
| Formaldehyde | Asthma aggrevation (<3 yr) | 20 | 0 | 16 | 0 | -23 % |
| Lead | IQ loss | 106 621 | 457 | 31 453 | 135 | -70 % |
| | Increased blood pressure | 2 930 | 13 | 2 736 | 12 | -7 % |
| Road traffic noise | High sleep disturbance (HSD) | 167 916 | 720 | 167 916 | 720 | |
| | Ischaemic heart disease (IHD) | 13 105 | 56 | 5 890 | 25 | -55 % |
| Railway noise | High sleep disturbance (HSD) | 12 253 | 53 | 12 253 | 53 | |
| Aircraft noise | High sleep disturbance (HSD) | 7 202 | 31 | 7 202 | 31 | |
| Ozone | Total mortality (non-violent) | 8 365 | 36 | 8 122 | 35 | -3 % |
| | Minor restricted activity days | 4 484 | 19 | 4 484 | 19 | |
| | Cough days, children | 6 042 | 26 | 6 042 | 26 | |
| | LRS days in children | 1 470 | 6 | 1 470 | 6 | |
| $PM_{2.5}$ | Cardiopulmonary mortality | 1 081 750 | 4 642 | 499 063 | 2 141 | -54 % |
| | Lung cancer mortality | 348 623 | 1 496 | 72 512 | 311 | -79 % |
| | Chronic bronchitis (COPD) | 289 711 | 1 243 | 151 089 | 648 | -48 % |
| | Restricted activity days (RAD) | 61 003 | 262 | 61 003 | 262 | |
| Radon | Lung cancers | 194 277 | 834 | 40 563 | 174 | -79 % |

* In calculations according to method 2B (see Table 3-19 and section 2.1), discounting, when applicable, was used without age-weighing.

TABLE 4-2. Aggregate burden of disease by stressors per country for 2004/2005, in undiscounted, un-age-weighted DALYs per million people.

| Stressor | Belgium | Finland | France | Germany | Italy | Netherlands | Average |
|---|---|---|---|---|---|---|---|
| Benzene | 2.5 | 3.0 | 3.4 | 2.7 | 4.2 | 1.6 | 3.2 |
| Dioxin | 453 | 330 | 586 | 466 | 483 | 242 | 482 |
| SHS | 1 110 | 891 | 550 | 1 235 | 975 | 749 | 945 |
| Formaldehyde | 0.2 | 1.6 | 0.1 | 0.1 | 0.0 | 0.0 | 0.1 |
| Lead | 298 | 118 | 461 | 235 | 946 | 217 | 470 |
| Traffic noise | 437 | 371 | 1 483 | 591 | 734 | 775 | 860 |
| Ozone | 52 | 47 | 81 | 73 | 138 | 34 | 87 |
| PM2.5 | 10 462 | 4 602 | 4 572 | 8 384 | 9 378 | 8 322 | 7 642 |
| Radon | 1 078 | 926 | 1 146 | 620 | 866 | 453 | 834 |
| Total | 13 892 | 7 289 | 8 883 | 11 608 | 13 525 | 10 793 | 11 324 |

TABLE 4-3. Mortality and morbidity components of the non-discounted burden of disease by stressor in the six participating countries per population of a million. Cases where over 90 % of the burden of disease is caused by either morbidity or mortality are highlighted in color blue and bold.

| Stressor | Deaths[a] per year | YLD per year | YLL per year | DALY per year | YLL % |
|---|---|---|---|---|---|
| Benzene | 0.2 | 0.1 | **3.1** | 3.2 | **97 %** |
| Dioxin | 30 | 36 | **446** | 482 | **93 %** |
| SHS[b] | 78 | n/a | n/a | 945 | n/a |
| Formaldehyde | 0.0 | **0.1** | 0.0 | 0.1 | **0.6 %** |
| Lead | 0.0 | **470** | 0.1 | 470 | **0.02 %** |
| Traffic noise | 4.2 | **810** | 50.3 | 860 | **5.8 %** |
| Ozone | 36 | 51 | 36 | 87 | 41 % |
| PM2.5 | 516 | 2 063 | 5 580 | 7 642 | 73 % |
| Radon | 51 | 16 | **817** | 834 | **98 %** |
| Total | 715 | 3 446 | 6 933 | 11 324 | 61 % |

[a] Numbers of deaths are not valid indicators for some effects (see Chapter 5).
[b] YLL+YLD split not available for SHS due to the different method of calculation.

## 4.2 Results by stressor

Figure 4-3 at the end of this paragraph shows the results (not discounted, not age-weighted) per stressor and per country.

### 4.2.1 Benzene

Based on the available information about leukaemia, the total impact of benzene on public health is estimated to be low (see Figure 4-1 and Figure 4-2). Benzene impacts are the highest in Italy and the lowest in the Netherlands. The great quantity of two-wheelers with two-stoke engines in Italy may partly explain the high benzene exposures. The low benzene related burden of disease in the Netherlands is due

to the lowest exposures. In France, data reflect a large number of dwellings, while in other countries data are limited to a smaller number of monitored houses; thus the high burden estimate for France is based on more reliable data than the other estimates. In addition, the presence or absence of tobacco smoke in indoor environments is not always reported, making comparison more difficult. This at least partly explains the higher levels in Finland, where benzene from smoking was included and was estimated to contribute approximately one third of the exposures (see also Table 3-1 in section 3.2).

Further sources of uncertainty in the benzene related burden of disease estimates relate mainly to the availability of exposure data, exclusion of other health effects than leukaemia, and the potential interaction of benzene with other components of tobacco smoke.

### 4.2.2 Dioxins and dioxin-like PCBs

The relative burden of disease related to dioxins is estimated to be medium (see Figure 4-1 and Figure 4-2), however, uncertainties are large. Effects of dioxins cannot easily be distinguished from other occupational risk factors; low-dose effects are very difficult to assess; thresholds for effects are mostly unknown; and exposure data are often only indirectly available. Our estimates only include effects of dioxins on total cancer incidence. This is a rather crude aggregate end-point. In addition, each cancer case was assumed to be fatal during the first year, which may also have lead to overestimation. Also, numerous more specific health end-points (see paragraph 3.3) were not modelled. Therefore, it is yet unclear whether our estimates over- or underestimate the total burden of disease.

The burden of disease related to dioxin exposure is relatively low in Finland and the Netherlands, and highest in France. Potential explanations for this are different eating habits and differences in food contamination. But also the different methods to evaluate the daily intake may contribute to an unknown extent of uncertainty.

### 4.2.3 Second hand smoke

The burden of disease related to SHS is estimated to account for 600-1200 DALYs per million people (medium impact). As outlined in paragraph 3.4, not all health effects could be included in the calculations due to unavailability of statistics. Besides, uncertainties in our estimates relate to e.g. survey-based exposure measurements (as opposed to measuring personal exposures), relative risks and the various assumptions made in the method (e.g. smokers are not susceptible to SHS). The provided range around the best estimate has been based on a sensitivity analysis varying the main assumptions made in the method. Nonetheless, most evidence for SHS-related impacts is fairly consistent, and the estimates of the burden of disease are considered relatively stable.

Burden of disease from second hand smoke are remarkably low in France and high in Germany. Potential explanations for the higher levels in Germany are the slightly higher exposure levels combined with higher prevalence of the relevant diseases, such as ischaemic heart disease, which increases the susceptibility to the risk factor.

The disease burden due to SHS is still substantial. Trends, however, are decreasing due to European-wide implementation of smoke-free policies to reduce SHS exposure. The predicted exposure reductions between 2004 and 2010 as presented in Tables 3.7 and Table 4-5 are quite significant. 100% smoke-free policies have shown significant reductions in mortality and should be implemented in all indoor workplaces, public places and public transports. Complementary educational strategies may be necessary to extend the protection of children and adult non-smokers at home.

### 4.2.4 Formaldehyde

The burden of disease related to formaldehyde, based on asthma incidence in children under 3 years of age, is estimated to be relatively low. However, the consistency of the knowledge base is low, with uncertainties related to the difficulty of establishing a threshold for effects, a lack of epidemiological data and a large discrepancy in widely used models.

Formaldehyde exposures are remarkably high in Finland (see also Figure 3-3). The absolute average concentration levels do not vary so drastically between the countries. However, because a threshold of

100 μg $m^{-3}$ was applied, the relative differences between countries increased. The formaldehyde levels in Finland are higher than in many other developed countries due to the types of construction materials used and the relatively tightly sealed buildings.

The risk estimates for formaldehyde depend strongly on the chosen threshold level. Even though identified as a known human carcinogen, it is likely that cancer effects are negligible in Europe, due to the fact that almost all exposures are below the threshold level as proposed by WHO (2000a, 2011). The currently available exposure data cannot be used for reliably estimating the fraction of the population that is exposed to formaldehyde level exceeding the threshold levels. That is caused by the fact that current formaldehyde monitoring techniques are not suitable for detecting peaks of exposure, which may occur during some domestic tasks or soon after home refurbishment, especially in the absence of proper ventilation. Other common sources of formaldehyde, such as widespread use of fragrances and SHS, may also cause exceedance of the threshold value. This may cause underestimation of the true formaldehyde-related burden of disease.

### 4.2.5 Lead

Lead is estimated to contribute to 100-500 DALYs per million people (medium impact, see Figure 4-1 and Figure 4-2). These estimates are based on a limited population representativity and partly older data with uncertain trend estimations (see paragraph 3.6). Other uncertainties relate to the availability of dose-response functions over the complete exposure spectrum, and the aggregation of effects. Lead impacts have only been based on IQ loss and mild mental retardation. At least at higher exposure levels that prevailed during earlier decades, lead exposures were associated with a larger number of health endpoints, ranging from hearing impairment to kidney failures. However, the evidence for these effects at the prevailing low level exposures is very limited (WHO, 2007b). Nonetheless, our burden of disease estimates may underestimate the true lead-related burden of disease (see also Chapter 5).

Lead exposures are the highest in Italy. One of the most important reasons for this may be the relatively old age group in which blood lead levels were measured. As was shown in paragraph 3.6, the blood lead levels in Italy were presented for people aged 18–64 years. In the Netherlands, in contrast, the sample included children aged 1–6 years. Because lead accumulates in the body over the years, this is probably the most important reason for lead-related burden of disease in the Netherlands being relatively low and in Italy relatively high. In addition, the data for Italy (Apostoli *et al.*, 2002) are quite old (from 2000). Lead levels are expected to be lower in 2004.

Some other uncertainties that may affect the comparability of lead results among countries include the fact that data from Finland, Germany and the Netherlands are not representative for the age-group considered (let alone for the whole population); and that some countries provided a Geometrical Mean (GM) instead of an Arithmetical Mean (AM). The GM is expected to be lower than AM, because there are few high values within the samples.

### 4.2.6 Transportation noise

Since so many people are exposed to noise, the total associated disease burden is substantial despite the relatively small disability weights (0.04-0.09) and is estimated to cause an undiscounted, un-age-weighted average of 860 DALYs per million people. Transportation noise plays a great role in each included country but there are numerous differences between the countries. DALYs range from about 371 per million inhabitants in Finland (less densely populated and highly urbanized but with only very few cities with more than 250 000 inhabitants) up to 1 483 DALYs per million people in France (due to the fact that only data for the greater Paris area were available). These differences point out some major limitation due to incomplete exposure data from environmental noise directive reporting (for Belgium e.g. only available for the Flanders region) and due to different population and traffic densities in the countries.

Furthermore, a strong limitation results from the fact that exposure data reported in the first stage of END-reporting represent only "hot spots" where noise levels are supposed to be much higher than in the countryside. Therefore, the results from the calculations are an underestimation of the total burden of a country but may be overestimating the risks when applied for the whole population of a country

(as done when normalized by million inhabitants). Country comparability consequently is affected by the variability of representativity (more representative in highly urbanized countries). In addition, only exposure levels above $L_{night}$ 50dB ($L_{den}$ 55 dB) were available from the END database, so no health impacts could be calculated for the lower exposure levels.

Of all the estimated DALYs for transport noise, 94 % result high sleep disturbance (HSD). Because so many people are estimated to suffer from HSD, the DALYs are very sensitive to changes in the disability weight (with confidence intervals ranging from 0.04 to 0.09) and less sensitive to changes in exposure levels (e.g. due to other constant used for conversion of $L_{den}$ to $L_{night}$) or the exposure-response-functions. Further uncertainties result e.g. from missing exposure-response-functions for certain transport sources.

Other potential sources of uncertainty relate to individual and societal factors affecting noise levels indoors (such as regular location of sleeping rooms, window opening habits, window insulation etc.), and exogenous factors affecting those habits (such as climatic prerequisites and house ownership).

### 4.2.7 Ozone

The relative impact of ozone on public health is medium (40-200 undiscounted, age-weighted DALYs), see Figure 4-1 and Figure 4-2. Even though not all health effects could be included, the selected morbidity health endpoints are estimated to account for 90% of the total effects. Uncertainties in the calculations relate, amongst other issues, to the estimated number of years of life lost for mortality.

Ozone impacts are highest in the Mediterranean countries, represented here by Italy, as can be expected. Levels in the Netherlands are the lowest, probably because meteorological factors and relatively high levels of nitrogen oxide pollution consuming atmospheric ozone in congested areas.

