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Trends in the environmental burden of disease in the Netherlands 1980 – 2020

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Abstract

Trends in the environmental burden of disease in the Netherlands 1980 – 2020

Several aspects of the environment, such as exposure to air pollution or noise, can have effects on our health. In order to gain some perspective on the dimensions of this environment-related health loss in the Netherlands, we have calculated Disability Adjusted Life Years (DALYs) for the health effects of air pollution, noise, radon, natural UV-radiation and indoor dampness for the years 1980, 2000 and 2020. DALYs give a crude indication of the estimated number of healthy-life-years-lost in a population due to premature mortality or morbidity (the disease burden).

In the Netherlands, roughly 2 to 5 percent of the disease burden (as calculated for 49 (groups of) diseases) can be attributed to the effects of (short-term) exposure to air pollution, noise, radon, total natural UV and dampness in houses for the year 2000. Including the more uncertain long-term effects of PM_{10} exposure, this can increase to slightly over ten percent, assuming no threshold. Assuming a reference level of 20 $\mu g/m^3$ will give an estimate of roughly 3 to 9 percent.

Among the investigated factors, the relatively uncertain effects of long-term PM_{10} exposure have the greatest impact. Long-term PM_{10} is an indicator for a complex urban air pollution mixture. The levels of PM_{10} are decreasing; therefore the related disease burden is also expected to decrease. Noise exposure and its associated disease burden will probably increase up to a level where the disease burden is similar to that attributable to traffic accidents.

These rough estimates do not provide a complete and unambiguous picture of the environmental disease burden; data are uncertain, not all environmental-health relationships are known, not all environmental factors are included, nor was it possible to assess all potential health effects. The effects of a number of these assumptions were evaluated in uncertainty analyses.

Keywords: DALYs, disease burden, environment

Rapport in het kort

Trends in de milieugerelateerde ziektelast in Nederland, 1980 – 2020

In het jaar 2000 werd naar schatting 2 tot 5 procent van de ziektelast in Nederland veroorzaakt door acute blootstelling aan fijn stof (PM_{10}) en ozon, en blootstelling aan geluid, radon, (totaal) UV-straling en vocht in huizen. Dit kan oplopen tot iets boven de 10 procent als ook de meer onzekere langetermijneffecten van blootstelling aan fijn stof worden meegewogen (en geen drempelwaarde voor effecten wordt aangenomen). Wanneer een drempelwaarde van 20 van $\mu g/m^3$ wordt verondersteld leidt dit tot een totaal van ruwweg 3 tot 9 procent van de ziektelast. De relatief onzekere effecten van langdurige blootstelling aan fijn stof hebben de grootste invloed op het totaal milieu-gerelateerd gezondheidsverlies in Nederland. Fijn stof kan hierbij gezien worden als een indicator voor een complex mengsel van luchtverontreiniging. De ziektelast voor fijn stof loopt naar schatting terug over de periode 1980-2020. Voor geluid wordt een toename verwacht.

Dit blijkt uit een analyse van Nederlandse en buitenlandse gegevens over de relatie tussen blootstelling aan enkele milieufactoren en effecten op gezondheid. In deze analyse is de milieu-gerelateerde ziektelast uitgedrukt in de zogenaamde DALY: Disability-Adjusted-Life-Years. Dit is een maat waarin zowel *het aantal mensen* bij wie gezondheidseffecten optreden, als de *ernst* en de *duur* van deze effecten, verwerkt is. Deze DALY's zijn berekend voor de gezondheidseffecten van luchtverontreiniging, geluid, radon, UV straling en vocht in huizen voor de periode 1980 - 2020. Dit geeft een ruwe indicatie van het aantal verloren gezonde levensjaren in de Nederlandse populatie door milieu-gerelateerde ziekte of vroegtijdige sterfte (de ziektelast).

Bij het berekenen van de milieu-gerelateerde ziektelast zijn een groot aantal aannames gedaan. De effecten van die aannames zijn geanalyseerd met behulp van onzekerheidsanalyses.

Trefwoorden: DALYs, ziektelast, milieu

Contents

Samenvatting

Milieufactoren kunnen op verschillende manieren van invloed zijn op de gezondheid en de kwaliteit van leven van de Nederlandse bevolking. Zo is luchtverontreiniging bijvoorbeeld gerelateerd aan luchtwegklachten of hartvaatziekten, geluidsoverlast kan leiden tot hinder, en blootstelling aan bepaalde straling kan in sommige gevallen kanker veroorzaken. Het is lastig om deze problemen te vergelijken, omdat ze allen van verschillende aard en omvang zijn. Daarom is het nuttig om de milieugerelateerde gezondheidseffecten onder één noemer te brengen met behulp van een geïntegreerde maat.

In deze studie hebben we de effecten op de volksgezondheid in Nederland van luchtvervuiling (fijn stof en ozon), geluid, straling (radon en UV straling) en vocht in woningen samengevat met behulp van DALYs (Disability Adjusted Life Years). In een DALY berekening wordt *het aantal mensen* dat een bepaalde aandoening heeft ten gevolge van blootstelling aan een milieufactor vermenigvuldigd met *de tijd* dat ze de aandoening hebben (of de levensduurverkorting in geval van sterfte) en *de ernst* van de aandoening (variërend van 0 voor perfecte gezondheid tot 1 voor sterfte). Op deze manier kunnen zowel ziekte als sterfte in één getal worden uitgedrukt, waardoor milieugezondheidsproblemen kunnen worden vergeleken en beleid op dat terrein kan worden gepland of geëvalueerd. DALYs zijn een vereenvoudiging van een zeer complexe werkelijkheid, waardoor de maat slechts een zeer ruwe indicatie geeft van (milieu-gerelateerd) gezondheidsverlies.

Voor elke onderzochte milieufactor is bepaald welke gezondheidseffecten relevant zijn en welke gegevens het best gebruikt konden worden voor de DALY berekeningen. Het gaat daarbij bijvoorbeeld om gegevens over het vóórkomen van ziektes, de relatie tussen milieufactoren en gezondheidseffecten en weegfactoren die een indicatie geven van de ernst van een aandoening. Wanneer informatie ontbrak of bepaalde bronnen verschillende informatie weergaven, is met behulp van inhoudsdeskundigen bepaald welke (alternatieve) gegevens gebruikt zouden moeten worden. Er is een onzekerheidsanalyse uitgevoerd waarin het effect van de verschillende aannamen op de uitkomsten is geanalyseerd. Daarnaast zijn de uitkomsten ook kwalitatief beoordeeld op hun bruikbaarheid.

Van de milieufactoren die in deze studie zijn onderzocht, levert de langetermijnblootstelling aan fijn stof op dit moment de grootste bijdrage aan de ziektelast in Nederland. Fijn stof kan worden gezien als een indicator voor een complexer geheel aan luchtvervuilende stoffen. Het relatief grote aantal DALYs dat wordt toegeschreven aan blootstelling aan fijn stof wordt echter voornamelijk bepaald door de langetermijneffecten, waar nog veel onduidelijkheid over bestaat. Blootstelling aan geluid zorgt ook voor een aanzienlijk aantal DALYs in Nederland, vooral omdat er zoveel mensen blootgesteld zijn aan geluid. De effecten van radon en UV zijn iets minder omvangrijk, terwijl blootstelling aan ozon en vocht in huizen de geringste hoeveelheden DALYs opleveren in deze studie.

In dit onderzoek is gekeken naar tijdtrends in ziektelast, door zowel berekeningen voor het verleden (1980 of 1990) als voor de toekomst (2010 of 2020) uit te voeren. De effecten van UV en radon blijven ongeveer gelijk. De ziektelast ten gevolge van PM_{10} blootstelling neemt af, terwijl het geluidsgerelateerde gezondheidsverlies in de toekomst waarschijnlijk juist zal toenemen.

Bij benadering wordt geschat dat zo'n 2 tot 5 procent van de ziektelast in Nederland (die is berekend voor 49 (groepen van) ziekten en aandoeningen) toe te schrijven is aan de effecten van luchtverontreiniging (fijn stof en ozon, kortetermijneffecten), geluid, radon, UV en vocht in huizen. Inclusief de relatief onzekere effecten van langetermijnblootstelling aan fijn stof kan het zelfs gaan om iets meer dan 10 procent, indien geen drempelwaarde wordt verondersteld. Dit lijkt echter een minder realistisch scenario. Wanneer een drempelwaarde van 20 van $\mu g/m^3$ wordt verondersteld leidt dit tot een totaal percentage van ruwweg 3 tot 9 procent van de ziektelast.

Summary

Environmental factors can affect health and quality of life of the Dutch population in various ways. Air pollution is associated with respiratory or cardiovascular diseases, noise exposure can lead to annoyance, and exposure to certain forms of radiation can cause the development of cancer. It is difficult to compare these problems, since they differ in type and scope. Therefore it can be useful to quantify the health impact of the environment in an integrated measure.

We have quantified the public health impacts of air pollution (PM_{10} and ozone), noise, radiation (radon and UV) and indoor dampness using DALYs (Disability Adjusted Life Years). In DALY calculations, *the number of people* with a certain disease is multiplied by *the duration* of the disease (or loss of life expectancy in case of mortality) and *the severity* of the disorder (varying from 0 for perfect health to 1 for death). In this way, morbidity as well as mortality can be expressed in one similar value, making environmental health problems more or less comparable and providing ways to plan or evaluate environmental health related policies. However, DALYs are a simplification of a very complex reality, and therefore only give a very crude indication of (environmental) health impact.

In our study, we have assessed which effects are relevant to investigate for each environmental factor, and which data are best to use. Necessary data include for example prevalence numbers, exposure-response relationships, and weighting factors that give an indication of the severity of a certain disorder. When information was missing or ambiguous, we have consulted experts in order to decide which (alternative) data sources to use. An uncertainty analysis was carried out in order to analyze the effects of different assumptions. Results have also been qualitatively evaluated for their usefulness within different contexts.

The effects of PM_{10} make the greatest contribution to the environment-related disease burden in the Netherlands in our study. PM_{10} can be regarded as an indicator for a complex mixture of urban air pollutants. However, the relatively large amount of DALYs potentially attributable to PM_{10} is mostly caused by *long-term* effects of PM_{10} exposure, which are very uncertain. Exposure to noise also leads to a significant number of DALYs in the Netherlands, mainly because there are so many people exposed to noise. The health effects of radon and

UV radiation are also significant -although slightly less substantial-, whereas ozone and indoor dampness make the smallest contribution to the environment-related burden of disease in the Netherlands as calculated in this study.

We have studied time trends in the environmental burden of disease by making calculations for the past (1980 or 1990) and for the future (2010 or 2020). Based on these calculations, we have concluded that the disease burden related to PM_{10} exposure will probably decrease, the noise-related disease burden will further increase, and the number of DALYs caused by radon and UV radiation is supposed to remain fairly similar.

Overall, around 2 to 5 % of the disease burden in the Netherlands (which was calculated based on data for 49 (groups of) diseases) is estimated to be attributable to the effects of air pollution (PM_{10} and ozone, short-term effects), noise, radon, UV and indoor dampness. Including the relatively uncertain effects of long-term exposure to PM_{10}, this may increase over 10%, assuming no threshold.

Introduction

Just as we are affecting our environment, the environment can also affect us. Several environmental determinants are known to have impacts on our health. Due to better control of these environmental factors and better environmental policies aimed at protecting population health, the contribution of our current environment to our total disease burden (morbidity and mortality) is probably reduced to a few percent. Lifestyle factors, such as smoking, food, alcohol, and exercise, presumably have a bigger influence on health.

Persistent environmental problems such as air pollution and noise still can affect human health quite considerably. Effects from air pollution range from aggravation of asthma to premature mortality, while noise exposure is associated with annoyance, sleep disturbance and effects on cognition. Because of the divergence in magnitude, duration and severity of these health effects, integrated health measures which convert all effects to a comparable unit, can be very useful for the interpretation and comparison of different (environmental) health problems. This is especially useful for evaluating and comparing different policy options and assessing cost effectiveness of mitigating measures or prevention. Experience with integrated health measures in environmental health decision-making is limited to the use of monetary cost estimates of health impacts, the Years of Life Lost (YLL) due to mortality and the total amount of healthy life lost expressed in Disability Adjusted Life Years (DALYs).

DALYs 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 has endorsed the procedure, and the DALY approach has been used in various studies on a global, national and regional level.

In spite of the increasing use of DALYs, the method is being debated. Some people address the very essence of the method by discussing whether it is ethically sound to quantify health and prioritize health problems. Furthermore, people argue that the method is -at least partly- subjective, since the severity values of health conditions (which can range from 0 (perfect health) to 1 (death)) are assigned by ‘expert panels’. Also, implicit to this weighing method,

people that are already disabled have ‘less health to lose’, and are therefore discriminated when policy measures are based solely on DALY outcomes. In general, DALY outcomes are relatively unstable, due to uncertain data used in the calculations. When using DALYs for health impact assessment of specific (environmental) determinants, an essential assumption is that of causality, as it is for any type of impact indicators. In these calculations, more variables, such as exposure assessment data and exposure-response relationships, are added, contributing to greater total variability of the output.

These drawbacks of calculating DALYs, which will be discussed in more detail later, are certainly valid. However, to this date, no better alternatives to quantify health are available, while the demand for such aggregated indicators is considerable. Policy makers need to allocate budgets, and, besides personal interest and public engagement, they want to base their decisions on information regarding severity, magnitude, policy options and costs. The only way to link these determinants is by making them comparable, and therefore by quantifying health, preferably aggregated in a single indicator. Yet it remains important to take all limitations of the method into account when presenting and interpreting the results.

This report presents the results of quantitative assessments of the environmental health burden in the Netherlands. It is based on (previous) work commissioned by the Dutch Health Care Inspectorate, the Ministery of Housing, Spatial Planning and the Environment (VROM) and the Netherlands Environmental Assessment Agency (MNP). Earlier, De Hollander *et al.* (1999) have completed a Dutch environmental health impact assessment using DALYs. They focused on numerous environmental exposures and concluded that around 5% of the annual burden of disease in the Netherlands can be attributed to these exposures, with long-term exposure to particulate air pollution as the greatest contributor. The Netherlands Environmental Assessment Agency (MNP) requested to repeat this exercise using more recent data, to describe trends over time and to further document all calculation steps. The outcomes presented in this report are used by MNP in policy documents, such as the Environmental Balance (Milieubalans).