Ozone levels have been slowly increasing during the last decade and due to the secondary nature of ozone air pollution, reduction of the exposures is challenging. From the point of view of health impact assessment the duration of loss of life at death is a key factor that hopefully will be estimated more accurately in future.

### 4.2.8 Particulate matter (PM)

In the six participating countries PM is estimated to cause a loss of 1.8 million DALYs annually, including 1.3 million years of life lost due to mortality (73% of the total DALYs). Overall 67 % of the estimated environmental burden of disease in the EBoDE study was explained by exposure to $PM_{2.5}$ making it the most significant environmental factor affecting public health (see Figure 4-1 and Figure 4-2).

Uncertainties in the PM related burden of disease relate to the exposure-response functions for e.g. chronic bronchitis; and the potential of double counting of morbidity effects by combining the restricted activity days and lower respiratory symptom days. Overall, the PM epidemiology has been most thoroughly reviewed of the stressors included in this study. In the context of the CAFE study the estimates calculated for total and cause specific mortality were debated; the scientific evidence and mechanistic understanding of the causal processes are stronger for cardiopulmonary causes, but the effect estimates are higher for total non-violent mortality. We followed the CAFE approach to report the slightly lower cause specific results, which also can be used to meaningfully look at the ratio of mortality versus morbidity impacts.

Particulate matter impacts are the lowest in Finland and France and the highest in Belgium and Italy, which are known to be hotspots of particulate matter air pollution.

Annual average concentrations of $PM_{10}$, monitored in EU urban background locations, show no significant decrease over the period 2000–2007 (Airbase, 2009). Available data are still too limited for reliable assessment of $PM_{2.5}$ trends, but particulate matter exposures are expected to have a very slight downward trend due to the improvements in vehicle engine technology and emission controls in industry and energy production. However, the emissions of resuspended particles created by road traffic are expected to continue to grow due to the increasing traffic volumes.

A significant reduction in current PM levels could be achieved only if all feasible emission reduction measures were implemented (the maximum feasible reduction scenario) (WHO, 2010c). This is a challenge for the current policy as it involves the implementation of new technologies (e.g. low-emission diesel cars) and the creation of conditions that support individual behavioural change. The use of health impact

assessments as a standard tool in air quality and health policy, together with follow-up programmes (accountability) focusing on health consequences, is urgently needed across the Region (WHO, 2010c).

### 4.2.9 Radon

Radon is estimated to contribute 600-900 DALYs (undiscounted) per million people in the participating countries (see Figure 4-1 and Figure 4-2).

The radon related burden of disease is the highest in France and Belgium; and lowest in the Netherlands. These differences are mainly caused by the differences in geological substrates beneath houses and the use of different building materials. For example, in Belgium the average radon concentration in houses is 35 $Bq/m^3$ for Flanders and 70 $Bq/m^3$ for Wallonia (southern part of the country). This difference is mainly due to the larger uranium concentration in rocks present in the southern part. On top, there are often relatively more cracks in these rocks simplifying the release of radon. Concentrations of radon in houses of more than 400 $Bq/m^3$ are sometimes measured in the southern part of Belgium. Measures as placing an impermeable screen and adapting the ventilation system may substantially reduce the radon indoor concentration.

For radon, we have used a relative risk to calculate cases of lung cancer (see paragraph 3.10). However, for this specific stressor, it is also possible to calculate attributable cases using a unit risk. The UR model is presented as part of the uncertainty analyses in Chapter 5.

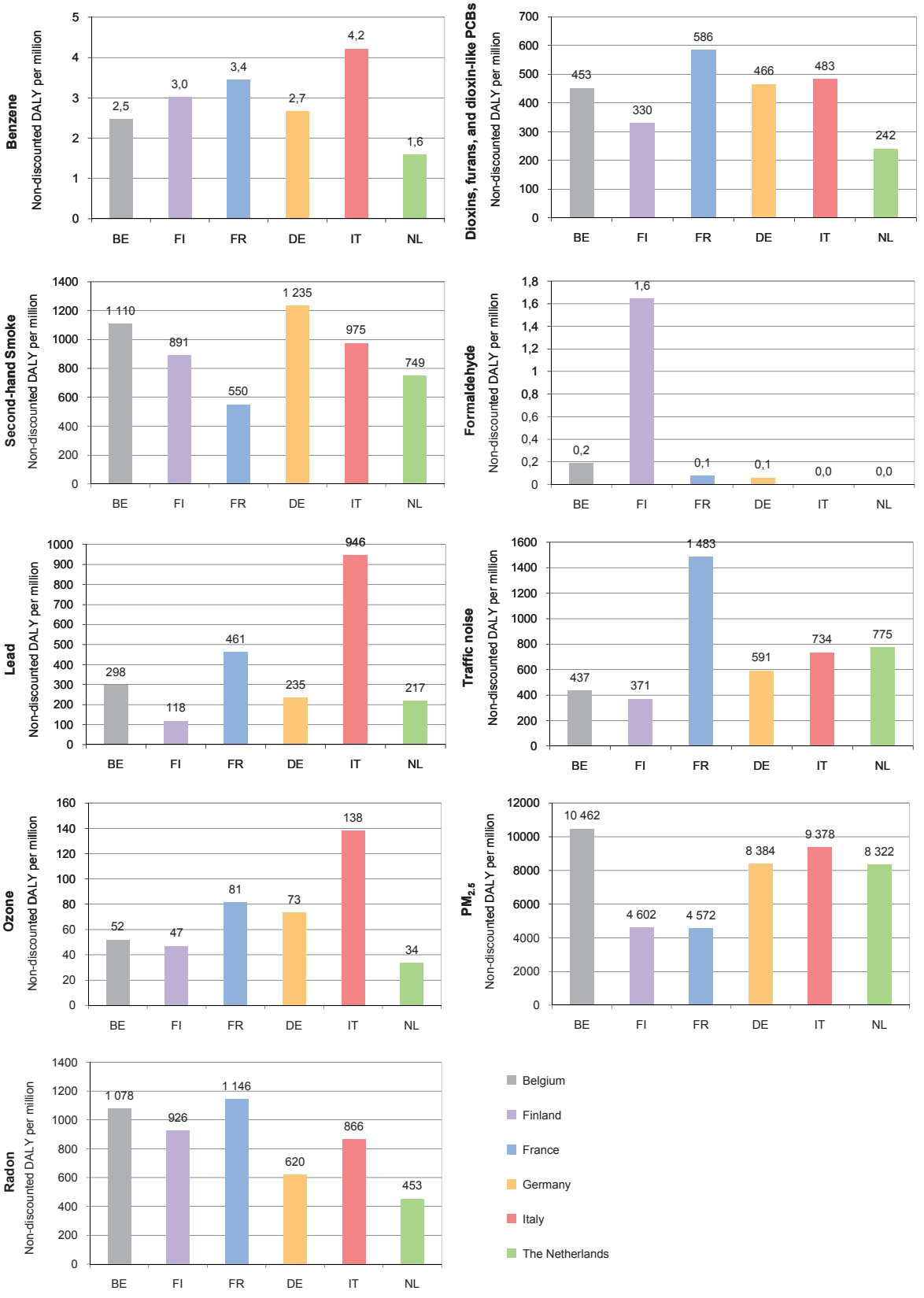



FIGURE 4-3. Undiscounted, un-age-weighted DALYs per million people per country.

## 4.3 Results by country

Using the WHO global burden of disease database (see section 3.11), we have estimated for each country what the fraction of the total burden of disease is that can be attributed to the nine environmental stressors considered in EBoDE. The results are shown in Figure 4-4.

This environmental fraction ranges from 3% in Finland to 6.5% in Italy. Relatively, Finland has the largest BoD and the smallest EBD, while for Italy this is vice versa.

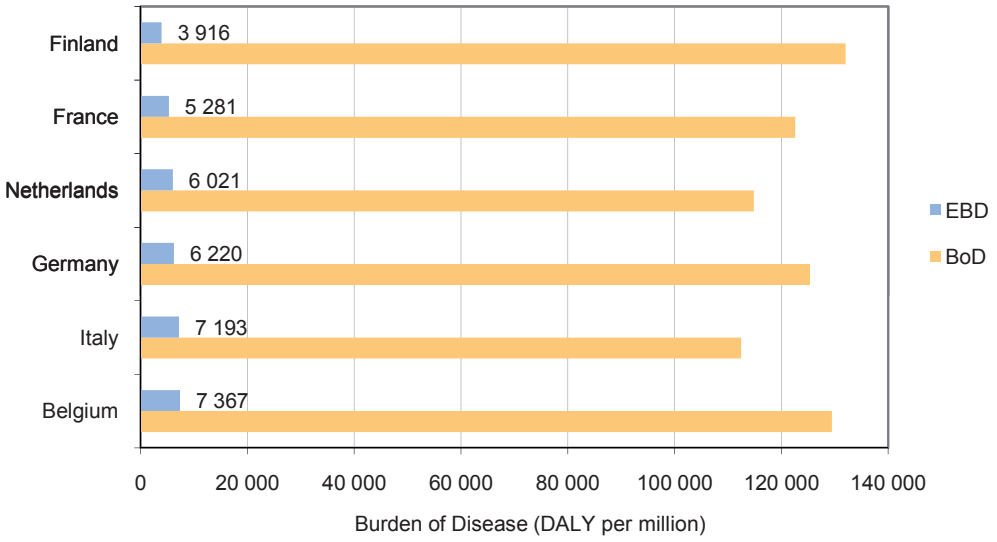



FIGURE 4-4. Environmental fraction attributable to the studied nine stressors (in blue) of the total burden of disease in the participating countries (discounted age-weighted DALYs per million people).

The results for individual countries are presented in appendix A. These can be used to look at the environmental burden of disease from a country perspective, and to see which factors contribute substantially in specific countries, relative to their contribution in other countries.

When comparing the relative contribution of all stressors per country, radon has the highest relative contribution in Finland. Relatively, the disease burden in France has the lowest contribution from PM. SHS comes only on the fifth place in France, whereas in other countries SHS is usually in the top of the contributors. On the other hand, traffic noise relatively has the largest contribution in France, due to the fact that only data from agglomerations are included in our estimates for France.

Relatively, the disease burden in Italy has the highest contribution of ozone. Italy is also the only participating country where the impact of lead exceeds that of ozone. Formaldehyde did not exceed the threshold in Italy, resulting in zero estimates. Also in the Netherlands, formaldehyde exposures did not exceed the threshold, resulting in zero estimates.

In Belgium, the impact of air pollution by particulate matter is relatively largest. Flanders is the Western European hot spot for PM pollution (IIASA, 2004). This situation is caused by a high population density, an intense industrial activity and a large volume of transit traffic linked to important harbors. The daily PM10 standard, enforced by the European Commission (1999/30/EC) is still being exceeded more times than allowed in Flanders.

## 4.4 Trends and policy implications

The main results of EBoDE are calculated for the year 2004 (2005 for PM and ozone). This was one of the latest years for which exposure and health data were still relatively completely available. The original objective of the project was set as estimation of the environmental burden of disease also for the present situation, year 2010. However, due to difficulties in collecting national data, reliable trend analysis for all the stressors in all the participating countries proved to be too challenging for the given time and resources. For many of the stressors sufficient data for reliable trend analysis was not available (e.g. formaldehyde, lead) or the variability was too large for identifying trends with statistical significance (e.g. ozone, dioxins).

However, in order to gain insight into the use of the EBD assessment methodology and the potential use of burden of disease estimates in developing and evaluating environmental policies, we also estimated exposure trends for the year 2010 (Table 4-4). Country specific factors affecting the trends were not evaluated due to the lack of data and statistical difficulties in the trend estimation. Only for second-hand smoke exposures, national trends were derived separately (see Table 4-5 and Figure 3-2).

TABLE 4-4. Crude estimated exposure trends from 2004 to 2010.

| Stressor | Estimated trend from 2004-2010 | Remarks |
|---|---|---|
| Benzene | -2% per year | Model based on trends in outdoor data. Decreasing indoor smoking may also lead to lowering exposures from SHS |
| Dioxin | No trend | Large variability and limited data availability hinder identification of a reliable trend |
| Second-hand smoke | -4% per year (see country data below) | Power model based on numerous data points from national and international surveys conducted between 1990 and 2008. |
| Formaldehyde | No trend | Large variability and limited data availability hinder identification of a reliable trend |
| Lead | No trend | Trend could also be slightly lowering. |
| Noise | No trend | Trend could also be slightly increasing. |
| Ozone | No trend | Increasing trends may occur in rural areas. Large year-to-year variation |
| PM2.5 | -2% per year | AirBase analysis and recommendation by ETC/de Leeuw (personal communication) |
| Radon | No trend | Changes in building stock and construction structures extremely slow |

TABLE 4-5. Estimated exposures for 2010 for second-hand smoke in children and non-smoking adults.

| Year 2010 | Children | | Adults | | Women | | Men | |
|---|---|---|---|---|---|---|---|---|
| | Lower* [%] | Upper* [%] | Lower [%] | Upper [%] | Lower [%] | Upper [%] | Lower [%] | Upper [%] |
| Belgium | *NA* | *NA* | 25 | 30 | 24 | 29 | 25 | 31 |
| Finland | 3 | *NA* | 14 | 14 | 14 | 14 | 14 | 14 |
| France | 17 | 27 | 13 | 22 | 15 | 25 | 11 | 19 |
| Germany | 20 | *NA* | 20 | 28 | 19 | 27 | 21 | 29 |
| Italy | 29 | *NA* | 22 | 26 | 19 | 23 | 24 | 28 |
| Netherlands | 14 | 28 | 18 | 27 | 16 | 23 | 21 | 31 |

*NA*: Adequate data not available
* Lower and upper estimates correspond to different computations of survey data.

# 5 Uncertainties and limitations

Assessment of uncertainties is essential in a comparison of quantitative estimates that are based on data from heterogeneous sources and slightly varying methods. Due to the wide range of data sources and models and the limited resources within the EBoDE project, systematic analysis of all uncertainties was not possible. However, we were able to assess a number of specific sources of uncertainties in more detail as part of the work, yielding some insights into the reliability of the overall assessment.

The studied health impacts span approximately four orders of magnitude in size from few DALYs per million to almost 10 000 DALYs per million. The overall ranking of the environmental stressors seems to be rather robust against the relatively large uncertainties in individual estimates or methodological choices like discounting and age-weighing. However, some of the estimated ranges are overlapping. This concerns especially second hand smoke, radon and transportation noise that compete for the questionable honour of being the second most important environmental stressor in the participating countries. Among these stressors the differences are smaller than the corresponding uncertainties of the estimates.

The health state of an individual person is the result of a complex mixture of genetic, environmental and behavioural factors. In a typical case of death, numerous factors play together. This means, for example, that a single death caused by a cardiovascular disease could be avoided by either reducing air pollution, or a better diet, or more physical activity. Therefore, if the individual attributable fractions are summed over a number of risk factors, a value over 100% may sometimes be found. For this and other reasons, it has been argued that death counts are not suitable for quantification of the impacts (Brunekreef *et al.*, 2007). Therefore the authors recommend to mainly use aggregate population measures of health like DALYs, YLLs and YLDs.

This chapter presents the quantitative results for selected sources of uncertainties and discusses the project limitations and author judgment of the reliability of the ranking.

*Uncertainties per stressor and comparison with other studies*

A list of the most important sources of uncertainty for each stressor in the EBoDE calculations is provided in Table 5-1. Some of these are further explained below. In addition, we will compare our estimates to results of a selection of similar studies. Comparison of different studies on environmental burden of disease helps to understand the role of various methodological and strategic selections made in each study, like the selection of stressors or health endpoints.

**Benzene.** No international burden of disease study utilizing DALYs for benzene was identified. Some studies using exposure proxies like proximity of gasoline stations have studies health impacts with inconsistent results.

**Dioxins.** Our calculations were based on the same approach as applied earlier by Leino et al (2008), but we utilized an updated cancer slope factor that is approximately seven times higher than the one used by Leino et al. Leino et al. did the calculations for Finland only. The work presented here also updated the exposure estimates in order to allow for good international comparability, yet some differences between the national intake estimation methods remained.

**SHS.** Our burden of disease calculation for SHS was based on a WHO model (Öberg et al., 2010). The exposure estimates were updated against available national and international data sources for the target year 2004, but otherwise the results are comparable with the WHO assessment. Other recent estimates of burden of disease for SHS were also available for Germany (Heidrich *et al.* 2007; Keil *et al.* 2005), which provided similar results as the current estimates.

**Formaldehyde.** No international burden of disease study utilizing DALYs for formaldehyde was identified. WHO Guidelines for Indoor Air Quality used eye irritation as the main health end-point in setting a safe exposure level. However eye irritation cannot be directly used as a health end-point in burden of disease calculation because no disability weight exists and therefore was not accounted for here. Scientific evidence on the association between formaldehyde and childhood asthma is not considered sufficiently consistent yet; thus the results presented here must be taken as provisional estimates of the magnitude of the health impacts, to be confirmed by future studies.

**Lead.** The calculation focused on mild mental retardation and hypertensive disease only. WHO EBD estimates (Fewtrell et al., 2003) include cerebro-vascular and other cardiovascular diseases besides hypertensive disease; therefore the current estimates for lead are slightly lower than the WHO estimates.

**Transportation noise.** Burden of disease estimation for transportation noise is currently under active development. The estimates presented here were based on the only available international exposure data source, the first stage version of the European Noise Directive database (2007), which is not conclusive yet. Therefore it is clear that most of the exposures for transportation noise are underestimated. In some studies annoyance and cognitive impairment have been used as an additional health end-points for environmental noise. However, due to the selected more limited definition of 'health' as ICD-classified health states used in our assessment, annoyance and cognitive impairment were not included here. Only road, rail and air traffic exposures were included; many other sources also contribute to the noise exposures. Low exposures below the END data collection limits (50 and 55 dB) were not included. For these reasons it can be expected that when these limitations are solved, the impact estimates will increase.

**PM and ozone.** The methodology developed in Clean Air for Europe -project (CAFE) (Hurley et al., 2005) was applied using updated exposure estimates. The updated exposures are based on ambient air quality monitoring data that contain, besides the anthropogenic components that CAFE focused on, also natural sources of $PM_{2.5}$. The spatial resolution of the updated model is 25 times higher (grid size 10x 10 km² instead of 50x50 km²). Compared to the CAFE estimates the current work adds estimation of the impacts in DALYs. The WHO Environmental Burden of Disease programme uses a non-linear exposure-response function (Ostro, 2004) that at higher exposures yields lower impacts than the linear CAFE model. WHO also sets a threshold level at 7.5 $\mu g\ m^{-3}$.

**Radon.** The exposure estimation and dose-response models are based on earlier international analysis conducted by Darby et al. (2006). In comparison with that the current work added estimation of the impacts in DALYs. Comparison of UR and RR models yielded similar results. The results using the RR approach, accounting for the national differences in the background rates of lung cancer, were selected for reporting.

TABLE 5-1. Identified sources of uncertainty in EBoDE calculations.

| | Excluded health endpoints and related assumptions | Exposure data | Exposure response function | Calculation method | Level of overall uncertainty [a] | Likely over- or underestimation [b] |
|---|---|---|---|---|---|---|
| Benzene | Anaemia; genotoxicity; other blood cancers than leukaemia; leukaemia morbidity; effects on the immune, endocrine and nervous system; acute effects.<br><br>All cases of leukaemia assumed to be fatal. | Population representativity varies<br>Differences in number of dwellings<br>Different types of measurements (indoor/outdoor; in – or excluding SHS, etc)<br>Sampling times differ | No specific relationships for children used (i.e. same UR used for all ages) | UR method of calculating PAF leads to overestimation because all cases are assumed to be fatal. | * | Underestimation due to excluded health endpoints, but overestimation due to UR method |
| Dioxins (plus furans and PCBs) | Effects on the immune, endocrine, reproductive and nervous system; tooth and bone defects.<br><br>All cases of cancer assumed to be fatal. | Indirect exposure metrics<br>Different measurement methods<br>Daily intake of food depends on age, body weight and eating habits<br>Exposure varies within countries (from region to region) | Uncertain cancer slope factor<br>Assumed additivity of the toxicity of different types | UR method of calculating PAF results in overestimation because all cases are assumed to be fatal. | *** | Underestimation of non cancer effects,<br>Overestimation of cancer effects (all lethal) |
| Second Hand Smoke | Sudden infant death syndrome; low birth weight; reduced pulmonary function among children; acute irritant symptoms. | Data from different years and consequent temporal interpolation<br>Differing definitions of exposures<br>Data gaps for some countries | ERF from earlier decades when questionnaire responses may have been less sensitive<br><br>Odds ratios used as RR estimates | Various assumptions made, e.g. smokers are not susceptible to SHS | * | Underestimation due to excluded endpoints<br>Potential overestimation due to increased questionnaire sensitivity |
| Formalde-hyde | Acute symptoms; nasopharyngeal and sinonasal cancers. | Data from different years<br>Population representativity varies<br>For some countries limited national coverage<br>Limitation in technique to detect peak exposures | Shape of ERF<br>Threshold level<br>Partly inconclusive evidence for the endpoint<br>ERF from <3 yr olds; potential effects at older ages not accounted for | Simulation of threshold exceedances<br>Selection of age groups | *** | Underestimation, mainly due to exclusion of ≥ 3year olds but also not accounting for eye irritation |
| Lead | Other cardiovascular diseases than hypertensive disease; kidney damage; miscarriages; other effects of the nervous system; declined fertility; alterations in growth and endocrine function; behavioural disruptions; hearing-threshold changes; hyperkinetic syndrome; lung and stomach cancers.<br>MMR: proxy for all lost IQ points | Differences in study year<br>Differences in studied age group<br>Incomplete data, temporal extrapolation and poorly known exposure trends | Threshold level<br>Shape of ERF | Evidence limited at prevailing low exposure levels<br>Estimation of threshold exceedances | ** | Underestimation due to excluded end-points |
| Transport noise | Annoyance; cognitive impairment, tinnitus | Small proportion of target population is covered<br>Conversion between different noise metrics<br>Different samples<br>Different data estimation years | | Disability weight for sleep disturbance is uncertain<br><br>MI vs IHD | ** | Underestimation due to uncovered populations and exclusion of low exposures, endpoints and noise sources |
| Ozone | Possible long-term effects | Spatial interpolation<br>Impact of urban areas | | YLL not known | ** | Overestimation (YLL set to 12 months)<br>Underestimation due to exclusion of potential long-term effects |
| Particulate matter | Morbidity outcomes evaluated using the CAFE simplifications | Total PM (not just anthropogenic emissions) | Potential threshold level | Unit risk simplifications for morbidity outcomes | * | No substantial error expected or overestimation due to inclusion of natural background |
| Radon | No health endpoints excluded | Possible oversampling of geographical regions known problematic | | | * | No substantial error expected |

[a] Estimated level of overall uncertainty in burden of disease estimates for specific stressor (authors' judgment):
* relatively low level
** medium level
*** relatively high level
This level may deviate from the level of evidence as presented in Figure 4-1, which provides an estimate of the certainty of the underlying knowledge about causality

[b] Authors' judgment about whether results are likely to over- or underestimate the true EBD, given the uncertainties.

## 5.1 Effects of age weighing, discounting and lag

When calculating DALYs, it is optional to discount life years gained in the future. When discounting is applied, those life years gained in the future are valued less than life years gained immediately. Discounting is important in the context of resource allocation. When the return for an investment is delayed, larger returns are needed for making the investment feasible. In the case of human life and health, it has been argued that it may not be ethical to downscale, as the discounting effectively does, the value of future generations. In other contexts, like in debates over nuclear energy, the life of future generations is often given a higher priority than the benefits for the current economy. Moreover, children's health has been set as a priority in the European Environmental Health Action Plan (WHO, 2010c), but both discounting and age-weighing downscale health impacts in children. Thus, the social preferences behind discounting (and age weighing) are not always clear and consistent (Murray & Acharya, 1997).

Discounting leads to lower valuation of impacts that take place later or that last for a long time in comparison with immediate and brief effects. The lag time, which is the time between the exposure to the environmental stressor and the initiation and manifestation of a disease, has not been routinely accounted for in discounting procedures. As part of the EBoDE project, we have tested alternative approaches for analysis of the impacts of lag times on the conclusions of the study. Therefore we have calculated (i) non-discounted non-age weighted estimates (ii) standard WHO discounted (3% per year) age-weighted estimates and (iii) discounted estimates supplemented with crude estimates of the lags from exposure to the manifestation of the disease. Adding the effect of discounted lag times to the estimation further downscales burden of disease estimates for diseases that take longer times to develop. This section compares the three approaches and discusses their implications. Overall, the role of discounting is not driving the ranking of our results: comparing the three alternative metrics (Figure 5-1), the relative order of magnitude between the stressors remains approximately intact. For some stressors like ozone there is a very limited effect of discounting lag times, as the health effects occur immediately after exposure and affect only the current year. Especially for stressors associated with cancer there is a substantial reduction in the burden of disease when discounting the lag time.

The current work demonstrates the potential impact of accounting for lag times by comparing the estimates with and without discounting these lag times. For the six countries addressed in EBoDE, the ranking of the population weighted averages for the nine stressors (Figure 5-1) is in no case seriously affected.

Figure 5-2 presents the effects of discounting, with an without the lag time, on the overall burden of disease estimates. Figure 5-3 shows the absolute effects of discounting by stressor and Figure 5-4 the relative effects.