We have assessed the health impact of some important environmental factors in the Netherlands: air pollution, noise, radon, UV, dampness in houses and traffic accidents. We have also examined the trends in these impacts over time, using past (1980/1990) and

scenario (2010/2020) data. In addition to comparing environmental health problems, this can enable evaluation of past policy measures and better future policy planning.

This report starts with an impression of the main environmental health problems in the Netherlands. Chapter 2 gives an overview of several health impact assessment measures. Chapter 3 describes the methods of our study, while Chapter 4 goes deeper into the specific data used for our DALY calculations. Chapters 5 and 6 present the results and discussion, as well as some conclusions and recommendations for future research. Further documentation on the calculations (in the form of spreadsheets) is available through the contact person (Anne.Knol@rivm.nl).

Various experts have given input for the calculations and this report. The sections on air pollution have been reviewed by Paul Fischer. Danny Houthuijs and Elise Van Kempen have assessed the paragraphs on noise, whereas Gert Kelfkens has worked on calculations and texts for radiation. Annemiek van Overveld has given input for the methodology section. Statistical analyses have been performed by Caroline Ameling. Erik Lebret has reviewed the report.

1. Health and the environment in the Netherlands

Health and quality of life are influenced by the environment. Most risks have been greatly reduced during the industrial revolution and its attendant economical progress. This is mainly caused by improvements in drinking water supplies, sewage systems, waste collection and housing (RIVM MNP and CBS and Stichting DLO, 2003a). However, industrialization and mass production have also introduced new risks. The introduction of chemical substances to water, air and food has lead to respiratory diseases and certain types of cancer, and has also caused some large scale industrial disasters, resulting in numerous victims (European Environment Agency, 2003). Nowadays, many of the known environmental risks have been regulated with standards, policies and risk abatement. Environmental policies have helped to reduce exposure to many substances. This has lead to increased life expectancies in the Dutch population (RIVM MNP and CBS and Stichting DLO, 2003a). However, several new environmental risks are affecting our health.

Environmental health impacts are driven by many factors, including economic growth, population growth and mobility, and urbanization (World Resources Institute, 1998). Due to economic and population growth, there is an increasing demand for transport, industry, energy and agriculture. This leads to increasing exposure to noise and air pollution, and decreasing quality of life in urban regions. In the Netherlands, many environmental health effects are driven by the increased use of transport. Although transport is considered an important part of the economy and lifestyle in western societies, it exacts a high price from society and the environment and it is an important source of pollution. The air pollution and noise emitted by traffic affect populations, particularly urban residents, reducing quality of life and promoting a range of less severe symptoms. However, the exact extent and impact of transport is not entirely known. The same is true for most other environmental health problems. Even if emissions are known, the translation to human exposure and health effects is difficult (World Health Organization, 2004a).

The basis of this report is an analysis of trends in the environmental disease burden associated with air pollution, UV radiation, radon, noise and dampness in houses in the Netherlands from 1980 up to 2020. We have also investigated the effects of traffic accidents. We will first describe the environmental factors and the associated health effects that we

considered. We have not been able to include all environmental factors which potentially cause health effects in the calculations, nor could we include all health effects potentially associated with these environmental factors. Therefore, final outcomes probably underestimate the environmental disease burden.

1.1 Air pollution

In previous years, the concentration of many air pollutants has been significantly reduced in the Netherlands. Various studies (World Health Organization, 2004b) suggest, however, that short-term variations in particulate matter (PM) are associated with adverse health effects even at low levels of exposure. PM is emitted by sources such as traffic (mainly diesel combustion) and industry. In the year 2001, around 14% of the urban population in Western Europe (the former EU15) was exposed to PM_{10} levels higher than 40 $\mu g/m^3$ (European Environment Agency, 2003). The effects of exposure to PM range from mild changes in respiratory function, through increased respiratory and cardiovascular morbidity, to increased total mortality. According to some studies and impact assessments, long-term exposure to particulate matter is associated with a reduction in life expectancy per victim in the order of about 10 years (Künzli *et al.*, 2001; WHO, 2005; AEA Technology, 2005).

High concentrations of ozone in the troposphere, typical for the summer months, lead to an increase in the frequency of respiratory symptoms. In addition to aggravating asthma, ozone-exposure can lead to more serious effects and is associated with premature death. Days with elevated ozone concentrations can also lead to decreased lung functioning (World Health Organization, 2003). The latter, however, has not been analyzed in our study, due to the difficulty in translating decreased lung function into a quantifiable health impact.

1.2 Noise

Many sources generate noise. Transport (road, rail and air traffic) is the most important source of community noise. Due to increasing transport demands, an increase in duration of exposure (due to the 24-hour economy) as well as an expansion of noise-exposed areas, noise will probably remain a major problem in the future.

Exposure to noise causes annoyance and sleep disturbance in a considerable part of the Dutch population (Miedema, 2001; Franssen *et al.*, 2004). Furthermore, noise exposure has also been associated with effects on children's learning (World Health Organization, 2004a).

However, the scope of the latter effect is as yet unclear and some necessary data unavailable; therefore it has not been included in the calculations.
Next to severe annoyance and sleep disturbance, we have also investigated the potential of noise exposure leading to hypertension (through stress) and hence cardiovascular diseases, potentially resulting in death (Van Kempen *et al.*, 2002). Although it is generally accepted that noise can affect the cardiovascular system, the exposure-response relationship between noise exposure and cardiovascular mortality is still being debated (Van Kempen *et al.*, 2002).

1.3 Radiation (radon and UV)

Radiation can broadly be divided into ionizing and non-ionizing radiation. Radon, which is a natural substance present in the soil and in certain building materials, is part of the first category, while UV radiation is non-ionizing.
Radiation can cause cell damage, potentially resulting in certain cancers. Radon is known to cause lung cancer due to inhalation of mainly indoor air which contains radon and radon decay products.
UV radiation, a component of sunlight, can cause effects such as sunburns or ageing of the skin, but can also cause immune suppression, cataract or skin cancer. Only skin cancer (morbidity and mortality) has been included in this study, since data and descriptive models for other effects were currently insufficient.

1.4 Indoor dampness

Besides more prominent indoor health problems, such as radon and ETS (environmental tobacco smoke), indoor dampness can also cause health effects, mainly affecting respiratory functions (Van Veen *et al.*, 2001). The most important effect of living in damp houses is (aggravation of) asthma. Children are especially vulnerable (Van Veen *et al.*, 2001). Damp houses can also indirectly cause allergic or rheumatic complaints (Pernot *et al.*, 2003), which were not included in this study.

1.5 Traffic accidents

Apart from causing noise and air pollution, traffic obviously has more direct health consequences in the form of traffic accidents. Although accidents might not fall under a

narrow definition of ‘environment’, it is part of the environment in a broader sense and is also a good standard for interpreting other (transport-related) health effects. In addition to mortality, traffic accidents can cause short-term as well as long-term (chronic) disability.

2. Health Impact Assessment

Health policies are based on many decisions and policy makers need to prioritize health problems in order to allocate their budgets. These types of decisions are often based on information regarding severity and magnitude of the problems as well as policy options and costs. Personal interest and public concern and engagement also play a role.

One way to compare different policy options is by carrying out a health impact assessment (HIA). HIA is a combination of procedures, methods and instruments used for assessing the potential health impacts of certain matters. These can vary from a single environmental factor to a more complicated set of factors, for instance in an infrastructural or industrial project. For quantifying health impacts, the following steps can be distinguished (Hertz-Picciotto, 1998):

- Selection of health endpoints with sufficient proof (based on expert judgements) of a causal relationship with the risk factor
- Assessment of population exposure (combination of measurements, models and demographic data)
- Identification of exposure-response relations (relative risks, threshold values) based on (meta) analyses and epidemiological and toxicological research.
- Estimation of the (extra) number of cases with the specific health state, attributable to exposure to the risk factor. This is a function of the population distribution, exposure-response relation and base prevalence of the health state in the population.
- Computation of the total health burden, or costs to society of all risk factors (if wanted/necessary)

A common problem is that the health effects of environmental factors can vary considerably with regard to their severity, duration and magnitude. These differences hamper the comparison of policies (comparative risk assessment) or the costs of policy measures (cost effectiveness analysis). An integrated health measure, using the same denominator for all health effects, can help with interpretation and comparison of health problems and policies.

2.1 Integrated health measures

Common health measures include mortality, morbidity, healthy life expectancy, attributable burden of disease measures, and monetary valuation. Some of these measures will be further described below. All methods have several associated difficulties, such as imprecision of the population exposure assessment; uncertain shapes of the exposure-response curves for the low environmental exposure levels; insufficient (quality of) epidemiological data; extrapolation from animal to man or from occupational to the general population; generalisation of exposure-response relations from locally collected data for use on regional, national or global scale; combined effects in complex mixtures, etc.

Mortality figures

The annual mortality risk or the number of deaths related to a certain (environment-related) disease can be compared with this risk or number in another region or country, or with data from another period in time. Subsequently, different policies can be compared and policies that do or do not work can be identified. Within a country, time trends can be analyzed. This method is easy to comprehend. No ethical questions are attached; everyone is treated equal. Since this method only includes mortality, it is not suitable for assessing factors with less severe consequences (morbidity). Also, it is difficult to attribute mortality to specific environmental causes.

Morbidity figures

Similar to mortality figures, morbidity numbers (prevalences or incidences based on hospital admissions or doctor visits) can be used to evaluate a (population) health state. Advantages and drawbacks are comparable to those applying to using mortality figures. The use of morbidity numbers is therefore similarly limited, especially when (environmental) causes of the diseases vary.

Healthy life expectancy

Using mortality tables, one can calculate the total average life expectancy for different age groups in a population, subdivided into years with good and years with less-than-good health. This measure is especially useful to review the generic health state in a country for the long term, but it doesn’t give insight into specific health effects, effects of specific policy interventions, or trends in certain subgroups.

Attributable burden of disease

Health impact assessments can also be executed by calculating the attributable burden of disease. There are several ways to assess the burden of disease attributable to an (environmental) factor, such as the QALY and the DALY.

Quality Adjusted Life Years, QALYs, capture both the quality and quantity elements of health in one indicator. Essentially, time spent in ill health (measured in years) is multiplied by a weight measuring the relative (un)desirability of the illness state. Thereby a number is obtained which represents the equivalent number of years with full health. QALYs are commonly used for cost-utility analysis and to appraise different forms of health care. To do that, QALYs combine life years gained as a result of these health interventions/health care programs with a judgment about the quality of these life years.

Disability adjusted life years, DALYs, are comparable to QALYs in that they both combine information on quality and quantity of life. However, contrary to QALYs, DALYs give an indication of the (potential) number of healthy life years lost due to premature mortality or morbidity and are estimated for particular diseases, instead of a health state. Morbidity is weighted for the severity of the disorder.

With QALY, the focus is on assessing individual preference for different non-fatal health outcomes that might result from a specific intervention, whereas the DALY was developed primarily to compare relative burdens among different diseases and among different populations (Morrow and Bryant, 1995). DALYs are suitable for analyzing particular disorders or specific factors that influence health. Problems associated with the DALY approach include the difficulty of estimating the duration of the effects (which have hardly been studied) and the severity of a disease; and allowing for combined effects in the same individual (first you have symptoms, then you go to a hospital and then you may die). The DALY concept, which has been used in our study, will be further described in the next chapter. More information on the drawbacks of the method can be found in Chapter 6.4.

Monetary valuation

Another approach to health impact assessment is monetary valuation. In this measure, money is used as a unit to express health loss or gain, thereby facilitating the comparison of policy costs and benefits. It can help policy makers in allocating limited (health care) resources and

setting priorities. There are different approaches to monetary valuation such as ‘cost of illness’ and ‘willingness to pay/accept’.

The *‘cost of illness’* (COI) approach estimates the material costs related to mortality and morbidity. It includes the costs for the whole society and considers loss of income, productivity and medical costs. This approach does not include immaterial costs, such as impact of disability (pain, fear) or decrease in quality of life. This could lead to an underestimation of the health costs. Furthermore, individual preferences are not considered.

The *‘willingness to pay’ (WTP)* approach measures how much money one would be willing to pay for improvement of a certain health state or for a reduction in health risk. The ‘*willingness to accep*t’ (WTA) approach measures how much money one wants to receive to accept an increased risk. WTP and WTA can be estimated by observing the individual’s behaviour and expenditures on related goods (revealed preference). For example, the extra amount of money people are willing to pay for safer or healthier products (e.g. cars with air bags), or the extra salary they accept for compensation of a risky occupation (De Hollander, 2004). Another similar method is *contingent valuation* (CV), in which people are asked directly how much money they would be willing to pay (under hypothetical circumstances) for obtaining a certain benefit (e.g. clean air or good health).

Advantages of these approaches are that the values represent individual preferences and include certain indefinable costs (e.g. pain, quality of life). The values also appear to be fairly stable in Western countries (De Hollander, 2004). A disadvantage is that the values are restricted to individual costs. Social costs are not incorporated. The reliability of the answers obtained in contingent valuation studies can be discussed, as people are spending ‘hypothetical’ money for ‘hypothetical’ health benefits. In addition, willingness-to-pay values have shown to be dependent on income.

2.2 Value of integrated health measures for policy makers

Health measures can assist policy makers in the decision making process, but cannot provide definite answers. However, depending on the methods used, they can facilitate comparison of environmental health risks in order to set priorities, evaluation of the efficiency of different

policy measures, and evaluation of accumulation of multiple environmental risk factors (in a certain area) (De Hollander, 2004).

Mortality figures are the simplest approach to integrated health measures, as they are easy to calculate and understand. A serious disadvantage is that non-fatal health outcomes are not incorporated in the calculations. Morbidity figures are also relatively easy to use, but do not include fatal outcomes or an indication of the severity of diseases. In addition, both mortality and morbidity figures are difficult to attribute to their exact causes. Therefore, mortality and morbidity figures only reveal part of a public health problem and are not very useful for complex policy questions related to environmental health.

In contrast, burden of disease measures (DALY's and QALY's) do include information on both fatal and non-fatal health outcomes and the quality of life associated with these outcomes. They provide information on public health in a country in a comparable way. In practice, however, there are still many disadvantages involved in these kinds of calculations, since very complex information has to be reduced to one single value. The results should therefore be handled with care, but can be very useful with proper explanation and clear description of the uncertainties involved.

Results of monetary valuation of health problems provide policy makers with crude estimations of the costs and benefits associated with certain policy decisions. However, expressing health in terms of money is complicated and many uncertainties are involved, as holds for attributable burden of disease measures.

It should be noted that comparing public health problems, whether in terms of DALYs or money or some other measure, should never be the only criterion on which to base policy. Other important factors not captured in these methods include for example solidarity and equity, and certain social impacts. However, the measures can provide valuable information as part of the whole decision making process.

3. Methods

The DALY is one of the most commonly used methods of the ones described in paragraph 2.1 and was first introduced by Murray and Lopez (1996) in collaboration with World Health Organization and the Worldbank in an attempt to introduce morbidity in mortality-based health discussions. The estimation of environmental DALYs starts with screening of the feasibility to describe the disease burden of certain environmental agents, e.g. availability of relevant and suitable information, weight of evidence, and severity estimates for specific effects. In this first step, environmental factors are selected for further analyses. We have analysed the environmental disease burden related to air pollution, UV, radon, noise, dampness in houses and traffic accidents from 1980 up to 2020. We have chosen these environmental factors because of their known relation to health, their importance in the Netherlands and the availability of data. Some other factors that can also be of great importance, such as some chemicals, infectious diseases and indoor tobacco smoke, have not been included, because we did not have access to all necessary data, and because of the difficulty to estimate the number of people exposed to some of these agents.

3.1 Consultation of experts

We have asked an internal group of experts to advice on general procedures, and to judge the weight of evidence and comment on the methods of our study. We have also consulted experts for each specific environmental health field, in order to use the most up-to-date information in our calculations, and to verify results. These experts have also been asked to make estimations for certain missing data, mainly for uncertainty ranges.

3.2 Calculations

DALYs incorporate three important factors of health: loss of life expectancy due to premature mortality, combined with the duration of living in a deteriorated health state, standardized to the severity of the deteriorated health state.

Some DALY calculations also use age weights and time preferences. Age weights indicate the relative importance of healthy life at different ages, for example, a rise of importance from birth until age 25 and decline thereafter. Time preference compares the value of health

gains today to the value attached to health gains in the future. In economic theory, the latter is assumed to be lower than the former.
However, for ethical reasons, we have chosen not to make a difference between elder and younger people and for sustainability and durability reasons, we have not discounted health benefits in the future. For more information on these and other discussion points: see paragraph 6.4.

In general, DALYs can be calculated using the equations below:

DALY = AB * D * S
AB = AR * P * F
AR = (RR'-1)/RR'
RR' = ((RR-1) * C) + 1

AB: Attributable Burden; the number of people in a certain health state as a result of exposure to the (environmental) factor that is being analyzed, not corrected for comorbidity.
D: Duration of the health state; for morbidity, prevalence numbers have been used and therefore duration is one year (except for hospital visits, for which the mean duration of the specific hospital visit has been used). For mortality, the duration of time lost due to premature mortality is calculated using standard expected years of life lost with model life-tables.
S: Severity; the reduction in capacity due to morbidity is measured using severity weights. A weight factor, varying from 0 (healthy) to 1 (death), is determined by experts (clinicians, researchers, etc).
AR: Attributive Risk; risk of getting a specific disease as a result of exposure to a certain (environmental) factor.
P: Base prevalence for morbidity; number of deaths for mortality
F: Fraction of the population exposed to the (environmental) factor under investigation (for air pollution, this fraction is set to 1, meaning that everybody is exposed to a certain degree)
RR': Adjusted Relative Risk
RR: Relative Risk
C: Concentration of the environmental factor, expressed in the unit of the Relative Risk

For some calculations, such as for noise, some calculation steps are unnecessary and are therefore skipped or replaced by other calculations. The individual theme chapter will discuss these specific methods. In this study, all calculations are corrected for changes in population size and composition (ageing), unless otherwise stated.

3.3 Data selection

The aim of this study was to analyze trends in the environmental burden of disease for various environmental factors for 1980, 2000 and 2020. If data for some of these years were not available, we have used alternative years (i.e. 1990 and 2010). Calculations were made on a national scale (the Netherlands).

As specified above, for the calculation of DALYs we need information on exposure to the environmental factor, and exposure-response relationships (relative risks), base prevalences at the reference level, duration, and severity for all associated health effects. Using the common steps of health impact assessments, as discussed in chapter 2, as a guideline, we will describe the methodological criteria of our data selection process. The selection of severity weights and years of life lost (duration), which is specific to DALY calculations, will be described as an extra step (step 5).

1) Selection of health endpoints

Health endpoints associated with the environmental factors have been selected based on the availability of sufficient proof for a causal relationship with the risk factor. We have consulted experts in order to gain insight into the weight of evidence for the relationships. In some cases, we have investigated health impacts of which there is no complete scientific consensus on the causal relationship (yet), but which we feel are important nonetheless. In these cases, the lack of scientific consensus is made explicit in the description of the results. Besides discussions with experts, we have used literature reviews, preferably by expert committees. If any data in subsequent steps were not available, the impact of the health endpoint under study could not be quantified. Therefore, some health effects have not or only partly (e.g. only hospital admissions) been included in the calculations.

2) Assessment of population exposure

We have calculated all DALYs for the general population, since exposure-response relationships were not based on individual exposure assessments, nor was extension to personal exposure considered useful, since such refinement and detail would be lost when combined with other more uncertain input data. Population exposure was assessed using a combination of measurements, validated dispersion models and demographic data. Usually, even though concentrations are measured at some locations, concentrations in other regions need to be estimated. Furthermore, the exposure of humans to these emissions is not measured and is therefore generally based on models. We have used a combination of measured and modelled values. If these values were not available for all years, they were estimated (expert judgements), interpolated or, as a last option, skipped. We have generally used the same year for data on exposure and data on health effects. However, some effects might occur much later than exposure (latency). It is difficult to allow for these effects. The individual thematic chapters will address the issue when relevant.

3) Identification of exposure-response relations

Relative risks are based on (meta) analyses of epidemiological and toxicological data. We have selected recent exposure-response relationships based on well founded Dutch epidemiological studies or, when Dutch data were not available or unsuitable, we have used the international estimate which best suited the Dutch situation. We have tried to select exposure-response relations from studies in which the exposure range of the subjects is similar to the exposure of the Dutch population. In addition to the difficulty to determine which exposure-response relation is best to use, potential problems can also relate to the slope of the relationship, the presence of a threshold and heterogeneity of the slope. Firstly, the slope and form of the curve influence the possibility to assess health effects at different exposures. We have tried to take the slope of (non-linear) curves into account. Secondly, whether there is a threshold or not can influence results substantially. We have used thresholds according to the latest insights of experts in the area under study. For example, we did not use thresholds for air pollution, UV and radon in our final calculations, but we did use thresholds for calculating the effects of noise annoyance and sleep disturbance, which are judged to only take place above a certain noise exposure level. Individual paragraphs in chapter 4 will go further into this issue. Lastly, heterogeneity of a relationship can result in

different values for different geographical regions or different age categories. We have therefore tried to the degree possible to use Dutch data and studies.

4) Estimation of the extra number of cases with the specific health state

To estimate the number of cases attributable to a certain environmental factor, a function of the population distribution, exposure-response relation and base prevalence of the health state in the population is used. Prevalence data were ideally taken from national mortality and morbidity registries, as used in the National Public Health Compass (RIVM, 2004). Most data were available per 5 year age categories. Past and future values were based on demographic projections and the assumption that the share of age groups within the total mortality/morbidity number has been and will remain equal over the years. All demographic data were based on data from Statistics Netherlands (CBS).
If prevalence data were not available, modelled or estimated values were used. For example, data for cardiovascular disease hospital admissions were available for the years 1980 and 2000 (Nederlandse Hartstichting, 2004), however, for 2010 the prevalence had to be estimated. We did this by assuming that the trend in cardiovascular hospital admissions is similar to the trend in the number of hospital admissions for coronary heart disease. For the latter, as well as for the number of clinical hospital admissions for COPD and total respiratory diseases, we have used the trend in data from 1980 to 2003 in order to estimate values for 2010 and 2020.

5) Selection of severity weights and duration of health effects

Severity weights
Severity weights (or disability weights) are determined by expert panels, such as doctors and scientists. For this exercise, we mainly used the weight factors as used by the Department of Public Health Status and Forecasting (RIVM, 2004a) and the background report for the Public Health Status and Forecast 1997 (Melse and Kramers, 1998).

These weights were evaluated by Stouthard *et al.* (1997) by comparing different stages of one disease, by comparing similar stages of different diseases and by comparing the severity weights with the predicted weights of the EuroQol (5D+) model. The latter is a model which evaluates health states based on six health dimensions: mobility, self-care, daily activities, pain or discomfort, anxiety or depression and cognitive functions. The weights have also

been compared with the severity weights of the Global Burden of Disease study (Murray and Lopez, 1996). Stouthard et al also studied the reliability of the weights by using panels and a written procedure. The values and ranking order of the weights attributed by the panels were very similar to one another. They were also similar to weights attributed by other experts using the written procedure. Based on these evaluations we consider these severity weights of sufficient quality.

If severity weights were not available from the abovementioned sources (such as weight factors for hospital admissions for respiratory and cardiovascular diseases) we have used weights from De Hollander *et al.* (1999). In the absence of a pre-existing weight factor, De Hollander used a calibration scale drawn up by Stouthard to have environment-oriented physicians evaluate health states.

For severe annoyance and sleep disturbance, we have slightly altered the estimate of De Hollander. These health outcomes are not often included in weighting exercises and the available weights vary considerably. We have chosen to use a weight factor of 0.02 and allow for the relatively great uncertainty by using 0.01 (weight as used in De Hollander, 1999) as a minimum value and 0.12/0.10 (for severe annoyance/ severe sleep disturbance respectively, as described in Van Kempen (1998)), as maximum values in our Monte Carlo analysis.

Duration

The duration of a health effect is based on the number of healthy life years lost. For morbidity, prevalence data were used and duration was therefore set to one year (assuming that prevalence approximately equals incidence multiplied by duration, and thereby assuming a steady-state equation where the rates are not changing).

For mortality caused by those environmental factors that are “completely responsible” for death (such as traffic is responsible for traffic accident mortality), we have used mean life expectancy minus mean age of death as the number of years of life lost. For factors only partly responsible for death (such as short-term exposure to PM_{10} accelerating death in people that are already diseased), we have used estimations of the attributable years of life lost. Years of life lost were calculated similarly for all years.

It is interesting to realize that two different and opposing factors play a role when estimating trends in years of life lost. If for example mortality numbers are decreasing, there will be fewer years of life lost. However, less mortality also means a greater life expectancy, thus increasing the number of years of life lost.

6) Computation of the total health burden of all risk factors

The last step in the general procedure of a quantitative health impact assessment is the computation of the total disease burden, if wanted, by adding up all DALYs. Since we were not able to include all environmental health factors that significantly affect health in our calculations, our total burden of disease is probably an underestimation. However, this study could be regarded as the current 'state of the art' and gives an indication of the total disease burden caused by the factors studied, and the overall trends in this burden.

3.4 Monte Carlo analysis

Due to the large number of variables in the DALY calculation, each with its own uncertainty range, results should be interpreted with care, and uncertainty analysis is relevant.

In this study, a Monte Carlo uncertainty analysis has been applied to all computations, using the uncertainty ranges (based on literature or expert judgements) for the exposure, relative risk, severity factor and duration. With this method, a 90% prediction interval can be estimated around the output. In a Monte Carlo analysis, all variables are attributed a series of random values within their range, thereby estimating the range of the output distribution. In this type of simulation, probability distributions are defined for each uncertain variable. Figure 1 shows some different types of these distributions. For each variable, the distribution type is based on conditions and assumptions relevant to that variable. Based on these distributions, a Monte Carlo simulation calculates multiple scenarios of a model by repeatedly sampling values from the probability distributions for the uncertain variables. We used the version 4.5 of @risk software (Palisade Decision Tools) to execute these simulations.

Figure 1 Some types of probability distributions (Decisioneering website, 2004).

We present the values of the 90% prediction intervals for all Monte Carlo calculations. The 90% prediction intervals of the cumulative numbers mentioned in this report (the totals per environmental factor, as shown in all graphs) can be slightly different from the intervals that

can be derived by adding up the effect-specific 90% prediction intervals (which are shown in the table in the appendix). This is because all separate health effects have been treated as independent variables, and therefore their most extreme values will not be likely to coexist when analysing all effects simultaneously.

Apart from uncertainty within variables, there is also uncertainty caused by qualitative differences between variables. All input variables have different origins. Some variables are based on Dutch data, but, if not available, international (meta-) analyses were used. Also, some variables are modelled, while others are measured. Some uncertainty ranges are modelled and calculated, while others are based on expert judgments. Severity factors are derived from different sources due to incompleteness of individual studies. Evidently, these differences hamper comparison. Also, one should realize that the DALY is a composite measure that cannot be directly measured in a population. Therefore, a direct validation of modelled outcomes against empirical data is not possible. More information on uncertainty in the DALY calculations can be found in paragraph 6.2.

4. Data

The following paragraphs describe the specific sources of the data we have used for the estimations of the disease burden that can be attributed to air pollution, noise, radon, UV, indoor dampness and traffic accidents. The values and uncertainty ranges for severity and duration estimates can be found in Appendix 1.

4.1 Air pollution

We have investigated the effects of PM_{10} and ozone on the health of the Dutch population. PM_{10} can be regarded as an indicator of a more complex mixture of urban air pollutants. For the effects of short-term PM_{10} exposure, health effects taken into account include mortality (total and subdivided in cardiovascular and respiratory mortality, including COPD), and hospital admissions for cardiovascular and respiratory disease (total and separately for COPD and asthma). For long-term PM_{10} exposure, we have analyzed total mortality.
The health effects of short-term ozone exposure that we have considered are mortality (total and subdivided in cardiovascular and respiratory mortality), hospital admissions for cardiovascular and respiratory disease (total and separately for COPD and asthma) and asthma attacks (separately for children and adults).
Time spent outside the hospital while still suffering from the disease or disability is not included in our DALY outcomes due to lack of data.

Exposure

Data on concentrations of PM_{10} are a based on dispersion models, and adjusted for underestimation of these models by using measurement data, which gives most accurate concentration data (RIVM MNP. Personal communication. Hammingh P. 2004). The modelled data were calculated for the years 1980, 1995 and 2010. These values have been used to create a 'best fit' line and calculate a value for the year 2000 (Figure 2). Average PM_{10} concentrations are estimated to decrease from around 48 $\mu g/m^3$ in 1980 to 29 $\mu g/m^3$ in 2010. Uncertainty of these values lies around 30%.