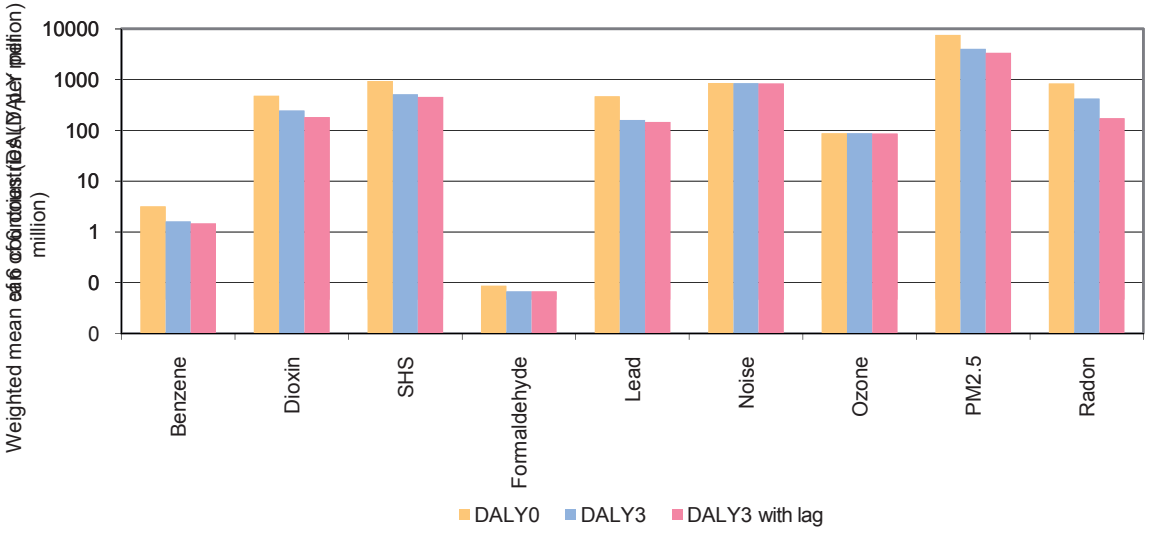



FIGURE 5-1. Comparison of non-discounted (orange), discounted (blue) and discounted with lag (red) results.

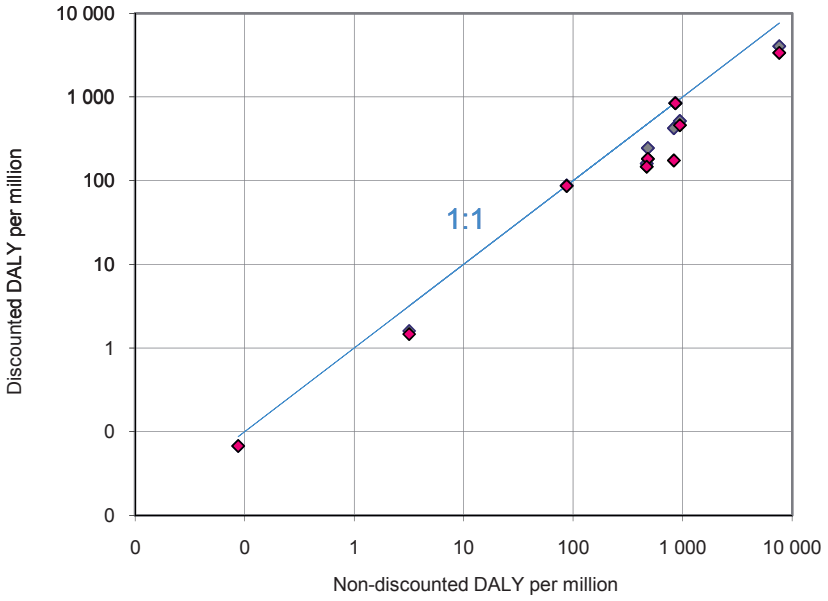



FIGURE 5-2. Effect of discounting on the burden of disease estimates (grey dots: discounting without lags; red dots: discounting with lags).

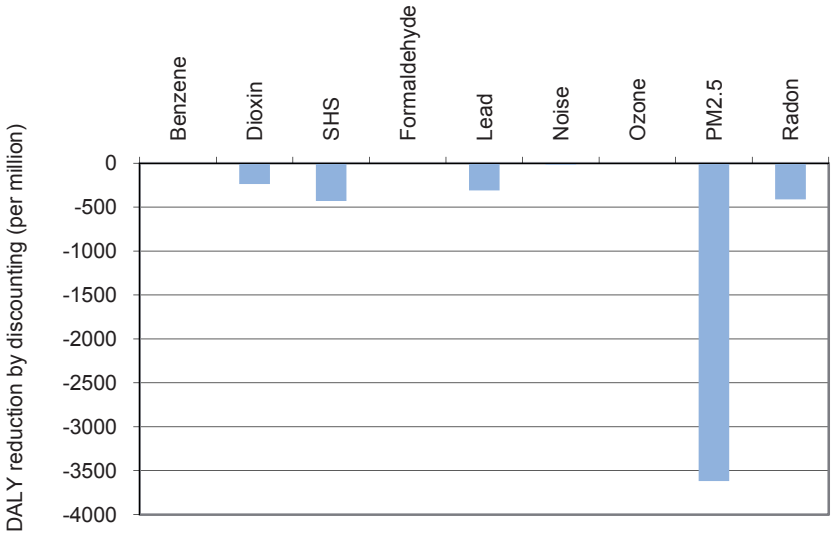



FIGURE 5-3. Absolute effect of discounting in DALYs per million people (no lag).

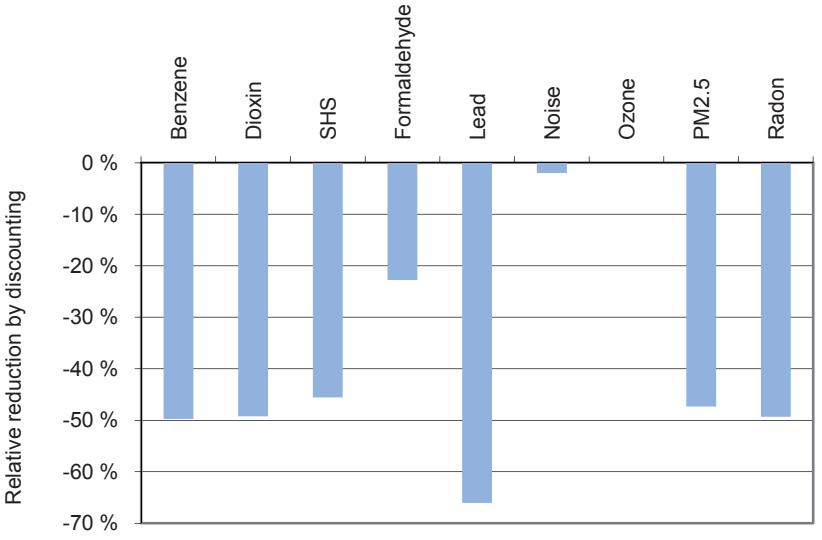



Figure 5-4: Relative effect of discounting in DALYs per million people (no lag).

Thus, while it important to harmonize the discounting procedures, the overall ranking of the results seems to be relatively robust against discounting when ranking the stressors. Discounting the lag times has the highest relative impacts for the cancer outcomes. The highest absolute impact of discounting the lag-times concerns the estimates for particulate matter, because so many cases are involved.

## 5.2 Quantitative estimates of context, model and parameter uncertainty

Additional calculations were performed for selected cases using alternative but realistic choices for some of the input parameters or context and model definitions (Table 5-2). With these cases we want to shed some light on the potential effects of uncertainties on the modelling results. In this paragraph, we will present the results of the calculations based on these alternative choices, and discuss the causes and implications of the observed differences.

TABLE 5-2: Parameters of alternative calculations. Grey boxes indicate the varied parameters in comparison to the baseline (see Table 3-19).

| Stressor | Health endpoint | Population | Exposure estimate | Unit of exposure | Type of ERF | Point estimate of ERF [a] | LCL (95%) | UCL (95%) | Reference(s) for ERF | Thres-hold | Calcu-lation method [b] |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Formal-dehyde | Asthma aggravation | Children (<3 yr) | Mean residential indoor concentration | µg $m^{-3}$ | RR | 1.017 | 1.004 | 1.025 | Rumchev et al., 2002 | 40 | 1A |
| | | | | | | | | | | 60 | 1A |
| | Nasopharyngeal cancer | All | Mean residential indoor concentration | µg $m^{-3}$ | UR | $1.30 \times 10^{-5}$ | N.A. | N.A. | Kerns et al., 1983 | 0 | 2B |
| Lead | Hypertensive disease | Adults | Blood lead level | µg/l | UR | $2.50 \times 10^{-2}$ | $1.7 \times 10^{-2}$ | $3.2 \times 10^{-2}$ | Fewtrell et al. 2003 | 50 | 2B |
| | Ischemic heart disease<br>Cerebrovascular disease<br>Other cardiac diseases | Adults | Blood lead level | µg/l | RR | N.A. (Age- and gender specific calculations conducted using a separate WHO model) | | | Fewtrell et al. 2003 | 50 | 1A |
| Transport noise | Myocardial infarction | All | Persons exposed to exposure categories | $L_{day16h}$ (dB) | OR | function | function | function | Babisch, 2006 | 55 | 1A |
| | Ischemic heart disease | All | Persons exposued to exposure categories | $L_{day16h}$ (dB) | OR | function | function | function | Babisch, 2006 | 55 | 1A |
| $PM_{2.5}$ | Cardiopulmonary diseases | Adults (>30 yr) | Population weighted ambient level | µg $m^{-3}$ | RR | 1.0077 | 1.0020 | 1.0132 | Pope et al., 2002, WHO, 2006a | 2 | 1A |
| | | | | | | | | | | 4 | |
| | Lung cancers | Adults (>30 yr) | Population weighted ambient level | µg $m^{-3}$ | RR | 1.012 | 1.004 | 1.020 | Pope et al., 2002, WHO, 2006a | 2 | 1A |
| | | | | | | | | | | 4 | |
| | Total mortality (non-violent) | Adults (>30 yr) | Population weighted ambient level | µg $m^{-3}$ | RR | 1.0058 | 1.0020 | 1.0096 | Pope et al., 2002, WHO, 2006b | 0 | 1A |
| $PM_{10}$ | LRS symptoms days | School children (5–14 yr) | Population weighted ambient level | µg $m^{-3}$ | UR | 0.186 | 0.092 | 0.277 | Hurley et al., 2005, WHO, 2006b | 0 | 1A |
| | LRS symptom days | >15 yr with chronic LRS | Population weighted ambient level | µg $m^{-3}$ | UR | 0.13 | 0.015 | 0.243 | Hurley et al., 2005, WHO, 2006b | 0 | 1A |
| Radon | Lung cancer (mortality) | All | Residential mean level | Bq $m^{-3}$ | UR | $6.6 \times 10^{-7}$ | *N.A.* | *N.A.* | Darby et al., 2005 | 0 | 2A |

[a] These exposure response functions are all expressed per 1 unit of exposure.
[b] For description of the calculation methods, see paragraph 2.1.

### 5.2.1 Formaldehyde: different health endpoints, thresholds and age groups

A sensitivity analysis was performed for Finland in order to assess the effects of changing the selected threshold level and target population group. For comparison purposes corresponding estimates were calculated for sinonasal cancer, which was excluded as plausible endpoint at the prevailing exposure levels in our baseline calculations. The selected population groups and thresholds are presented in Table 5-3.

Results show that the estimated ranges of impacts for asthma and sinonasal cancer are largely overlapping, so the selection of the endpoint does not significantly affect the order of magnitude of the estimates. For both endpoints, however, the range of estimates spans two orders of magnitude, from below 10 DALYs in Finland (population 5 million) to above 500 DALYs, which equals to over 100 DALYs per million inhabitants. Formaldehyde exposure levels were the highest in Finland in comparison with the other countries in this study. The result highlights that the formaldehyde impact model is highly sensitive to the selection of target population and setting of the threshold level. The E-R function is based on a single study from non-European conditions (Rumchev *et al.*, 2002, Australia), further highlighting the significant uncertainty.

TABLE 5-3. Two health endpoints, four threshold levels and three target age groups compared for formaldehyde.

| Endpoint and model | # | Threshold µg $m^{-3}$ | Population | | Exposed | | EBD |
|---|---|---|---|---|---|---|---|
| | | | Age group | million | % | million | DALY |
| Nasal cancer | 1 | 0 | All | 5.00 | 100 | 5.000 | 450 |
| Unit risk 1.30E-05 cases (µg $m^{-3}$)$^{-1}$ | 2 | 100 | All | 5.00 | 2 | 0.100 | 30 |
| | 3 | 1000 | All | 5.00 | 0 | 0.000 | 0 |
| Asthma | 4 | 40 | <3 yr | 0.17 | 42 | 0.071 | 100 |
| | 5 | 100 | <3 yr | 0.17 | 2 | 0.003 | 6.7 |
| Relative risk 1.017 (µg $m^{-3}$)$^{-1}$ | 6 | 40 | <15 yr | 0.85 | 42 | 0.357 | 560 |
| | 7 | 100 | <15 yr | 0.85 | 2 | 0.017 | 38 |

In conclusion from the uncertainty analysis and accounting for our 'best' estimate, it can be stated that the impacts from formaldehyde are expected to be low, but we cannot completely exclude the possibility of higher impacts.