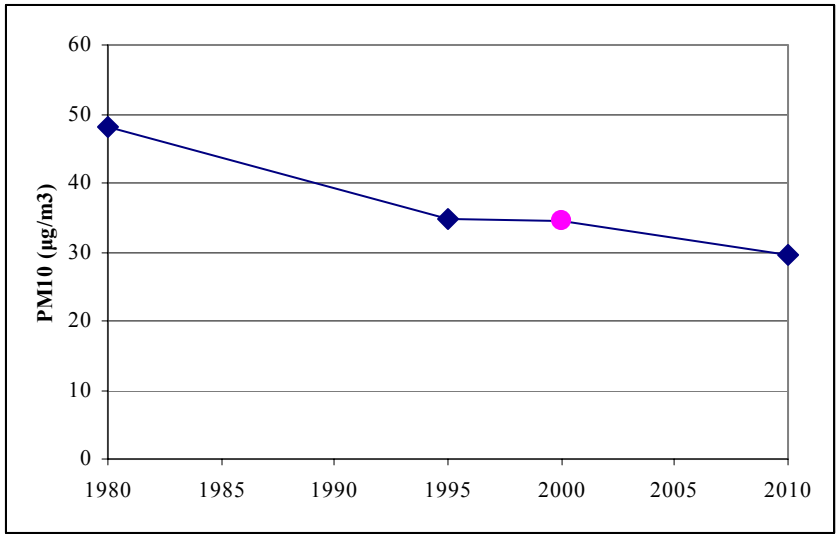


Figure 2 Annual PM_{10} concentrations ($\mu g/m^3$) in the Netherlands, 1980 – 2010 (PM_{10} concentration for the year 2000 based on a 'best fit' line)

It is generally agreed that a 'no effect level' for PM_{10} does not exist: health effects can occur at all levels of exposure. However, it can be realistic to calculate health effects of PM_{10} using certain fixed levels as minimum exposures. For example, an ambient PM_{10} concentration of 10 $\mu g/m^3$ can be regarded as a 'background concentration'. DALY outcomes based on this level will only include attributable mortality due to man-made pollution. An alternative reference level is 20 $\mu g/m^3$, which represents the target set for the year 2010 by the European Union. DALY outcomes then give an indication what part of mortality could be avoided if this level was to be achieved. We have calculated DALYs using no threshold (=0 $\mu g/m^3$), a 10 $\mu g/m^3$ reference level and a 20 $\mu g/m^3$ reference level. The DALYs calculated without a threshold were used in the final outcome presentation in order to show the complete PM_{10}-related disease burden, which is what we have done for the other environmental factors as well.

For ozone, mean daily max 8-hour values were available for 1990 and 2000, based on measured values. For the year 1990, in order to correct for annual peaks, the mean value of 1990-1992 has been used (1989 was not available) and for the year 2000, we have used the mean of the years 2000 to 2002. The uncertainty range is around 15% (RIVM MNP. Personal communication. Hammingh P. 2004). There is no clear trend visible in the ozone concentrations from 1990 – 2002 (Figure 3) and it is currently not possible to estimate stable future values.

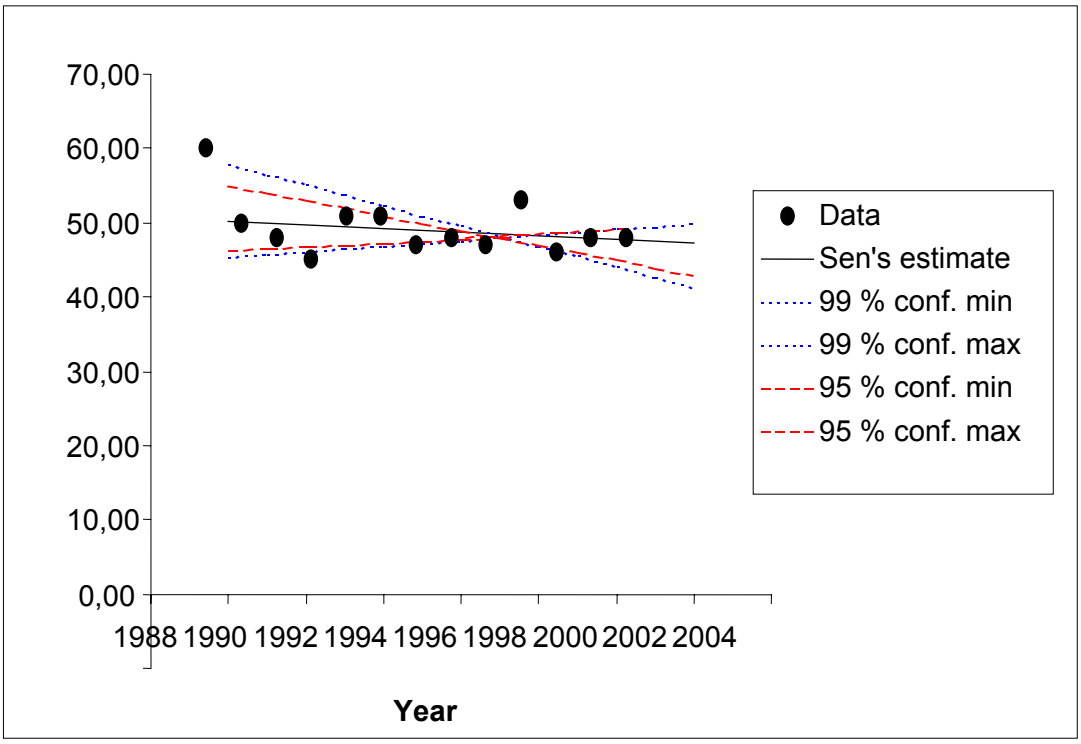


Figure 3 Annual mean of daily 8 hour (13-20h) mean ozone values in the Netherlands in µg/m^3, 1990 - 2002

Prevalence

Prevalence data are derived from various registrations as used in the National Public Health Compass (RIVM, 2004a). The prevalence data originated mainly from general practitioners registries. Some are supplemented with numbers from hospitals or nursing homes.

Exposure-response

Numerous epidemiological studies have investigated the health effects of short-term exposure to PM_{10} or ozone and several studies are currently underway. Still, the exposure-response relationships are not completely clear. This is partly due to the fact that a single best relationship does not exist and can therefore not be found: the exposure-response relationships can differ in place as well as in time. For example, for PM_{10}, the exposure-response relationships might have changed in the last decades. Current research (RIVM, not published yet) analyzes new PM_{10} data series in order to investigate these potential trends, which could for instance be caused by changes in the mixture of air pollutants that PM_{10} represents.

The tables in this paragraph show the air pollution-related health effects that were taken into account in the calculations, and the accompanying exposure-response relationships we have used. We have tried to use the most recent studies that we considered most applicable to the Netherlands. For the short-term effects, we have chosen to use a constant lag time (lag 1), indicating that, in order to derive exposure-response relationships, health effects have been related to PM_{10} exposure one day earlier. This assumption affects the relative risks and thereby the DALY outcomes. For the year 2000, we were able to make calculations using a 2 component-model, in which the effects of PM_{10} and ozone are integrated (see paragraph 5.1). In this model, a lag-time of seven days is used.

PM_{10} long-term exposure

As yet, not many studies have investigated the potential health effects related to long-term PM_{10} exposure. For the calculation of the DALY for long-term exposure to PM_{10} we used the estimate for the concentration – response curve from Künzli *et al.* (2000), which is a 4.3% increase in mortality per 10 $\mu g/m^3$ increase in PM_{10}. This estimate is the weighted average from two American cohort studies (Dockery *et al.*, 1993; Pope *et al.*, 1995). Dockery measured the 14-to-16-year mortality follow-up of 8111 adults in six U.S cities and related this to air pollution. Pope linked ambient air pollution data from 151 US metropolitan areas in 1980 with individual risk factor on 552,138 adults. These two American cohort studies are at the moment the best cohort studies available for application to the Dutch situation. Two additional studies have assessed the long-term effects of ambient air pollution in a cohort of 50,000 US-veterans (Lipfert *et al.*, 2001) and in a cohort of 6,338 nonsmoking Californian Seventh-day Adventists (Abbey *et al.*, 1999). We think that the results of these two studies are less transferable to the Dutch population because they were performed in specific groups, and therefore less representative for the general population than the studies of Dockery *et al.* and Pope *et al.* In 2002, Pope has published an updated analysis of the mortality follow-up for a longer period (Pope *et al.*, 2002), which confirmed the results of the first analyses. For comparing purposes, the various relative risks of the individual studies are presented in Table 1. We have converted the $PM_{2.5}$ relative risks into PM_{10} relative risks by applying a $PM_{2.5}/PM_{10}$ ratio of 0.67 (Cyrys *et al.*, 2003). When using the individual study results and the ranges of uncertainty between the individual studies, our risk estimate of 4.3% increase in mortality per 10 $\mu g/m^3$ increase in PM_{10} is surrounded by a lowest point estimate of 4.2% (Pope *et al.*, 2002) and a highest point estimate of 9.2% (Dockery *et al.*, 1993). Taken into account the range in confidence intervals of the individual studies, a lowest estimate of 1.4%

was found in the Pope (2002) study, while the highest 95% confidence estimate of 16.1% increase per 10 µg/m^3 PM_{10} was calculated in the Dockery (1993) study.

Table 1 Relative risks of different studies for mortality due to long-term PM_{10} exposure

	Reported RR	**Based on**	**PM_{10} per 10 µg/m^3 (recalculation)**
Kunzli 2000	1.043 (1.026 - 1.061)	PM_{10}	1.043 (1.026 - 1.061)
Pope 1995	1.066 (1.036 - 1.099)	$PM_{2.5}$	1.046 (1.025 - 1.070)
Dockery 1993*	1.085 (1.028 - 1.146)	PM_{10}	1.085 (1.028 - 1.146)
Dockery 1993	1.132 (1.042 - 1.230)	$PM_{2.5}$	1.092 (1.029 - 1.161)
Pope 2002	1.060 (1.020 - 1.110)	$PM_{2.5}$	1.042 (1.014 - 1.077)
Abbey 1999	1.045** (0.992 - 1.101)	PM_{10}	1.045 (0.992 - 1.101)
Lipfert, 2000	n.a.	n.a.	n.a.

* based on EPA 8-124
** For men only. For women <1

PM_{10} short-term (peak) exposure

For the effects of peak exposure to PM_{10}, valid estimates based on the Dutch situation and population were available (Table 2), and therefore these were considered most appropriate for our study. Exposure-response relationships for mortality are based on the most recent 7-year time-series analysis of PM-associated premature mortality in the Netherlands (Buringh and Opperhuizen, 2002). For morbidity (hospital admissions), a recent analysis from the University of Groningen has been used (Vonk and Schouten, 2002), in which the linear short-term relationship between daily air pollution and the number of (emergency) hospital admissions was investigated, using data from 1992 to 1999.

Internationally, many other exposure-response relationships for acute effects of PM_{10} exposure have been published. Künzli *et al.*, for example, have also published relative risks (Künzli *et al.*, 2000, exposure-response relationships not presented here), based on a meta-analysis of various air quality studies, and calculated as the variance-weighted average of all studies. These relative risks are considerably higher than the estimates based on the Dutch data. For comparison, we have also calculated DALYs using the RR by Künzli for short-term PM_{10} exposure related effects, when available.

Table 2 Relative risks of morbidity and mortality effects of short-term exposure to PM_{10}, as used in the DALY calculations, based on Dutch data

	Reference	**Original Relative Risk**	**Relative Risk per per 10 µg/m³**
Mortality (total)	Buringh *et al.*, lag 1 (2002)	1.036 (1.025-1.047) (per 100 µg/m³)	1.0036 (1.0025 - 1.0046)
Cardiovascular disease mortality	Buringh *et al.*, lag 1 (2002)	1.025 (1.009-1.042) (per 100 µg/m³)	1.0025 (1.0009 - 1.0041)
Respiratory disease mortality	Buringh *et al.*, lag 1 (2002)	1.120 (1.084-1.157) (per 100 µg/m³)	1.0114 (1.0084 - 1.0157)
COPD mortality	Buringh *et al.*, lag 1 (2002)	1.111 (1.064-1.161) (per 100 µg/m³)	1.0106 (1.0062 - 1.0150)
Hospital admissions cardiovascular disease	Vonk et al lag 1, (2002)	1.020 (1.012-1.028) (per 63 µg/m³)	1.0032 (1.0019 - 1.0044)
Hospital admissions respiratory disease (total)	Vonk et al lag 1, (2002)	1.030 (1.016-1.043) (per 63 µg/m³)	1.0047 (1.0025 - 1.0067)
Hospital admissions COPD	Vonk et al lag 1, (2002)	1.054 (1.034-1.075) (per 63 µg/m³)	1.0084 (1.0053 - 1.0115)
Hospital admissions asthma	Vonk et al lag 1, (2002)	Not significant	-

Ozone

As mentioned above, we have used most recent Dutch studies to analyze ozone-related mortality. Although there have been previous estimates of ozone-related morbidity (hospital admissions), recent Dutch research does not yield reliable exposure-response relationships (Vonk *et al.*, 2002). The latest WHO meta-analysis of time-series and panel studies for PM_{10} and ozone (Anderson *et al.*, 2004) does not find significant positive relationships for ozone-related morbidity either.

There is also insufficient evidence to quantify a relationship between long-term ozone exposure and health effects (World Health Organization, 2003), apart from a reduction in lung function development (World Health Organization, 2004b), which is not quantifiable. Therefore, we have chosen not to include long-term effects nor morbidity effects of ozone in this study. Table 3 shows the relative risks used in our study.

Table 3 Relative risks of morbidity and mortality effects of short-term exposure to ozone as used in the DALY calculations

	Reference	**Original Relative Risk**	**Relative Risk per per 10 µg/m^3**
Mortality (total)	Buringh *et al.*, lag 1 (2002)	1.041 (1.024-1.059) (per 150 µg/m^3)	1.0026 (1.0016 – 1.0038)
Cardiovascular disease mortality	Buringh *et al.*, lag 1 (2002)	1.032 (1.003 – 1.061) (per 150 µg/m^3)	1.0021 (1.0002 – 1.0040)
Respiratory disease mortality	Buringh *et al.*, lag 1 (2002)	*Not significant*	-
COPD mortality	Buringh *et al.*, lag 1 (2002)	*Not significant*	-
Hospital admissions cardiovascular disease	Vonk *et al.* (2002), lag 1	*Not significant*	-
Hospital admissions respiratory disease (total)	Vonk *et al.* (2002), lag 1	*Not significant*	-
Hospital admissions COPD	Vonk *et al.* (2002), lag 1	*Not significant*	-
Hospital admissions asthma	Vonk *et al.* (2002), lag 1	*Not significant*	-

Duration

Short-term exposure to PM_{10} or ozone is thought to aggravate existing disease and only cause death when a person is already weakened by this other disease. Therefore, the added loss of life expectancy caused by the short-term exposure to PM_{10} or ozone is probably limited to several months (estimated as 1 to 5 months; expert judgement (RIVM. Personal communication. Fischer P. 2005)).

The mean duration of hospital admissions due to cardiovascular or respiratory disease is estimated to be around 2 weeks, ranging from 4 days to 2 months (De Hollander *et al.*, 1999). In this study we assume that long-term PM_{10} exposure is related to all cause mortality and that the PM_{10}-related deaths have the same age distribution as the total population. We did not apply age-specific relative risks since these are currently not available from the underlying studies or the differences in age-specific relative risks are not statistically significant (Pope, 2002). Long-term exposure to particulate matter is associated with a reduction in life expectancy per victim in the order of about 10 years (Künzli *et al.*, 2001, WHO, 2005, AEA Technology, 2005). In the absence of data, we have used an uncertainty interval of 10%, which is what we have done for most durations derived from mortality tables. However, given the substantial uncertainty within the long-term PM_{10} research, this may be an underestimation, leading to greater uncertainty in the final DALY outcomes. In

order to estimate the magnitude of this potential variation in the number of years of life lost, we have made alternative calculations using a greater uncertainty interval, which we created by using age-specific duration estimates. It is as yet unclear to what extent PM_{10}-related mortality occurs more frequent in certain age categories. However, this could influence the estimate of the number of years of life lost. Using age-specific mortality data from Statistics Netherlands (CBS), we have estimated that the years of life lost might range from around 4 years (deaths only occurring in the population over 85 years old) to around 13 years (deaths occurring in the population over 25 years old). We have made an alternative calculation of the long-term PM_{10}-related disease burden using these values in the uncertainty analysis, thereby potentially giving a more realistic view of the uncertainty interval (90% prediction) surrounding the DALY output.

Severity

All severity factors have been derived from the sources mentioned in paragraph 3.3. For hospital admissions for asthma and COPD, the weight factors for ‘severe asthma’ and ‘severe COPD’ have been used (0.36 and 0.53 respectively).

4.2 Noise

Noise can have several effects on human health. We have analyzed severe annoyance and severe sleep disturbance caused by several traffic noise sources (industrial noise, neighbour noise, etc, have not been included). We have also included the potential effect of noise on hypertension, which could lead to cardiovascular diseases and potentially premature mortality. Other effects, such as the potential influence on cognition, have not been incorporated, because there is no usable exposure-response relationship available as yet.

Exposure

For calculation of the noise exposure of the Dutch population, the EMPARA model (Dassen, 2001) has been used, which uses characteristics of the noise sources to calculate noise emissions and generate noise maps. These emissions are then translated to human noise exposure, using noise propagation paths and demographic data. We have calculated the number of dwellings exposed to certain levels of transport noise (per 1 dB) for the years 1980, 2000 and 2020. By multiplying the percentages of exposed dwellings with population numbers, the number of people exposed to the various noise levels has been calculated. The

noise sources include road traffic (municipal, provincial and national roads), rail traffic and air traffic (only around the major Dutch airport, Schiphol). For road traffic, cumulative data have been used to account for simultaneous exposure to noise from multiple road types. Cumulative data could also be derived for simultaneous exposure to road, air and rail traffic. However, these combined source data could not be used for annoyance and sleep disturbance calculations, because the exposure-response functions used to calculate the number of annoyed or sleep disturbed people are specific for each source. The cumulative numbers have been used to calculate mortality numbers, for which we made calculations using the same exposure-response relationship for each source. The validity of this assumption of source similarity for mortality can, however, be debated (see paragraph on exposure-response).

For the year 2020, two scenarios have been assessed: a scenario based on current policy efforts (policy as usual) and a scenario based on optimal policy efforts to reduce noise at the source. This second scenario assumes a 5 dB noise reduction for all sources (except air traffic), which can be useful to evaluate or plan policy measures aimed at reducing noise. Measures that can be implemented in order to achieve such a noise reduction are described in paragraph 5.2. The noise exposure distribution (in percentages) for all years is shown in Figure 4. The number of people in dwellings exposed to the higher noise classes has increased from 1980 to 2000, and will continue to increase in a policy-as-usual scenario. When optimal source policy is implemented, however, the number of (highly) exposed people will presumably be significantly reduced.

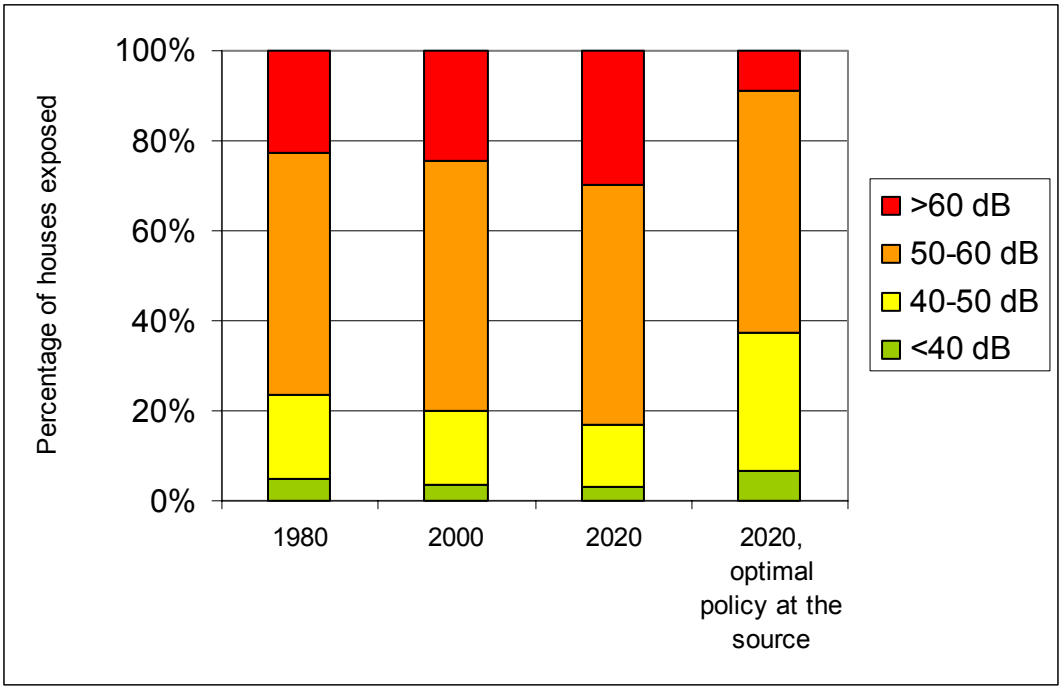
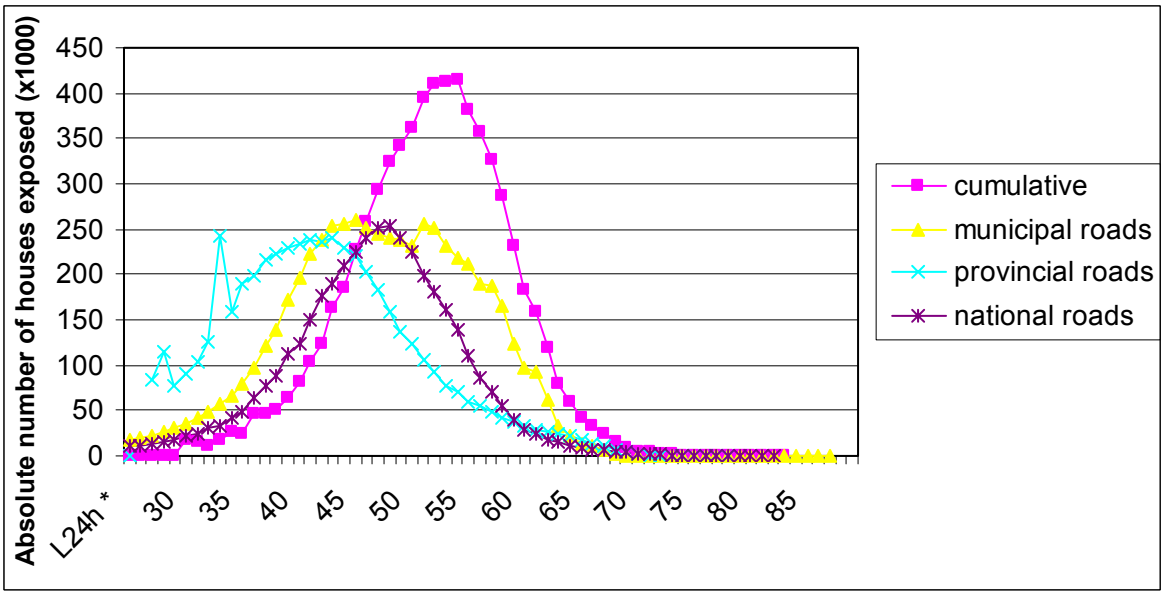


Figure 4 Noise exposure distribution (Lden, outside) based on exposed dwellings in the Netherlands for 1980, 2000, 2020, and for 2020 with optimal policy for road and noise traffic.

Road traffic accounts for the largest share of transport noise, with municipal (urban) roads as the greatest contributor to road traffic noise (Figure 5).



* $L_{24h} = \max (L_{day}, L_{evening} + 5, L_{night} + 10)$

Figure 5 Absolute number of dwellingss exposed to certain sources of road transport noise in the Netherlands, 2000

These estimations of noise exposure should be interpreted with caution. Exposure is based on the noise level on the most exposed part of the house. Varying degrees of isolation of these

houses can influence personal exposure and affect the exposure distribution. Some other uncertainties also play a role, such as uncertainty in the estimated traffic volume (which has been measured for the year 1980 and 2000, but estimated for 2020 using the EC scenario of the 5th Environmental Outlook (Idenburg, 2001) and uncertainties in the models (influence of buildings, noise barriers, etcetera). The form of the exposure curves is probably realistic, however, uncertainty lies mainly in the locations of the peaks of the curves.
As an estimate, we have used an uncertainty range of +/- 1 dB(A). Although uncertainty might be larger for estimates in the past and the future, we have kept the same uncertainty intervals for all years, treating the past and future values as scenarios.

Prevalence

In contrast to other health outcomes, by definition there is no base prevalence for noise annoyance or sleep disturbance. The prevalence of noise annoyance and sleep disturbance is estimated as described in the following paragraph, using exposure-response models. This is an indirect way of estimating the prevalence. These relationships are based on combined results from various studies. Because questions and response categories in these studies differ, all results have been translated to a 0-100 scale (Miedema *et al*, 2001), in which a 72 cut-off is applied for the percentage of people that is highly annoyed (cut off at 50 is the percentage of people being 'annoyed').

Another option to estimate the prevalence of annoyance and sleep disturbance is by directly using numbers from surveys. The number of people reporting annoyance in surveys is generally higher than the numbers that might be expected based on models using the established exposure-response relationships (numbers from the most recent noise annoyance inventory in the Netherlands are given in the results section, paragraph 5.2) even though these models are principally based on survey data. This discrepancy can be caused by various reasons, which will also be addressed in the results section where outcomes will be compared.

The prevalence of hypertension is estimated to be 24% in men and 19% in women in age category 20-60. For older people, these percentages are higher (RIVM, 2004a). These prevalences have been corrected for population size, but not for composition of the population (ageing).

Exposure-response

Exposure-response curves indicating the percentage of people (severely) annoyed or sleep disturbed at certain noise exposure levels have been derived by Miedema *et al.* (Miedema *et al.,* 2001, 2003). These curves are recommended for use in the EU Directive on Noise (World Health Organization, 2004a). They have been derived for road, rail and air traffic noise and severe annoyance, and road and rail traffic noise and severe sleep disturbance. A relationship for air traffic and sleep disturbance was not proposed by Miedema (2002), because of the large variance in outcomes. In a follow-up study (Miedema and Vos, 2004), Miedema included some new studies and proposed a relationship for air traffic and sleep disturbance. However, these curves are only indicative and involve much more uncertainty than the curves for road and rail traffic-related sleep disturbance. Therefore, we did not use them for our current study.

The annoyance curves of Miedema and Oudshoorn (2001) are based on a pooled analysis of datasets from noise annoyance studies in several regions, inside as well as outside Europe. Methodological differences in these studies could have influenced the relationships. However, more recent analyses (TNO, not published yet) based on newer datasets do not show any systematic changes. Results from noise annoyance and sleep disturbance research around Schiphol have not been included in the exposure-response relationships by Miedema, since outcomes of these studies were considered incomparable to the other studies that were used for the derivation of the relations (Ministerie van Verkeer en Waterstaat, 2000). However, vice versa, this would imply that the curves derived by Miedema are not applicable to Schiphol airport, which is the largest source of air traffic-related noise annoyance in the Netherlands. Therefore, it might not be completely valid to use these relations for the Dutch situation.

Also, the curves can only be applied to long-term stable situations (no changes in number of flight, flight routes, etc) and cannot be used to analyze short-term or local noise problems. Whether this precondition is realistic is questionable, since a stable situation is hardly ever reached at airports, where development and change is practically ongoing (Van Kempen *et al.*, 2005).

In our calculations, only severe annoyance and severe sleep disturbance have been included. Figure 6 shows the exposure-response curves by Miedema et al for road traffic and severe annoyance and sleep disturbance. We have used third order polynomials that Miedema

proposed as workable versions of the more complicated original curves. For severe annoyance, data below 45dB and above 75dB (Lden) were excluded from his study because these were judged less essential and too uncertain (Miedema, 2001). Furthermore, for the polynomial, a zero severe annoyance level has been set to 42 dB. In our study, people exposed to levels below 42 dB have not been included in the calculations, while people exposed to levels above 75 dB have been regarded as exposed to 75dB. The curves for severe sleep disturbance are valid in the range 45-65 dB (Lnight). People exposed to noise below 45 dB have not been included. Noise levels above 65 dB have been regarded as 65 dB. The validity ranges of these curves are based on the available measurement data, and do not necessarily imply that no annoyance or sleep disturbance will occur beneath these thresholds.