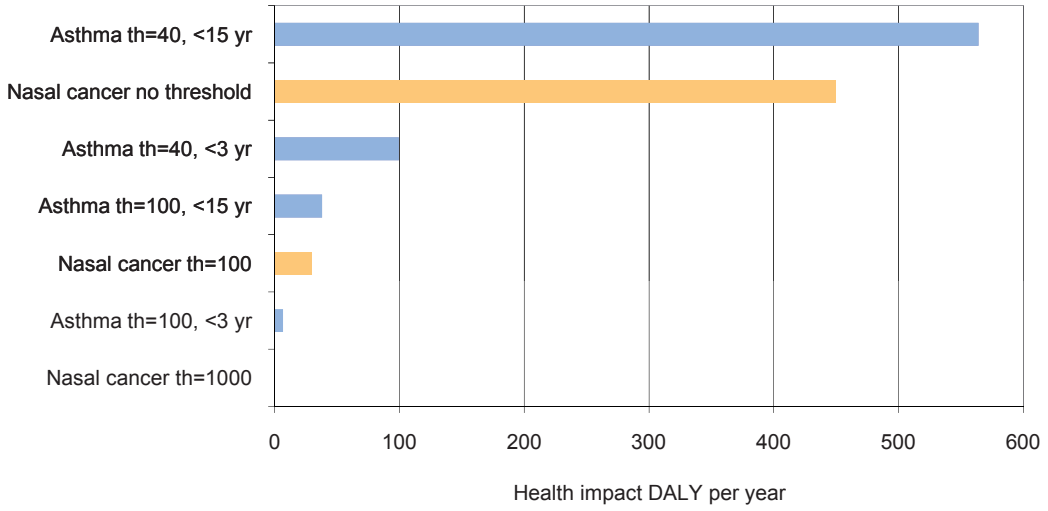



FIGURE 5-5. Comparison of alternative burden of disease models for sinonasal cancer (orange) and asthma (blue) in Finland. Selection of the effect threshold affects the estimates more than the selection of the health endpoint (th = threshold μg $m^{-3}$).

Figure 5-5 shows results of the alternative models for formaldehyde. Estimates for the burden of disease due to cancer, assuming no effect threshold and linear effect, are more than two orders of magnitude higher than the corresponding estimates for asthma aggravation. However, there is no scientific evidence for cancer effects below industrial occupational exposure levels which are 1–2 orders of magnitude higher than the population exposures. Therefore in can be concluded that the risk of underestimating the effects of formaldehyde by looking only at asthma is expected to be small.

The formaldehyde model is thus quite sensitive for the selection of threshold levels for cancer effects. Setting the threshold at the exposure level at which Rumchev *et al.* (2002) observed effects, 60 μg $m^{-3}$, yields ten times higher estimates than using the WHO guideline level. Extrapolating Rumchev *et al.* data below the threshold level would lead to four-fold impacts for children below 15 years.

Scientific evidence on the effects at the low levels is weak, but the impacts cannot be completely excluded. Thus the impacts estimated for the current WHO guideline level of 100 μg $m^{-3}$ have a theoretical possibility of being underestimates.

### 5.2.2 Lead: Comparison to WHO modelling tool

For our baseline results, burden of disease calculations for lead were conducted for mild mental retardation (MMR) and hypertensive disease (HTD) as health endpoints and using modelling approach 2B (see Figure 2-1). WHO has developed a tool for estimating the EBD due to lead based on national exposure levels that takes three additional health endpoints into account and uses gender and age specific population attributable fraction approach (corresponding to EBoDE model 1A, see Figure 2-1) in the calculation (Fewtrell et al. 2003). The health endpoints taken into account in the WHO tool include:

- Mild mental retardation
- Hypertensive disease
- Ischaemic heart disease
- Cerebrovascular disease
- Other cardiac diseases

To estimate the magnitude of the difference in the burden of disease estimates resulting from these two approaches, more complete exposure data from Germany were used to calculate the estimates using the WHO approach and compared with the EBoDE results in Table 5-4.

TABLE 5-4. Comparative calculations of the EBD due to lead in Germany in 2004 with two different models (all values are given in undiscounted DALYs).

| Health endpoint | WHO model | EBoDE model |
|---|---|---|
| Mild mental retardation | 25 757 | 19 005 |
| Hypertensive disease | 239 | 370 |
| Ischaemic heart disease | 1 397 | n.c. |
| Cerebrovascular disease | 773 | n.c. |
| Other cardiac diseases | 701 | n.c. |
| Total EBD | 28 867 | 19 375 |

n.c. these endpoints were not calculated in the EBoDE model

As expected, the WHO model yields a higher overall EBD due to lead in Germany. However, the comparison reveals that the three additional endpoints in the WHO model are not the main reason for the overall difference, as they account for less than 3 000 DALYs in Germany. In contrast, for mild mental retardation the simplified EBoDE method resulted in approximately 7 000 less DALYs than the WHO model. This can be explained by different distribution assumptions for lead in the blood of the population between the two models. Correspondingly, EBD estimates for hypertensive disease exhibit a considerable relative difference due to the different modelling approach (1A vs 2B). This endpoint, however, only contributes marginally to overall EBD due to lead.

The assumptions on the distribution of lead in blood within the population is one of the main elements of uncertainty in EBD calculations. Ideally the population exposure distribution would be based on measured data instead of models and assumptions. Additionally, all five endpoints should be considered in future models in order to reduce underestimation.

### 5.2.3 Noise: myocardial infarction or ischemic heart disease

The original studies by Babisch (2006, 2008) on noise and health used myocardial infarction as indicator of all cardiovascular health effects. However, the current understanding of the mechanistic process leading from noise exposure to myocardial infarction (MI) is consistent for associating noise exposures with also other forms and symptoms of ischemic heart disease as discussed by Babisch (2008). Therefore in the current assessment IHD data were used in conjunction with the relative risks and estimated population attributable fractions.

From the WHO data for EUR A Region it was estimated that 57% of burden of disease from ischemic heart disease is caused by myocardial infarctions. The same ratio for assumed for all participating countries, resulting a similar ratio in the estimates for MI/IHD. In the baseline calculation it was assumed that the relative risk estimated for road traffic noise is applicable for ischemic heart disease, which increases the estimate for cardiovascular diseases to 157% and total noise effects by less than 5%, indicating that the noise impacts are mainly driven by sleep disturbance.

For sleep disturbance, part of the uncertainties in the burden of disease estimates were modelled by calculating the high sleep disturbance (HSD) effects using the best estimate for the disability weight (0.07) as well as using the lower and upper confidence intervals (0.04 and 0.09, respectively. (WHO, 2009a, 2010d)

### 5.2.4 Particulate matter: PM10 or PM2.5; total or cause-specific mortality

The particulate matter models allow for estimation of impacts based on both $PM_{10}$ (for which geographically much larger data coverage is available) and $PM_{2.5}$ (for which the epidemiological evidence suggests higher impacts). Health endpoints for PM can be selected to cover both a variety of morbidity outcomes as well as mortality. Furthermore the latter can be estimated in various ways by looking either at total non-violent mortality, or cause specific mortality for causes for which the causality is established. The table below compares the results of the calculations using alternative model definitions.

TABLE 5-5. Comparison of selection of various health endpoint models for particulate matter ($PM_{2.5}$ and $PM_{10}$).

| Indicators used: | DALYs for particulate matter (undiscounted, un-age-weighted, per million people, aggregated over 6 countries) |
|---|---|
| $PM_{2.5}$ cause-specific mortality and $PM_{2.5}$ cause-specific morbidity (original calculation) | 7 642 |
| $PM_{2.5}$ total mortality and $PM_{2.5}$ cause-specific morbidity (COPD+RAD only) | 10 242 |
| $PM_{2.5}$ cause-specific mortality and $PM_{10}$ cause-specific morbidity | 6 488 |
| $PM_{2.5}$ total mortality and $PM_{10}$ cause-specific morbidity | 9 088 |

It can be seen that the impact for cause specific mortality is somewhat lower than that estimated using total non-violent mortality. It can be concluded that the selection of cause specific calculations for the final results therefore may represent a slight underestimation of the total impact.

In comparison with the previous Clean Air for Europe (CAFE) assessment (Hurley *et al.*, 2005), the current exposure estimates are based on monitoring data and cover both anthropogenic and natural sources of particles. WHO Guidelines for Air Quality (WHO, 2006a) concluded that evidence below 10 $\mu g\ m^{-3}$ levels is very limited due to the fact that at inhabited areas this concentration is mostly exceeded. Linear extrapolation to zero has been used, but the possibility of a threshold has also been suggested. In the CAFE assessment the problem was circumvented by modelling only the anthropogenic fraction of particles, leaving a natural background of 1–3 $\mu g\ m^{-3}$ to alleviate the effects of potential threshold. In the current work the burden of disease calculations were repeated with two alternative threshold levels to asses the magnitude of the impact of a potential threshold for $PM_{2.5}$ particles (Table 5-6).

TABLE 5-6. Comparison of particulate matter ($PM_{2.5}$) impacts using alternative thresholds for cardiopulmonary and lung cancer morbidity and mortality.

| Endpoints | Threshold | DALYs for $PM_{2.5}$ (undiscounted, un-age-weighted, per million people, aggregated over 6 countries) |
|---|---|---|
| $PM_{2.5}$ cause-specific mortality and morbidity (original calculation) | 0 | 7 642 |
| $PM_{2.5}$ cause-specific mortality and morbidity | 2 | 6 936 |
| –"– | 4 | 6 217 |

In Table 5-6 it can be seen that while the inclusion of a threshold decreases the impact estimates, the order of the magnitude remains clearly higher than that estimated for the other stressors.

### 5.2.5 Radon: UR versus RR modelling

For radon we have compared burden of disease estimates calculated using both a relative risk and a unit risk approach. The relative risk approach accounts for the differences in the background disease rates and assumes that the impact of the exposure is of relative nature, i.e. increasing the incidence of lung cancer by the same percentage per unit exposure. The unit risk approach assumes, erroneously one could claim, that the same exposure is always associated with a same number of cases per million inhabitants regardless of the background disease rates.

The comparison shows that the differences between these two models are relatively small, in the order of 15%, higher estimates being created using the RR model (see Table 5-7 and Figure 5-6).

TABLE 5-7. Comparison of UR and RR models for lung cancer cases from radon.

| Exposure-response function used | DALYs for radon (undiscounted, un-age-weighted, per million people, aggregated over 6 countries) |
|---|---|
| RR = 1.0016 (1.0005–1.0031) per 1 Bq $m^{-3}$ (original calculation) | 834 |
| UR = 6.6 × 10-7 per 1 Bq $m^{-3}$ | 722 |

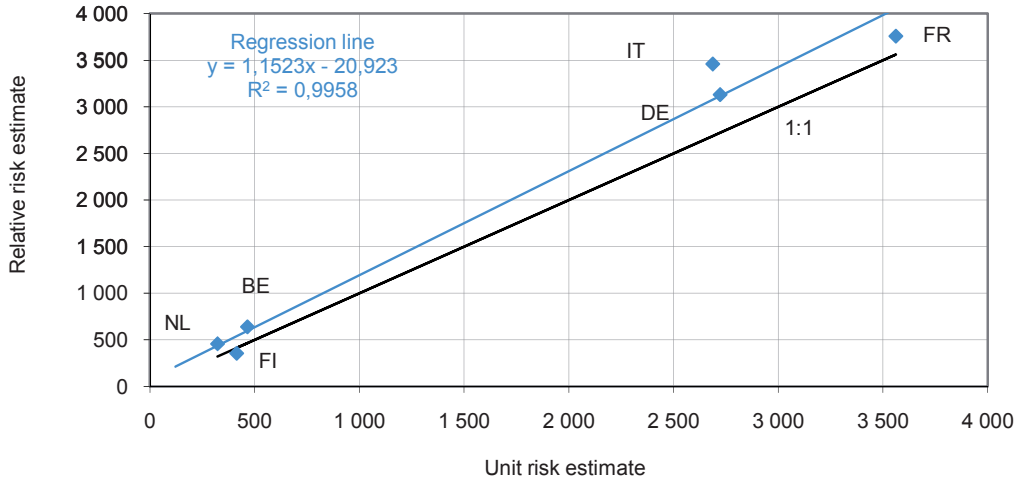



FIGURE 5-6. Graphical comparison of the estimated number of cancer (UR model) and attributable mortality (RR model) cases in the six participating countries.

## 5.3 Discussion of the limitations of EBoDE-approach

The environmental burden of disease is the fraction of disease burden that can be attributed to selected environmental exposures. When estimating the avoidable burden of disease, complications caused by natural background exposures, un-avoidable anthropogenic exposures, and multi-causality have to be taken into account. Moreover, development of efficient policies has to consider the exposure reduction costs against the expected health benefits. Thus environmental burden of disease estimates are an essential input to the policy development process, but need to be accompanied by information about effects, costs, and feasibility of policy measures, information about environmental equity and in some cases, risk perception in the population..

Based on the qualitative and non-conclusive quantitative estimates of uncertainties presented in this chapter, and author judgment, at least the following limitations must be taken into account when interpreting the study results and when developing the methods further:

1. Exposure data for selected risk factors vary in their degree of temporal, population, and geographical representativity. Full comparability between countries would require harmonized data collection procedures to ensure high degree of population representativity and comparability.
2. The current approach is based on scientific evidence on the exposure-response functions; only impacts for which sufficient evidence is available in quantitative format have been included. This means that the total EBoDE estimate do not include burden of disease for which as yet incomplete or only qualitative evidence exists.
3. Estimates of the disease burden could only be performed for risk factors for which sufficient exposure data at population level was available.
4. The population based approach allows for the estimation of the total national impacts, but does not allow for identifying gender differences or health impacts in specific population groups, e.g. highly exposed or those especially vulnerable due to a lower background health status. If sufficient data are available for (potentially) vulnerable groups, this could be included in follow-up research. In addition, a life table approach (see appendix B) would allow modelling of population dynamics, so ageing of the population can be included, because ageing may increase future risks at current exposure levels. Currently, efficient use of lifetable models was not possible, because this would require more information about the temporal relationships between exposures and the health impacts. Further life table modelling is also one of the recommendations for follow-up.
5. Calculations were performed for the year 2004. This was one of the latest years for which exposure and health data were already relatively completely available. Time trends of exposure at the national level were hard to establish.