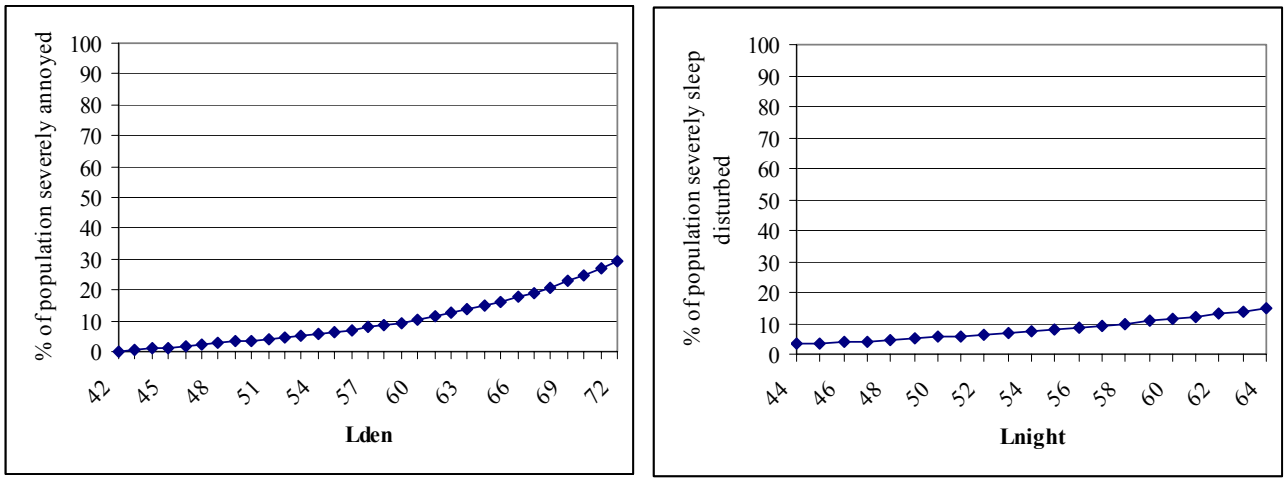


Figure 6 Exposure-response relationships for road traffic noise exposure and severe annoyance (left) and severe sleep disturbance (right). (Miedema et al., 2001- 2004)

Partly due to the influence of non-acoustic factors, (severe) annoyance or (severe) sleep disturbance is difficult to quantify. This difficulty is illustrated by the fact that different approaches to measure noise annoyance/ sleep disturbance can yield diverse results. In order to demonstrate this variability and its effects on calculations, we have also calculated DALYs using a recent Dutch study on environment-related annoyance and quality of life (Franssen *et al.*, 2004). In this study, 2000 randomly selected people in the Netherlands were interviewed and were asked how often they experienced annoyance or sleep disturbance as a result of exposure to noise from various sources. In this study, no exposure data were examined. The study took place in the year 2003 and showed that 29% of the respondents reported severe annoyance due to noise from one or more road traffic sources, 12% of the people experienced

severe annoyance caused by air traffic, whereas rail traffic was a source of severe annoyance for 2% of the respondents. For severe sleep disturbance, these percentages were 12%, 3% and 1% respectively. We have used these percentages to calculate DALYs in order to compare them with the results based on the curves by Miedema.

The exposure-response relation for noise and hypertension is based on a meta-analysis (Van Kempen *et al.*, 2002), which was based on data for air traffic ($RR_{5\ dB(A)}$ = 1.26 (1.14 - 1.39). In this study, we have used this same relation for road and rail traffic as well, which may be an overestimation. This relative risk is valid from 55-72 dB(A). The relative risk for hypertension based mortality is estimated to be 1.4 (1.2-1.6) based on meta-analysis (RIVM. Personal communication. Hoogenveen R. 2004).

Duration

We have used prevalence data for annoyance and sleep disturbance, and the duration is therefore one year. As an estimation of the loss of life expectancy via noise → hypertension → cardiovascular disease → mortality, we have used the mean loss of life expectancy for mortality due to cardiovascular disease (almost 11 years), which has been derived from mortality tables (RIVM, 2004a).

Severity

Severe annoyance and sleep disturbance are hard to weigh, because there is little information on their relationship with quality of life measures. We have used a severity factor of 0.02, with a relatively large uncertainty interval (0.01-0.12 for annoyance, 0.01-0.10 for sleep disturbance). The minimum value (0.01) is based on De Hollander *et al.* (1999), who used a panel of environment-oriented physicians to attribute severity weights to various health states based on a protocol by Stouthard (1997). The maximum values (0.10 and 0.12) are based on Van Kempen (1998) who did a panel study with 13 medical experts, also based on a protocol by Stouthard. In that study, sleep disturbance and annoyance were weighted relatively high.

Since the weight factors are so small, these variations have a relatively big impact on the outcomes.

4.3 Radon

Radon is a radioactive gas that is formed during the natural breakdown of uranium in soil, water and stony materials. It finds its way into houses through cracks and holes in the

foundation. Radon and its decay products can cause lung cancer. Studies show that radon is more of a risk to smokers than to non-smokers. However, the latter also have an elevated chance of developing lung cancer when exposed to radon. We have calculated the radon-related burden of disease based on lung cancer mortality.

Exposure

As a relevant exposure measure, we have used the mean individual dose over 10 years before lung cancer diagnoses. This dose is proportional to the mean indoor radon concentration over the same period of time. Since we are investigating lung cancer, we have confined ourselves to inhalation doses.

Over the period 1990-2000, the mean radon concentration for the total housing stock was 22.7 Bq/m^3 (2000: 23.2; 1990: 22.2 Bq/m^3). This concentration corresponds to an inhalation dose of radon decay products of 700 microsievert. For 1970-1980, the mean dose was 19.6 Bq/m^3 (1979: 20.3; 1969: 18.8 Bq/m^3). Indoor radon concentrations are increasing over time (Figure 7).

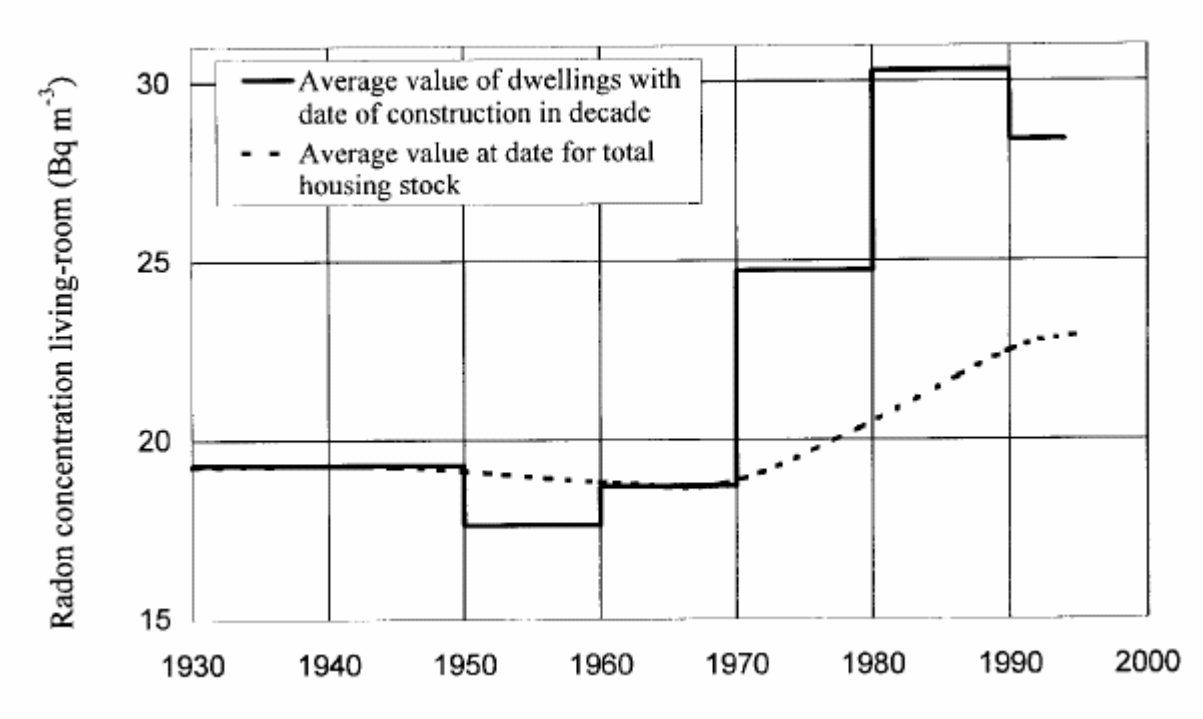


Figure 7 Average radon concentrations in Dutch houses and mean values per decade (Stoop et al., 1998).

Prevalence

Prevalence data for lung cancer are derived from mortality registration as used in the National Public Health Compass (RIVM, 2004a). These prevalences have been corrected for population size, but not for composition of the population (ageing).

Exposure-response

Calculations by the Health Council of the Netherlands (Gezondheidsraad, 2000) indicate 100 to 1200 extra cases of lung cancer (central estimate: 800 deaths) caused by indoor radon exposure. This is based on data for mine workers, assuming a linear relationship between radon and lung cancer. The effect of smoking has been incorporated using a multiplicative model. It has been assumed that the indoor radon concentration of 23 Bq/m³ leads to these 800 radon-related deaths, implying that a radon concentration of 1 Bq/m^3 roughly corresponds to a mortality of 2.2 people per million. Using the characteristics of these estimates, we have been able to calculate radon attributable lung cancer for 1980 and 2020.

Duration

By setting the radon-related deaths as a fixed fraction of the overall lung cancer deaths, we inherently imply that the age and gender specific incidence and disease development are identical to lung cancer cases in the general population. The durations used are based on standard mortality tables and vary between 11 and 17 years (dependent on year and gender).

Severity

Severity for mortality is 1.

4.4 UV

Ultraviolet (UV) radiation is responsible for several adverse health effects. Exposure to UV radiation can potentially lead to skin cancer, suppression of the immune system, cataracts and premature skin aging. We have calculated the UV-related disease burden based on skin cancer (basal cell carcinoma, squamous cell carcinoma and melanoma), morbidity and mortality. Together, these 3 types of skin cancer make up almost 100% of all skin cancer incidence (70%, 20% and 10% respectively). Mortality calculations for basal cell carcinoma have not been made, since hardly anybody dies due to that type of cancer. Changes in UV-exposure do not have an immediate effect on skin cancer risk, due to long latency periods

(Slaper, 1996; Kelfkens, 2002). Thus it is to be expected that the increase in skin cancer resulting from ozone depletion will hardly be visible during the period of this study (1980-2020)

Therefore, the estimated disease burden during this period is predominantly due to outdoor UV-exposures that are only marginally affected by ozone depletion. This disease burden is therefore only marginally open to environmental policy. It is, however, open to public health policy, since behavioral aspects dominate actual exposure.

Effects associated with exposure to artificial UV sources, e.g. the use of tanning equipment or for medical reasons, have not been estimated in our study.

Exposure and exposure-response

The large majority of skin cancer cases are thought to be attributable to UV radiation. However, induction and development of skin cancer is a complicated and long-term process. The cumulative UV-dose over 30-50 years before diagnosis is relevant for the induction of squamous cell carcinoma. For basal cell carcinoma exposure early in life is probably most important. For the induction of melanoma, additional information on the variation in UV exposure and skin burning history may be more relevant. Model calculations can in retrospect yield information on the ambient UV dose, but none of these models can incorporate the changing sunbathing behavior of the population with any sophistication. Consequently, a exposure-response relationship relating the ambient UV dose to skin cancer incidence, incorporating people's behavior, is currently not available. Therefore, the DALY calculations are based on the observed number of skin cancer cases in the year 2000. For the years 1980 and 2020, these numbers have been corrected for the size and age distribution of the population, but not for possible differences in exposure, for instance due to a change in behavior or in stratospheric ozone content, or for ethnicity and potential changes in the prevalence of certain skin types .

Prevalence

Prevalence data for cancer are derived from mortality registration as used in the National Public Health Compass (RIVM, 2004a), which are data from the Dutch Cancer registration. Since age specific prevalences for basal cell carcinoma were lacking, we have used the same age distribution as for squamous cell carcinoma, multiplied by 3.5 (based on data from the Dutch Cancer Society (KWF Kankerbestrijding, 2004)).

In order to assess the impact of our input choices, we have also calculated UV-related DALYs using incidence data. We have used age specific incidence data for mortality and morbidity. In order to estimate morbidity numbers, we have calculated the fraction of people that survive the disease each year (for basal cell carcinoma 100%, for squamous cell carcinoma 95-98%, for melanoma 77-86% over the years).

Duration

For the morbidity calculations based on prevalences, duration is standard one year. The average number of years of life lost due to premature mortality have been derived from the age specific incidence data. For squamous cell carcinoma, this number varies from around 5 to 9 years (depending on year and gender). Some 18 to 28 years of life are averagely lost due to melanoma-related mortality.
The alternative incidence-based morbidity calculations were made using the number of remaining life years after the occurrence of cancer as input for the duration. This duration is around 9 to13 years for basal and squamous cell carcinoma and 22 to 29 years for melanoma (depending on year and gender).

Severity

All severity factors have been derived from the sources mentioned in paragraph 3.3.

4.5 Indoor dampness

The main health effect of living in damp houses is (aggravation of) respiratory diseases, such as asthma (Van Veen *et al.*, 2001). We have analyzed the contribution of indoor dampness to asthma complaints for children and for adults.

Exposure

The percentage of houses with dampness problems has been estimated using various research and registrations (Van Veen *et al.*, 2001). Data were available for 1971, 1995, 2000 and 2020. The value for 1980 has been interpolated using the data for 1971 and 1995, as is shown in Figure 8. The percentages have been multiplied with population numbers and split into

children (< 15 years) and adults in order to estimate the number of exposed children and adults.

It is possible that the type of dampness, and consequently the concentration and type of mould associated with this dampness, has changed over the years. This can potentially affect our disease burden estimates; however, data to include these potential effects in the calculations was unavailable.

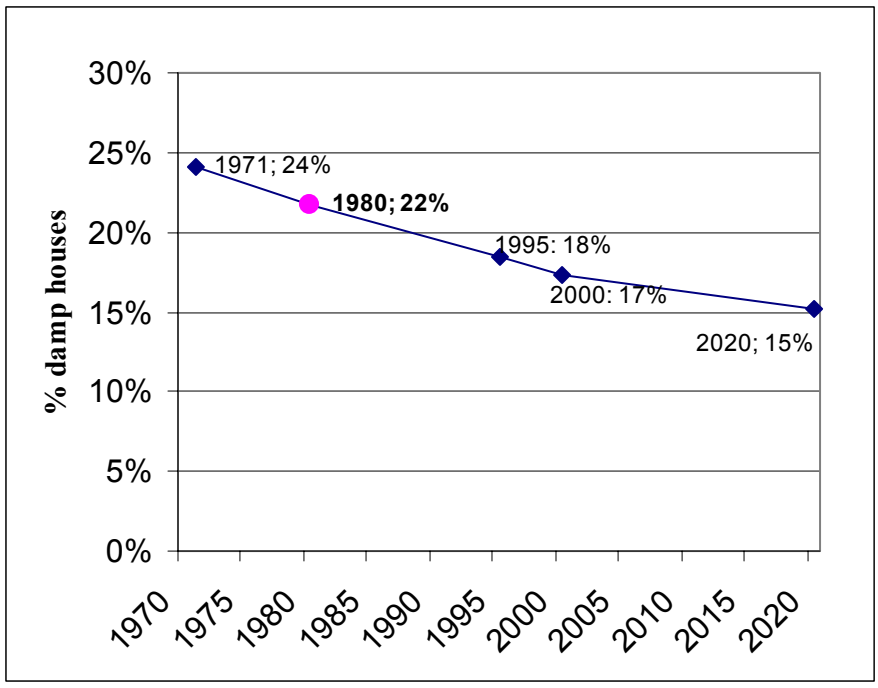


Figure 8 Percentage of damp houses in the Netherlands. The value for 1980 has been interpolated.

Prevalence, exposure-response, duration and severity

Prevalence of asthma is based on doctor registrations. Since these data were available per 5 year age category, it was possible to perform separate calculations for children and adults (RIVM, 2004a). As children are more vulnerable to asthma then adults, their relative risk is higher (1.5 - 3.5) than for adults (1.5 - 2) (Van Veen *et al.*, 2001). These estimates are based on a literature review and a dissertation using international studies.

Severity is similar for children and adults, at a severity weight of 0.078 per year with asthma (RIVM, 2004a). Duration of asthma is standard one year using prevalence rates.

4.6 Traffic accidents

Traffic accidents can cause mortality and (severe) morbidity. For calculations on traffic accidents, there is no need to model the 'attributable risk': all traffic accidents are by definition related to traffic. Also, all people are in principle exposed to traffic. Therefore, data on exposure and exposure-response are not needed.

Prevalence

In the year 2000, around 1,200 people died and some 136,000 people were injured in traffic accidents (Stichting Wetenschappelijk Onderzoek Verkeersveiligheid (SWOV). Personal communication. Bos N, 2004). These numbers relate to all victims of traffic accidents in the Netherlands on public roads with at least one driving vehicle involved.

For the years 1980 and 2000, mortality numbers are based on registered numbers, which are raised (since there is around 7% underreporting) using court reports and death certificates to get the true numbers. The uncertainty margin is relatively small and mainly caused by definition issues: foreign people having an accident on Dutch roads are registered, while Dutch people having an accident on foreign roads are not. Therefore, registrations are not a 100% precise reflection of the Dutch population, but are a 100% reflection of the Dutch traffic situation.

For 2020, index numbers for mobility in 2020 (Bos and Nagelhout, 2001) and an estimation of risk development (number of victims per vehicle kilometre) are used to estimate mortality rates; uncertainty is around 20%. This is based on a constant transport system and composition of the traffic fleet.

Due to a lack of data (registries as well as weight factors), it was not possible to include all possible traffic accident-related disabilities. We have included people suffering long-term injury (longer than one year). In 1994, 6360 people are estimated to have such disability (Ruwaard and Kramers, 1997). Using this information combined with injury incidence numbers, prevalence of disability lasting longer than one year has been estimated for all years in the study (assuming a constant share of long-term injuries within the total number of injuries, a constant registration degree, constant transport system, etc). Due to estimation and scaling errors, varying registration degrees, etc, the uncertainty range of injury estimations is greater than for mortality (total range: 30% for injuries and 10% for mortality for the years 1980 and 2000. For 2020, this is 20% and 40% respectively) (Stichting Wetenschappelijk Onderzoek Verkeersveiligheid (SWOV). Personal communication. Bos N, 2004).

Traffic prevalence data have been corrected for population size, but not for composition of the population (ageing).

Duration

The injury calculations are based on prevalence, and duration is set to one year. Duration of mortality (loss of life expectancy) is based on the mean age of the victims. More men than women are involved in accidents (3:1 rate) and since life expectancy for men is generally lower than for women, this has been allowed for in the calculations. A source of uncertainty in these estimates is the higher number of older people within the 7% of non-registered victims.

Severity

The weight factor for traffic accidents with remaining health consequences after one year is set to 0.43, with a standard deviation of 0.15 (RIVM, 2004a).

5. Thematic results and discussion

We calculated the burden of disease in the Netherlands attributable to (exposure to) air pollution (PM_{10} and ozone), noise, UV, radon dampness in houses and traffic accidents as described in the previous chapters. This chapter describes the results for each of these environmental factors. All input and output numbers can be found in Appendix 1.

5.1 Air pollution

Air pollution is one of the main environmental health issues in Europe, and PM_{10} and ozone are assumed to be two of the main contributors to the air pollution-related burden of disease. PM_{10} and ozone show some seasonal correlation, but on a yearly basis, concentrations are rather uncorrelated. Most other outdoor air pollutants (e.g. NO_2, CO, total suspended particles, SO_2) are highly correlated with PM_{10}, and effects are difficult to separate. Therefore, PM_{10} can be regarded as an indicator for this complex ambient air pollution mixture.

Figure 9 and Figure 10 show the total disease burden in DALYs for long-term PM_{10} exposure and short-term PM_{10} and ozone exposure calculated for the Netherlands.

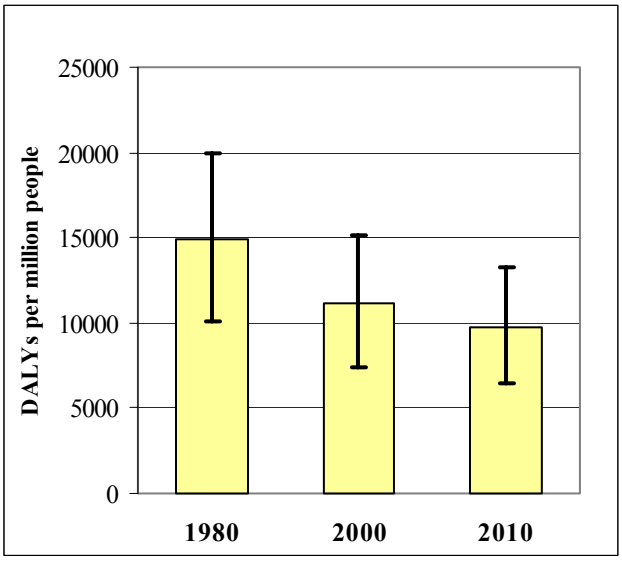


Figure 9 Burden of disease expressed in DALYs per million people caused by long-term exposure to PM_{10}, 1980 – 2010, Netherlands, with 90% prediction intervals. Estimates based on calculations using a conservative (10%) uncertainty interval for the number of years of life lost.

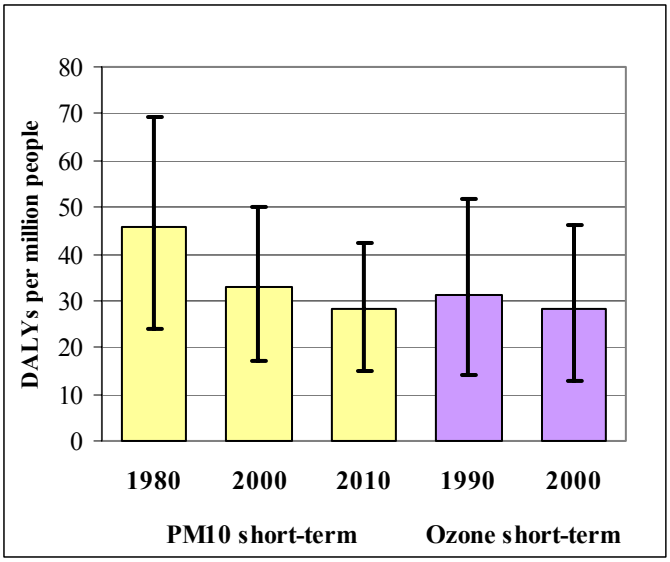


Figure 10 Burden of disease expressed in DALYs per million people caused by short-term exposure to PM_{10} and ozone, 1980 – 2010, Netherlands, with 90% prediction intervals.

In addition to contributing to cardiovascular and respiratory disease, it is estimated that short-term exposure to PM_{10} has lead to 1,200 to 2,200 deaths in the year 2000, whereas ozone may have caused 1,200 to 2,400 deaths in that same year (both PM_{10} and ozone calculations based on the individual component-models; calculations made with a 2 component-model are presented later in this paragraph). For long-term exposure to PM_{10}, the mortality number is many times higher (around 12,000 to 24,000), although, as mentioned earlier, the epidemiological evidence base for these effects is still limited. DALYs related to long-term PM_{10} exposure range from 7400 to 15000 per million people for the year 2000, using a conservative uncertainty interval (10%) for the number of years of life lost. This conservative estimate is based on the uncertainty in the estimation of life expectancy in general. Alternative calculations were made with an interval of 4 to 13 years, to illustrate the impact of a greater uncertainty interval around the duration estimate. There are currently no clear cut approaches to reliably estimate the uncertainty interval. The interval of 4 to 13 years is based on alternative assumptions about the age-categories affected by mortality attributed to PM10. The 4 year stems from an assumption that only older age-categories are affected, predominantly from cardiovascular mortality. The higher estimate of 13 years assumes that also younger age groups are affected. This wider uncertainty interval results in roughly 4,100 to 29,000 DALYs.

For the year 2000, the total number of PM_{10}-related DALYs decreased compared to the year 1980. With the current policy, this trend will probably continue from 2000 to 2020.

We have calculated the PM_{10}-related disease burden without a threshold value for PM_{10}. We have repeated these calculations using minimum levels of 10 μg/m^3 ('background concentration') and 20 μg/m^3 (EU target value for 2010). Table 4 shows the impact of using such reference levels. Since the average PM_{10} concentrations are decreasing over time, the relative impact of a reference level is greatest in the year 2010: a decrease in DALYs of around 31% for a 10 μg/m^3 reference level and around 65% for a 20 μg/m^3 reference level, compared to no reference levels).

Table 4 PM_{10}-related disease burden (DALYs per million people) using no reference level, a 10 μg/m3 reference level and a 20 μg/m3 reference level

DALYs per million people		**No reference level**	**Reference level: 10 μg/m^3**	**Reference level: 20 μg/m^3**
PM_{10} short-term exposure	1980	46 (24 – 71)	36 (19 - 59)	27 (14 – 43)
	2000	33 (18 – 51)	23 (13 – 36)	14 (7 – 23)
	2010	28 (15 – 44)	19 (9 – 29)	9 (4 – 15)
PM_{10} long-term exposure *	1980	14863 (10075 – 19934)	12225 (8077 – 16721)	9379 (5914 – 13183)
	2000	11163 (7437 – 15133)	8233 (5267 – 11493)	5057 (2862 – 7539)
	2010	9759 (6501 – 13270)	6712 (4332 – 9421)	3406 (1781 – 5351)

* DALY estimates based on calculations using a conservative (10%) uncertainty interval for the number of years of life lost.

In another alternative calculation, we have compared our short-term PM_{10} morbidity calculations based on Dutch exposure-response relationships (Vonk and Schouten, 2002) with calculations based on exposure-response estimates by Künzli (2000).

Table 5 shows the potential magnitude of the acute PM_{10}-related morbidity burden and also gives an indication of the potential variability of results caused by the choice of exposure-response relationships and health endpoints. As far as these results can be compared, using the international exposure-response relationships seems to generate significantly more DALYs than by using the Dutch ones.

Table 5 Morbidity effects relating to short-term exposure to PM_{10}, based on Dutch exposure-response relationships (Vonk et al., 2002) and based on international exposure-response relationships (Künzli et al., 2000)

	DALYs per million people for short-term PM_{10} exposure-related morbidity effects	
	Based on Dutch exposure-response relationships (Vonk *et al.*, 2002)	Based on international exposure-response relationships (Künzli et al 2000)
Mortality (total)	27 (13-44)	
Cardiovascular disease mortality	7 (2-12)	
Respiratory disease mortality	9 (4-15)	
COPD mortality	4 (2-6)	
Hospital admissions cardiovascular disease	5 (1-10)	18 (5-42)
Hospital admissions respiratory disease (total)	1 (0-2)	10 (3-22)
Hospital admissions COPD	1 (0-1)	
Hospital admissions asthma	*Not significant*	
Asthma attacks adults		166 (91- 249)
Asthma attacks children		90 (56-130)

For ozone, no significant trend is visible between 1990 and 2000 (Figure 10). Currently, it is difficult to make a reasonable estimation of future ozone levels. Although the number of days with elevated (peak) ozone levels is expected to decrease in the future (RIVM, 2004b), this trend is not necessarily as strong for the mean annual 8 hours concentrations.

Exposure to ozone would lead to an estimated number of around 1,800 deaths in the year 2000. Since the life-shortening effect of short-term ozone exposure is estimated to be limited to several months, the total ozone-related disease burden is around 450 DALYs. As mentioned in Chapter 3, there is currently little evidence for an independent long-term ozone effect on lung cancer or total mortality (World Health Organization, 2003).

It is still uncertain if there is a threshold value for ozone exposure beneath which no significant health effects occur. As for PM_{10}, such a threshold could greatly influence the calculated ozone-related DALYs. Currently, there is insufficient evidence to support a threshold value for ozone effects. However, WHO (UNECE, 2004) recommends to calculate

health impacts using a threshold value of 35ppb (35 ppb = 70 μg/m^3), thereby ignoring health effects that are potentially occurring below this level. In their calculations, a relative risk of 1.003 (1.001 - 1.004) for a 10 μg/m^3 increase in the daily maximum 8-hour mean was used, which is very similar to our relative risk of 1.041 (1.024-1.059) per 150 μg/m^3. However, WHO claims that 'it (is) highly likely that the overall effects of ozone (are) underestimated by this approach'. To apply the WHO approach, *daily* ozone data is needed in order to calculate the health impacts of peak value ozone concentrations (above 70 μg/m^3). For this study, we used modelled *yearly* average 8-hour concentrations in the Netherlands, which, as shown in Figure 3 are never above 70 μg/m3. Since (modeled) one hour ozone concentrations were not available for all years, we have not calculated ozone-related mortality based on these recent WHO assumptions within the context of this study.