Overall, it is important to understand that DALYs should not be perceived as a fixed and undisputable number. It is an indicator of the magnitude of various health effects that allows for crude comparison of impacts across a wide range of effect types and environmental stressors. When interpreting DALYs, the methodological complexities and underlying assumptions need to be kept in mind. The representativity of our results for some other regions in Europe, where exposures may be very different, is limited.

# 6 Conclusions and recommendations

Development of efficient environment and health policies and evaluation of their success requires quantitative information about environmental exposures and their health impacts. Disability adjusted life years (DALYs) can be used as an indicator for the environmental burden of disease by expressing both morbidity and mortality effects in one number. World Health Organization Global Burden of Disease and Environmental Burden of Disease programmes have developed methodologies for estimating environmental burden of disease. However, harmonized exposure data and established methods are still lacking for a large number of stressors that have relevance in the developed world. The current study aimed to test the available methods in six European countries using a harmonized approach. Nine stressors were selected that were considered relevant and interesting for Europe. The selection was intended to cover the most important environmental causes of public health impacts, but also to cover less important exposures that have had high significance in public debate or policy development.

The results showed that the EBD methodology can be used to estimate the burden of disease in a harmonized way over a number of stressors and countries. The highest overall public health impact was estimated for ambient fine particles ($PM_{2.5}$; annually 6000-9000 non-discounted DALYs per million in the six participating countries) followed by second-hand smoke (600-1200) transportation noise (500-1100), and radon (600-900). Lower impacts were estimated for dioxins and lead, followed by ozone, all containing also larger relative uncertainties. Lowest impacts were estimated for benzene and formaldehyde.

Quantitative assessment of the various factors affecting the relative ranking of the stressors based on their health impact indicated that the ranking of non-overlapping estimates seems rather robust, even when the exact numbers contain variable amount of uncertainties. The scientific evidence on the causality and quantitative understanding of the exposure-response relationship was considered to have highest reliability for fine particles, second-hand smoke, radon and benzene. Medium uncertainties in the exposures and exposure response-relationships were identified for noise, lead and ozone. Quantitative results for dioxins and formaldehyde were considered most uncertain when evaluating the scientific evidence base.

Differences in the representativity of the exposure data affect the comparability of estimates between the countries. Well comparable exposure data was available for particulate matter and ozone, followed by radon, second hand smoke, benzene, and dioxins. Lowest comparability was found for lead and formaldehyde. Transportation noise exposure data collection is well defined in the European Noise Directive (END), but the comparability of the data available from the first phase of data collection has not reached these standards yet. The comparability of estimates between the stressors is affected also by the selection of the health endpoints and the uncertainty in exposure response functions. It is unlikely that these differences in health response models could be solved in the near future.

Environmental burden of disease estimates support meaningful policy evaluation and resource allocation. Besides, policy analysis also needs to account for the reduction potential of exposures, and other factors such as costs of policy measures and equity issues. The proposed methods for burden of disease estimation should be developed further to cover a larger range of environmental factors and health impacts and to include a systematic evaluation of uncertainties.

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# Appendix A: Additional results by country

# Belgium

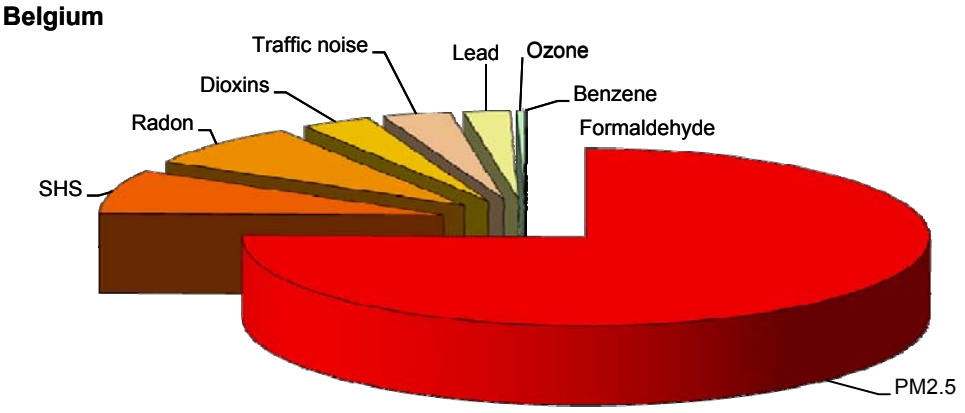


FIGURE A-1. Relative contribution of the nine targeted stressors on the non-discounted environmental burden of disease in Belgium.

TABLE A-1. Results aggregated per stressor in Belgium.

| | Total DALYs | | DALYs per million | |
|---|---|---|---|---|
| | non-discounted | discounted with lag | non-discounted | discounted with lag |
| $PM_{2.5}$ | 106 988 | 47 024 | 10 462 | 4 598.3 |
| SHS | 11 349 | 5 289 | 1 110 | 517.2 |
| Radon | 11 028 | 2 294 | 1 078 | 224.3 |
| Dioxins | 4 628 | 1 745 | 453 | 170.7 |
| Traffic noise | 4 467 | 3 292 | 437 | 321.9 |
| Lead | 3 044 | 927 | 298 | 90.6 |
| Ozone | 530 | 523 | 52 | 51.2 |
| Benzene | 25 | 12 | 2.5 | 1.1 |
| Formaldehyde | 2 | 1 | 0.2 | 0.1 |
| Total | 142 062 | 61 107 | 13 892 | 5 975 |

# Finland

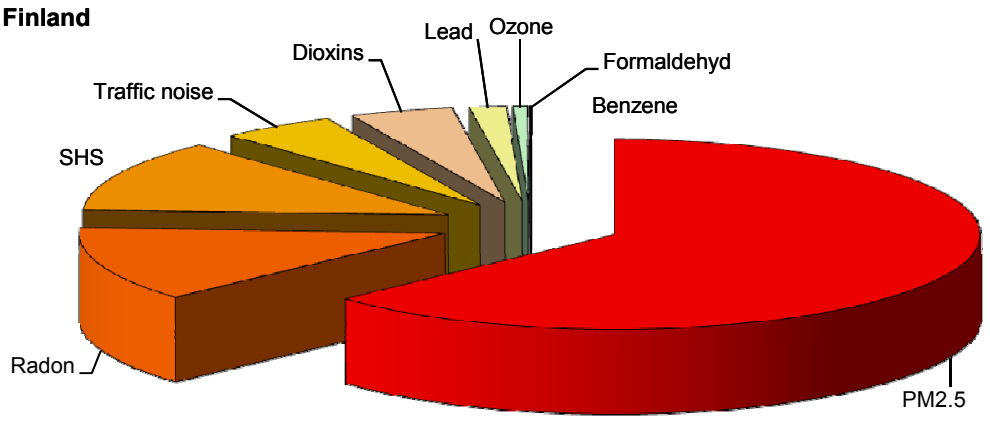


FIGURE A-2. Relative contribution of the nine targeted stressors on the non-discounted environmental burden of disease in Finland.

TABLE A-2. Results aggregated per stressor in Finland.

| | Total DALYs | | DALYs per million | |
|---|---|---|---|---|
| | non-discounted | discounted with lag | non-discounted | discounted with lag |
| $PM_{2.5}$ | 24 062 | 11 146 | 4 602.3 | 2 131.8 |
| Radon | 4 840 | 992 | 925.7 | 189.8 |
| SHS | 4 657 | 2 326 | 890.8 | 444.9 |
| Traffic noise | 1 939 | 1 378 | 370.8 | 263.6 |
| Dioxins | 1 723 | 648 | 329.5 | 124.0 |
| Lead | 618 | 186 | 118.3 | 35.6 |
| Ozone | 245 | 242 | 46.9 | 46.4 |
| Benzene | 16 | 7 | 3.0 | 1.4 |
| Formaldehyde | 9 | 7 | 1.6 | 1.3 |
| Total | 38 108 | 16 933 | 7 289.0 | 3 238.8 |

# France

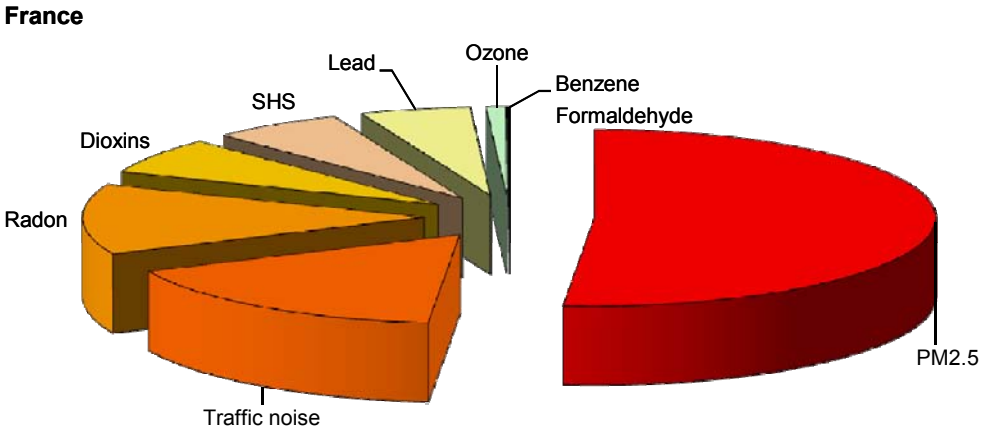


FIGURE A-3. Relative contribution of the nine targeted stressors on the non-discounted environmental burden of disease in France.

TABLE A-3. Results aggregated per stressor in France.

| | Total DALYs | | DALYs per million | |
|---|---|---|---|---|
| | non-discounted | discounted with lag | non-discounted | discounted with lag |
| $PM_{2.5}$ | 277 264 | 121 242 | 4 572.0 | 1 999.3 |
| Traffic noise | 89 958 | 81 432 | 1 483.4 | 1 342.8 |
| Radon | 69 502 | 14 759 | 1 146.1 | 243.4 |
| Dioxins | 35 511 | 13 528 | 585.6 | 223.1 |
| SHS | 33 350 | 17 726 | 549.9 | 292.3 |
| Lead | 27 959 | 8 526 | 461.0 | 140.6 |
| Ozone | 4 940 | 4 889 | 81.5 | 80.6 |
| Benzene | 209 | 96 | 3.4 | 1.6 |
| Formaldehyde | 5 | 4 | 0.1 | 0.1 |
| Total | 538 697 | 262 201 | 8 883.0 | 4 323.7 |

# Germany

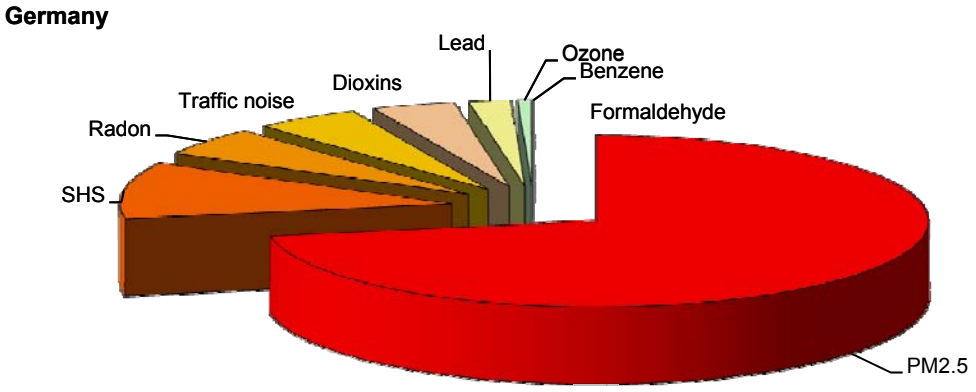


FIGURE A-4. Relative contribution of the nine targeted stressors on the non-discounted environmental burden of disease in Germany.

TABLE A-4. Results aggregated per stressor in Germany.

| | Total DALYs | | DALYs per million | |
|---|---|---|---|---|
| | non-discounted | discounted with lag | non-discounted | discounted with lag |
| $PM_{2.5}$ | 691 732 | 307 617 | 8 384.5 | 3 728.6 |
| SHS | 101 909 | 47 684 | 1 235.2 | 578.0 |
| Radon | 51 154 | 10 636 | 620.0 | 128.9 |
| Traffic noise | 48 770 | 38 110 | 591.1 | 461.9 |
| Dioxins | 38 422 | 14 482 | 465.7 | 175.5 |
| Lead | 19 377 | 5 954 | 234.9 | 72.2 |
| Ozone | 6 062 | 5 985 | 73.5 | 72.5 |
| Benzene | 220 | 101 | 2.7 | 1.2 |
| Formaldehyde | 5 | 4 | 0.1 | 0.0 |
| Total | 957 650 | 430 574 | 11 607.7 | 5 219.0 |

# Italy

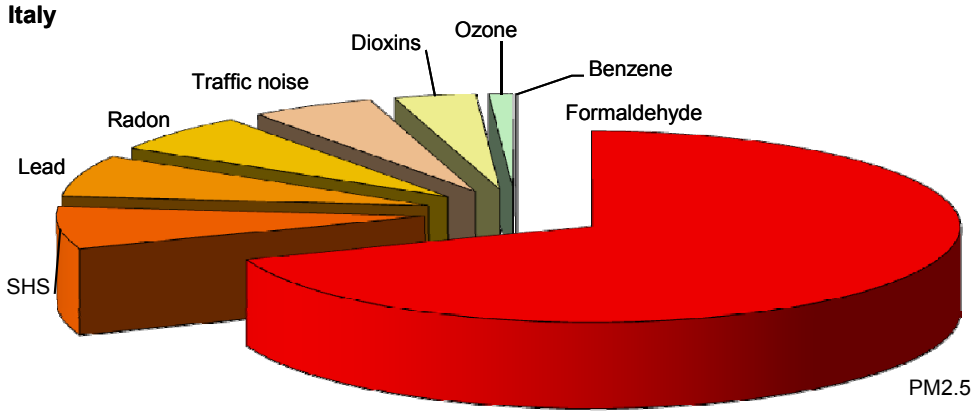


FIGURE A-5. Relative contribution of the nine targeted stressors on the non-discounted environmental burden of disease in Italy.

TABLE A-5. Results aggregated per stressor in Italy.

| | Total DALYs | | DALYs per million | |
|---|---|---|---|---|
| | non-discounted | discounted with lag | non-discounted | discounted with lag |
| $PM_{2.5}$ | 545 543 | 237 619 | 9 377.6 | 4 084.5 |
| SHS | 56 746 | 27 139 | 975.4 | 466.5 |
| Lead | 55 018 | 17 530 | 945.7 | 301.3 |
| Radon | 50 378 | 10 357 | 866.0 | 178.0 |
| Traffic noise | 42 728 | 38 791 | 734.5 | 666.8 |
| Dioxins | 28 113 | 10 531 | 483.2 | 181.0 |
| Ozone | 8 038 | 7 936 | 138.2 | 136.4 |
| Benzene | 245 | 113 | 4.2 | 1.9 |
| Formaldehyde | 0 | 0 | 0.0 | 0.0 |
| Total | 786 808 | 350 015 | 13 524.8 | 6 016.6 |

# The Netherlands

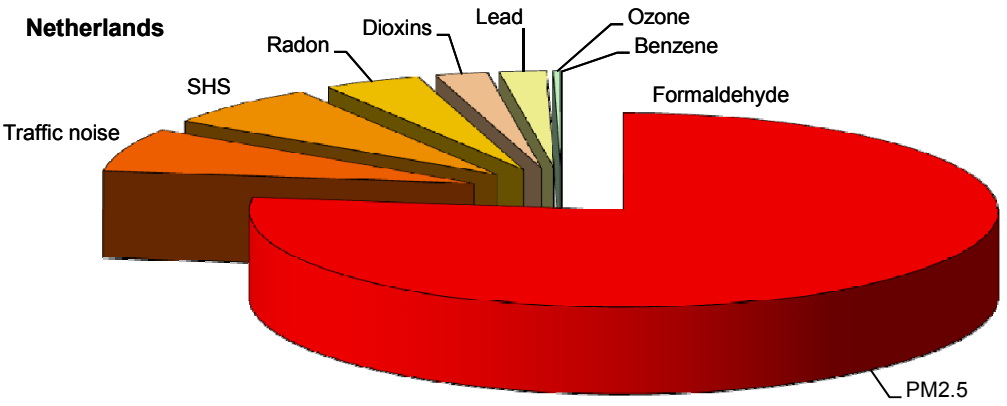


FIGURE A-6. Relative contribution of the nine targeted stressors on the non-discounted environmental burden of disease in the Netherlands.

TABLE A-6. Results aggregated per stressor in the Netherlands.

| | Total DALYs | | DALYs per million | |
|---|---|---|---|---|
| | non-discounted | discounted with lag | non-discounted | discounted with lag |
| $PM_{2.5}$ | 135 500 | 59 020 | 8 322.2 | 3 624.9 |
| Traffic noise | 12 615 | 10 803 | 774.8 | 663.5 |
| SHS | 12 201 | 6 725 | 749.4 | 413.0 |
| Radon | 7 374 | 1 526 | 452.9 | 93.7 |
| Dioxins | 3 936 | 1 494 | 241.7 | 91.8 |
| Lead | 3 535 | 1 067 | 217.1 | 65.6 |
| Ozone | 547 | 541 | 33.6 | 33.2 |
| Benzene | 26 | 12 | 1.6 | 0.7 |
| Formaldehyde | 0 | 0 | 0.0 | 0.0 |
| Total | 175 734 | 81 189 | 10 793.3 | 4 986.5 |

# Appendix B: Comparison with life-table model

## Impact Calculation Tool (ICT) – Probabilistic health impact analysis with life-tables

The reported EBoDE results were calculated based on point estimates and population group averages for relevant input parameters. In the case of most stressors, the analysis was based on WHO total burden of disease estimates for 2004. This is a relatively simple and handy modelling approach for determining and ranking health impacts of different environmental stressors at a given time period. However, more advanced life table modelling is required when trying to predict how age and gender specific health impacts will change in time as population structure, background mortality risks and exposures change. Given the time frame of the EBoDE project, a detailed life table modelling approach was not feasible. However, a parallel development project of a probabilistic life table model called Impact Calculation Tool (ICT) allowed for a limited comparison of the two modelling approaches and a view on the advantages of probabilistic life table modelling.

Impact Calculation Tool (ICT) is a user-friendly modelling tool for quantification of health impacts from environmental exposures. It applies dynamic life table modelling for calculation of population specific loss of disability adjusted life years (DALY) from an exposure of interest. The model has been developed in the context of the EU project INTARESE and the Finnish Academy project CLAIH, and is a collaboration between the National Institute of Health and Welfare (THL, Finland), the National Institute for Public Health and the Environment (RIVM, the Netherlands), and Netherlands Environmental Assessment Agency (PBL, the Netherlands).

ICT allows analysis of health impacts from one environmental exposure in one given population at a time. The follow-up period (time period for which impacts are determined), specific mortality and morbidity impacts analysed, and the target population can be defined according to the needs of the assessment at hand. In one model run, impacts are modelled for a reference, business-as-usual (BAU), and one alternative exposure scenario. Impact indicator outputs include age-specific mortality/disease cases, life expectancy (age-specific and birth cohort) and age-specific loss of disability adjusted life years (DALY, YLL (years of life lost due to mortality), and YLD (years of life lost due to disease)). Both time discounting and age-weighting can be applied. All key input data has to be provided by the user. The input data requirements include:

- age-specific population data
- age-specific baseline mortality incidence and morbidity incidence/prevalence data
- birth rate
- exposure levels (reference, BAU, alternative scenario)
- exposure-response functions for the health endpoints of interest (relative risk or absolute risk)
- severity weights and durations for the morbidity endpoints.

ICT runs in Analytica, which is a modelling software with a user-friendly graphical interface. The software enables probabilistic modelling using Monte Carlo simulation and, therefore, advanced uncertainty analysis. Full use of the Analytica programme requires a software licence. However, ICT can also be run with a free Analytica player, which can be downloaded from www.lumina.com. The player allows the user to view to the model contents and calculation specifics, to input data for key parameters, and to calculate results and run probabilistic uncertainty analysis. ICT contains a simple user interface, which enables these functions without advanced knowledge of Analytica or the model technicalities.

## Health impact modelling in ICT

Mortality impacts are modelled using dynamic life table approach. First, total mortality risk is modelled for the reference and alternative exposure scenarios using the population and baseline mortality data, exposure levels and exposure-response functions. Based on the total mortality risk and population input data, the future population structure is then projected for each scenario using life table methodology. These population projections are applied in determining the age-conditional life expectancies in the different scenarios. YLL (years of life lost due to mortality) can subsequently be calculated directly from the life tables as the difference in the life years lived by the projected populations in different scenarios, or indirectly based on the age-specific attributable deaths and age-specific life expectancies in a given scenario.

In the case of morbidity impacts, the morbidity risk attributable to the exposure is first modelled for each scenario based the population and baseline morbidity data, exposure level and exposure-response functions. Number of attributable morbidity cases is then calculated using the attributable morbidity risk and the modelled population projections for each scenario, and YLD (years of life lost due to disease) subsequently based on the attributable cases, severity weight and duration.

## Benefits of life table modelling

The advantage of dynamic life table modelling in health impact assessment is that it enables to predict impacts in a real life population over time as the population structure and risk level changes. Full use of a life table model takes into account that changing the risk of a certain cause of death at a given point in time will affect the population available to die from any cause of death at later time points. Thus, it gives the net change in the life years saved or lost over time and prevents over-estimation of the overall mortality impact when evaluating effects from multiple mortality endpoints for a single exposure or combined mortality effects from multiple exposures.

Life table modelling provides most benefits in terms of estimating the net total impacts in a real life population when used in a direct way, i.e. comparing the life tables and life years predicted for different scenarios. However, the indirect use, i.e. when life table modelling is used to determine age-conditional life expectancy in a real life population, which can be further multiplied with attributable deaths to derive YLL, can be more preferable in some situations. This is, for example, in cases where impacts are modelled for a short follow-up period (one or few years), but the aim is to estimate total loss of life years due to the attributable deaths. A simplified solution would be to use age-conditional life expectancy data for the current population. However, if the aim is to model impacts due to an existing risk factor, this approximation would lead to underestimation of YLL because it ignores that in the (theoretical) absence of the risk life expectancy would, in fact, be a fraction higher. In many cases this difference would be negligible, but could in some cases be of importance. This source of bias is avoided when applying life table modelling in the impact assessment, because the model also predicts the impact of the risk factor to the current life-expectancy in the target population.

## ICT vs. EBoDE modelling approach

To compare results from ICT modelling with the results from the more simple modelling approach applied in EBoDE, and to demonstrate possibilities for further impact analysis with life table modelling, health impacts from fine particle (PM2.5) exposure in Finland were calculated with ICT. Impacts were modelled for a 40 year follow-up period starting from 2004. The indirect impact calculation approach (YLL is calculated from age-specific attributable deaths and age conditional life expectancies) was used in order to express the annual loss of life years in a way that is comparable with the EBoDE results. Modelling was based on the same exposure level, exposure-response functions, time discount factor and age weights as in the EBoDE model. Population and baseline mortality incidence data used was for year 2004.

Health impacts modelled using the EBoDE approach and ICT are compared in Table B-1. As expected, the differences are small. In principle, the EBoDE modelling approach should yield somewhat higher mortality impact values, because the model is based on the WHO total burden of disease data for 2004. In WHO burden of disease method, YLL is calculated based on age-conditional standard life expectancies,

which are a little bit higher than age-conditional life expectancies in the real Finnish population used in ICT. However, all the EBoDE impact estimates are lower, most likely dueto discrepancy in the mortality incidence data used in the ICT modelling and WHO 2004 burden of disease calculations or difference in the specifics of how age-specific death counts and life expectancies are combined to yield YLL in ICT and WHO burden of disease.

TABLE B-1. Burden of disease from different mortality and morbidity impacts due to $PM_{2.5}$ exposure in Finland in 2004.

| Health end-point | Non-discounted, non-ageweighed DALYs | | | Discounted and age-weighted DALYs | | |
|---|---|---|---|---|---|---|
| | EBoDE | ICT | Difference | EBoDE | ICT | Difference |
| Cardiopulmonary mortality | 13 599 | 14 572 | 973 | 6 401 | 6 646 | 245 |
| Lung cancer mortality | 2 856 | 2 988 | 132 | 1 407 | 1 434 | 27 |
| Total mortality | 25 321 | 27 088 | 1 767 | 12 151 | 12 597 | 446 |
| | | | | | | |
| Restricted activity days (RAD) | 776 | 777 | 0 | 776[a] | 908 | 131 |
| Lower respiratory symptom days (LRS), children | 425 | 425 | 0 | 425[a] | 414 | -12 |
| Lower respiratory symptom days (LRS), adults | 607 | 606 | 0 | 607[a] | 641 | 34 |

[a] In EBoDE calculations age-weighing was not used for $PM_{2.5}$ morbidity outcomes due to causes related to model implementation. Difference to ICT results indicate the magnitude of these impacts.

ICT allows probabilistic impact modelling using Monte Carlo simulation, which enables advanced uncertainty analysis. Instead of determining only the upper and lower estimates to give the possible range of impacts, with probabilistic modelling one can get information on the likelihood of the estimates within that range. As an example, Figure B-1 shows a cumulative probability curve for the years of life lost from cardiopulmonary and lung cancer mortality impacts. Probabilistic analysis requires one or more key model inputs to be defined as probability distributions. In the example, the exposure-response functions were defined as following distributions: cardiopulmonary mortality: triangular(min 1.02, mode 1.08, max 1.14), lung cancer mortality: triangular(min 1.04, mode 1.13, max 1.22)

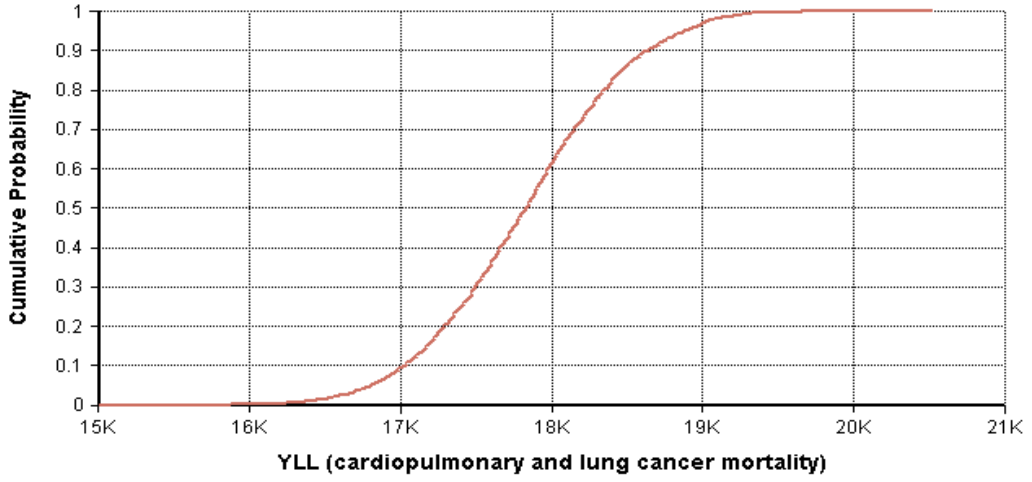


FIGURE B-1. Cumulative probability curve for total years of life lost from cardiopulmonary and lung cancer mortality due to $PM_{2.5}$ exposure in Finland in 2004 (no time discounting, no age weighting).

ICT automatically provides age-specific impact estimates. Figures B-2 and B-3 show how the mortality impacts from PM2.5 exposure are distributed among age groups.

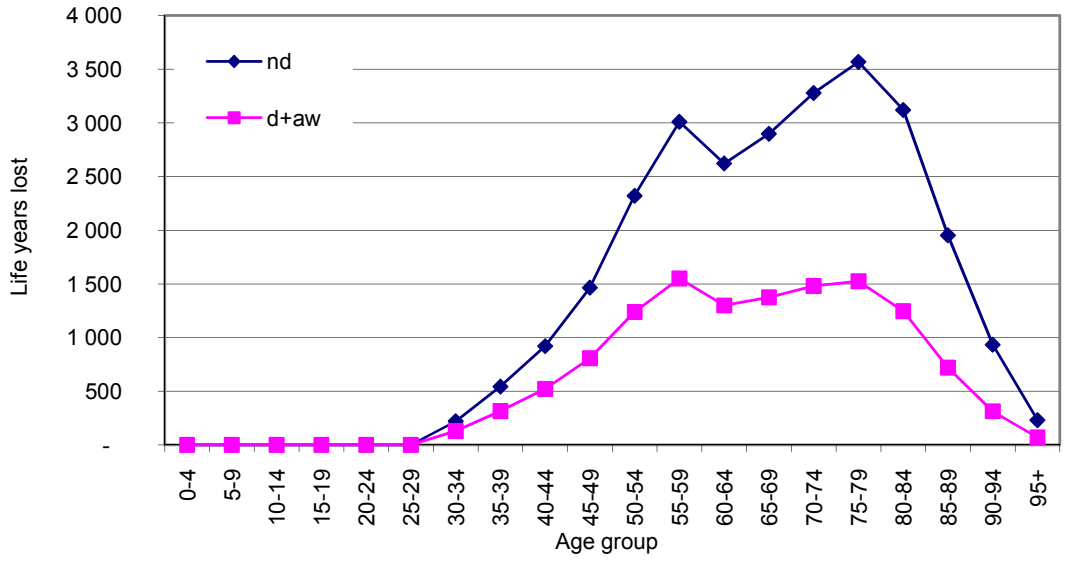


FIGURE B-2. Years of life lost in different age groups from total mortality (non-accidental) due to $PM_{2.5}$ exposure in Finland in 2004. nd = no discounting or age weighting, d+aw = time discounting and age weighting applied.

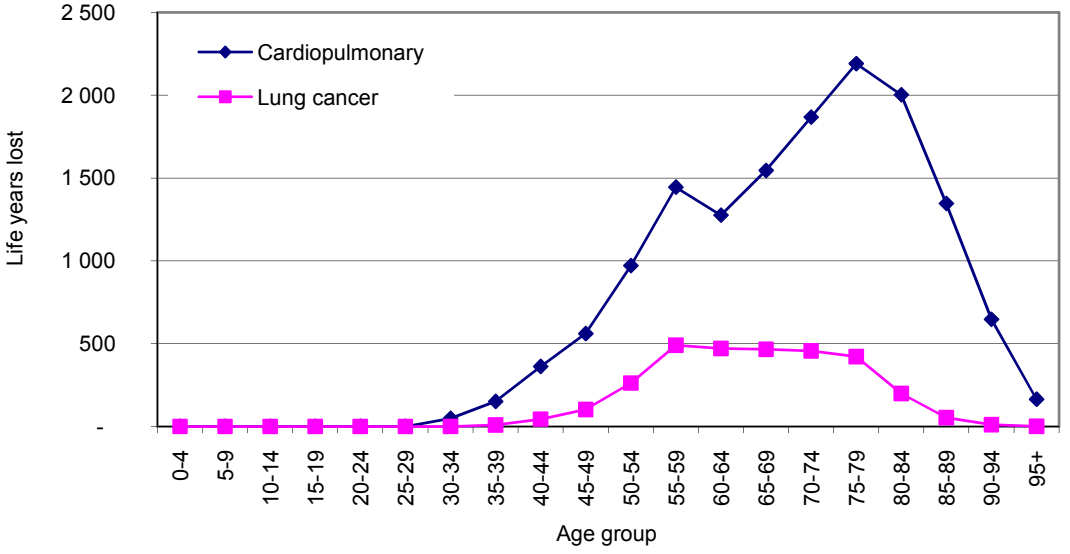


FIGURE B-3. Years of life lost (no time discounting or age weighting) in different age groups from cardiopulmonary and lung cancer mortality due to $PM_{2.5}$ exposure in Finland in 2004.

The main benefit of life table modelling is that it allows predicting how impacts in a real life population will change in the future as the population structure changes. As an example, Table B-2 and Figures B-4 and B-5 show how health impacts from $PM_{2.5}$ would change in time in the Finnish population, should the exposure stay on the 2004 level. Population projections require assumptions on the future birth rate. In this case birth rate for the years 2004-2009 was based on the Finnish birth statistics and for the years 2010–2043 the rate was assumed to stay on the 2009 level. In addition to population age structure, future impacts

depend on how the exposure level and background mortality risk change in time. In ICT, it would also be possible to model impacts for a situation where the exposure level changes through the follow-up time.

TABLE B-2. Burden of disease (no time discounting or age weighting) due to total mortality (non-accidental) due to $PM_{2.5}$ exposure in Finland when exposure is assumed to stay on the 2004 level.

| Age group | Years | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | 2004–2008 | 2009–2013 | 2014–2018 | 2019–2023 | 2024–2028 | 2029–2033 | 2034–2038 | 2039–2043 |
| 0-29 | - | - | - | - | - | - | - | - |
| 30-34 | 1 143 | 1 182 | 1 160 | 1 155 | 1 126 | 1 039 | 1 023 | 1 055 |
| 35-39 | 2 507 | 2 372 | 2 455 | 2 408 | 2 398 | 2 337 | 2 158 | 2 124 |
| 40-44 | 4 480 | 4 018 | 3 802 | 3 935 | 3 860 | 3 844 | 3 746 | 3 459 |
| 45-49 | 7 231 | 6 946 | 6 230 | 5 895 | 6 101 | 5 985 | 5 960 | 5 808 |
| 50-54 | 11 265 | 10 781 | 10 355 | 9 287 | 8 789 | 9 096 | 8 922 | 8 885 |
| 55-59 | 14 832 | 14 182 | 13 573 | 13 037 | 11 693 | 11 065 | 11 452 | 11 233 |
| 60-64 | 15 866 | 18 348 | 17 545 | 16 791 | 16 128 | 14 465 | 13 689 | 14 167 |
| 65-69 | 14 966 | 18 680 | 21 603 | 20 656 | 19 769 | 18 988 | 17 031 | 16 117 |
| 70-74 | 16 876 | 17 921 | 22 368 | 25 867 | 24 734 | 23 672 | 22 737 | 20 393 |
| 75-79 | 18 035 | 18 762 | 19 924 | 24 868 | 28 758 | 27 498 | 26 317 | 25 278 |
| 80-84 | 16 873 | 18 338 | 19 076 | 20 258 | 25 285 | 29 240 | 27 959 | 26 758 |
| 85-89 | 11 281 | 13 838 | 15 039 | 15 645 | 16 614 | 20 737 | 23 980 | 22 930 |
| 90-94 | 5 052 | 6 281 | 7 705 | 8 374 | 8 711 | 9 250 | 11 546 | 13 352 |
| 95+ | 1 327 | 1 628 | 2 024 | 2 482 | 2 698 | 2 806 | 2 980 | 3 720 |
| Total | 141 733 | 153 277 | 162 857 | 170 659 | 176 663 | 180 023 | 179 500 | 175 279 |

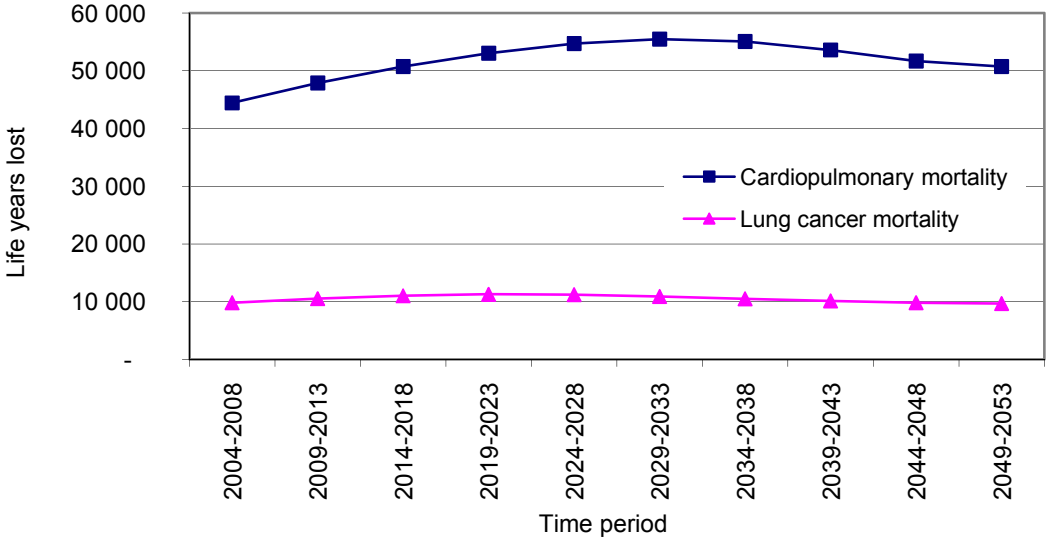


FIGURE B-4. Years of life lost (no time discounting or age weighting) from cardiopulmonary and lung cancer mortality due to $PM_{2.5}$ exposure in Finland from 2004 to 2043.

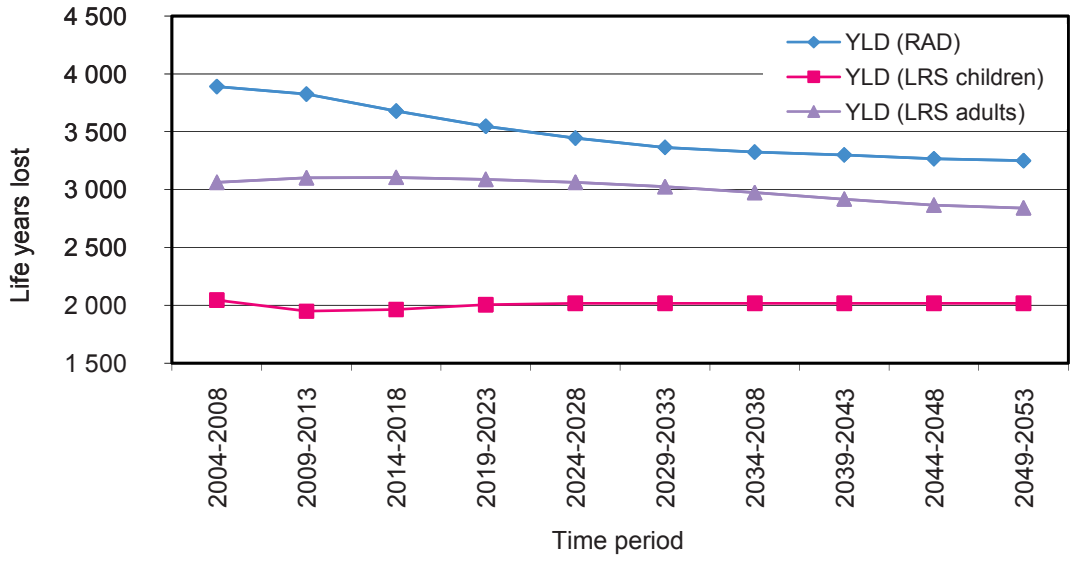


FIGURE B-5. Years of life lost (no time discounting or age weighting) from morbidity due to $PM_{2.5}$ exposure in Finland from 2004 to 2043. RAD = restricted activity days (age group 15–64), LRS = lower respiratory symptom days (children aged 5–14, adults aged >15).