We have calculated the effects of short-term peak exposure to PM_{10} and to ozone separately. Only for the year 2000, a so-called 2 component-model could be used, since the available years of data for PM_{10} and ozone did not overlap for the other years. This model is set up to produce more accurate results for multi-exposure assessments and is therefore likely to produce more correct outcomes. Calculations for the year 2000 resulted in a total estimation of around 5,000 air pollution-related deaths, of which 2,200 to 3,500 can roughly be attributed to PM_{10} and 1,600 to 3,100 to ozone. Using the 1 component-models, a total of some 3,500 air pollution deaths were estimated for that same year. The discrepancy between these two numbers is some 40% and should be considered while interpreting results. The difference is possibly caused by the fact that the covariance structure has not been taken into account in the individual calculations. Furthermore, a possible explanation for this discrepancy is the difference in the way the relative risks were calculated. For the individual models, *daily* concentrations have been related to the health effects of the day after the exposure (lagtime 1). For the 2 component-model, mortality numbers have been related to mean *weekly* concentrations. Several studies have shown that more days averages correlate better with mortality than single day exposures. This suggests that the effects of air pollution aggregate over air pollution levels of several days. Consequently, the 2 component-model, using weekly average values, will lead to higher mortality numbers than the estimates based on the individual models.

Overall, the effects of air pollution are considerable, and can affect the entire population, although some groups such as children and elderly are especially vulnerable. Our calculations

might underestimate the real burden of disease, since not all health responses could be included completely. For example, the long-term effects of PM_{10} are based on mortality numbers alone, while long-term PM_{10} exposure may also lead to increased morbidity (i.e. respiratory diseases). However, valid exposure-response relationships are currently unavailable (RIVM. Personal communication. Fischer P. 2005). As for the acute effects of PM_{10}, we could take into account more health effects, because more research has been done on this subject. However, we could only use hospital admission numbers, thereby not including morbidity effects of a less severe degree. The ozone-related burden of disease is only based on mortality numbers, since valid and positive exposure-response relationships for morbidity effects were not found in the latest Dutch study (Vonk and Schouten, 2002). This does not necessarily mean, however, that effects on morbidity do not occur. For example, days with elevated ozone concentrations have been linked to effects such as a decrease in lung function. An estimated 10 percent of children from 7 to12 years old have a decreased lung function on at least one day in summer, due to smog (RIVM, 1997). Due to the lack of a suitable weighting factor this effect has not been included in the DALY calculations.

On the whole, long-term effects of PM_{10} (as indicator for a complex urban mixture of air pollutants) are probably most hazardous and hardest to avoid. Several policy interventions are aiming to reduce air pollution and have been quite successful in the past. However, continued efforts are necessary, especially on an international scale (RIVM MNP, 2004). The European Union has set a target PM_{10} value of 20 $\mu g/m^3$ in 2010. Reducing PM_{10} levels to this level would significantly benefit health and reduce the health burden by 35% according to our calculations, assuming a proportional reduction in the causative fraction of the PM_{10} mixture. However, in the Netherlands it is highly unlikely this target will be reached, even at high costs (Beck *et al.*, 2003).

5.2 Noise

Exposure to noise, and consequent annoyance, sleep disturbance or even cardiovascular problems, is a significant problem in the crowded country of the Netherlands. Figure 11 shows the estimated noise-related disease burden, which has increased and will probably continue to increase: the burden will probably have risen by 20% in 2020 (compared to the year 2000). Although the majority of DALYs is attributable to annoyance and sleep disturbance, we have calculated that 110,000 to 270,000 people may have hypertension

which can be attributed to noise exposure in the year 2000. Hypertension can potentially lead to cardiovascular disease. We have estimated that around 600 people may have died due to noise induced cardiovascular diseases in the year 2000, accounting for around 400 DALYs per million people. Due to the exposure range of the relative risk, people exposed below 55 dB have not been included in these calculations. This cut-off point can significantly influence the results. As an illustration, Table 6 shows the outcomes of earlier calculations (Van Kempen *et al.*, 2001) investigating the effect of such a cut-off point on the estimated magnitude of some road traffic noise related effects. It might be useful follow-up research to repeat these calculations using more varying cut-off values (60 or 70 dB(A), for example). The same study (Van Kempen *et al.*, 2001) also showed that the investigated noise range and the composition of the population under investigation can affect these type of outcomes.

Table 6 The influence of a cut-off point on the number of myocard infarcts and ischemic heart disease cases, attributable to road traffic noise (Van Kempen et al., 2001)

	Cut-off point (dB(A))		
	50 dB(A)	**53 dB(A)**	**55 dB(A)**
Myocard infarct	1982 (0-51119)	1081 (0 – 2869)	469 (0 – 1299)
Ischemic heart diseases	2644 (1485 – 3764)	1478 (815 – 2143)	66 (357 – 993)

Policy interventions aiming to reduce noise exposure have been relatively effective in the past. Noise barriers, the introduction of porous road surfaces and speed limitations, along with more stringent test requirements for road traffic, have restricted the increase of noise-related DALYs for the 1980-2000 period. However, with current policy, the disease burden will increase in the upcoming years. Therefore, measures aiming to reduce noise exposure could be useful. Decreasing noise levels by around 5 dB for every source by 2020 could almost half the number of annoyance and sleep disturbance-related DALYs (Figure 12). This reduction could potentially be achieved by implementation of the use of silent tires, sound absorbing pavements and speed limitations (at highways, provincial- and municipal roads); and more silent break systems and rail construction for rail traffic. Since road traffic has the biggest share in the traffic-related disease burden, this would be an important source to tackle.

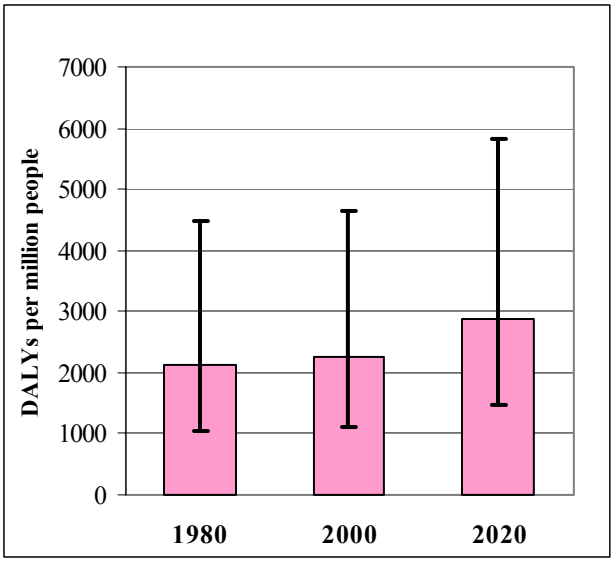


Figure 11 Burden of disease in DALYs per million people caused by exposure to noise, 1980 – 2020, with 90% prediction intervals.

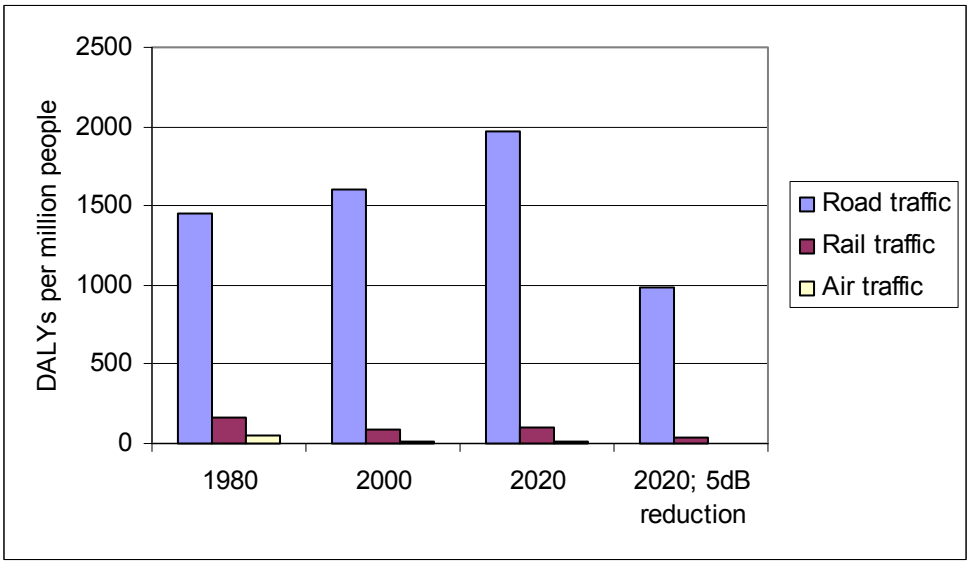


Figure 12 Burden of disease in DALYs per million people caused by severe annoyance or severe sleep disturbance due to road, train and air traffic noise, for 1980, 2000 and 2020, including an alternative scenario for 2020 (with a 5dB noise exposure reduction for road and rail traffic).

Alternatively, we have calculated noise-related annoyance and noise-related sleep disturbance using prevalence estimates from a recent survey on environment-related annoyance and quality of life (Franssen *et al.*, 2004). These results vary greatly from the results based on the exposure-response relationships derived by Miedema *et al.* (2001).

Table 7 DALYs for severe noise annoyance and severe sleep disturbance, based on exposure-response curves (Miedema,2001) and based on a survey (Franssen et al., 2004)

		DALYs per million people	
		Miedema exposure-response curves (Miedema, 2001), year : 2000	Environment-related annoyance and quality of life survey (Franssen 2004), year: 2003
Severe annoyance	Road	1,122 (441 – 2,753)	7,604 (3,119 – 18,387)
	Air	16 (6 – 38)	314 (129 – 761)
	Rail	65 (24 – 158)	524 (215 – 1,268)
Severe sleep disturbance	Road	526 (189 – 1291)	3045 (1,298 – 7,029)
	Air	-	761 (324 – 1,757)
	Rail	32 (10 – 80)	253 (108 – 586)

The variability of the outcomes is shown in Table 7. Even though basic assumptions and principles differ (for example, the noise maps are mainly based on car traffic, while the survey concludes that moped noise is the biggest contributor to the annoyance measured in their study), the difference in outcomes remains considerable. Most outcomes differ by a factor of around 8, with the exception of severe annoyance from air traffic noise, where the survey results are 19 times higher than the ones based on the EU recommended curves by Miedema, Statistics Netherlands (CBS) reports annoyance in a different way than the previously described studies. As part of a permanent investigation of the quality of life in the Dutch population (Centraal Bureau voor de Statistiek, 2005), they report for the year 2001/2002 that 6.4% (+/- 0.2) of the population is annoyed (*not necessarily severely and based on different survey questions*) by road traffic noise, which corresponds to some 64,000 people per million.

The large fluctuations in annoyance estimates can mainly be ascribed to disparities in methodologies and definitions. As the questions and aim of the Statistics Netherlands research differs from the other studies, results are incomparable.

In the survey by Franssen *et al.* (2004), effects were addressed per traffic source and in a representative sample of the population, whereas the EMPARA model only gives estimations of the total transportation-related noise exposure for the whole population. Furthermore, the applicability of the studies underlying the estimation of the exposure-response relationship by Miedema *et al.* (2001) can be debated: some seem rather outdated or were executed in a very different context (Van Kempen, 2005). Other factors that potentially influence the validity of our results include incorrect exposure measurements, or the fact that the (recent) surveys are

held at places where there is increased sensitivity to noise or an ongoing political debate, which influences peoples emotions and thereby perhaps the annoyance reporting frequency. As explained earlier, we have used the results based on the exposure-response relationships by Miedema in our final DALY figures.

As for air traffic-related annoyance, our results probably underestimate the real number of people being annoyed, since recent annoyance measurements around Schiphol airport (Breugelmans *et al.*, 2005) indicate a higher number of severely annoyed people (13%) than based on the Miedema relations. Results from former studies around Schiphol airport are not included in the Miedema relations, since the situation around Schiphol airport was not judged sufficiently stable (Ministerie van Verkeer en Waterstaat, 2000). In future studies, the recent exposure-response relations for annoyance and sleep disturbance based on data gathered around Schiphol (Breugelmans *et al.*, 2005) might be more suitable for estimating the air traffic noise-related disease burden in the Netherlands.

It is uncertain whether our outcomes are more likely to under- or overestimate the real noise-related burden of disease. Effects such as cognition could not be included. Furthermore, air traffic results are incomplete due to the fact that we didn't assess air traffic-related sleep disturbance and the relationships we used for noise annoyance (Miedema, 2001) might not be completely applicable to the Dutch situation. Also, air traffic-related annoyance is only based on exposure data from the area around Schiphol (main airport). These shortcomings point to potential underestimation of the noise-related disease burden. This corresponds with the fact that the DALY calculations based on the survey data are significantly greater than our estimates based on the curves by Miedema. However, since the magnitude of the effect of noise on cardiovascular diseases is still being debated, and since the qualification of annoyance and sleep disturbance as health effects can be contested, our outcomes could also be overestimates.

With regard to the estimated trends, the increasing number of people being annoyed and sleep disturbed can obviously be attributed to the rising noise levels. However, another aspect, not included in this study, could also affect the number of people being annoyed. Guski (2004) has suggested there is a possible trend in the number of people that experience annoyance at a certain noise level. This assumption is based on preliminary research, in which a constant percentage of 25% of respondents being highly annoyed by air traffic noise was found at

lower day/night noise levels over a time period of around 30 years. This would imply that people are getting annoyed more quickly. If this is an actual trend, this would mean for our study that more people than we have estimated might be annoyed in the future.

5.3 Radon

It is estimated that around 800 people died from radon induced lung cancer in the year 2000. This number is expected to increase because the average indoor radon concentration is still increasing as a result of better isolation, often combined with less ventilation (RIVM, 2004a). In 2004, a policy has been developed to achieve a stand-still situation of indoor radon concentrations in new dwellings. Our prognosis for the year 2020 is based on the assumption that this policy will be effective and that the radon level in new houses will remain at the level of the year 2000. But despite this policy effort focused on new dwellings, indoor radon concentrations for an average Dutch dwelling will continue to rise during the coming decades as a result of the replacement of old dwellings (with low indoor radon concentrations) with new dwellings (with relatively high indoor radon levels).

Females generally lose more healthy life years due to lung cancer than males do (in the year 2000, the lung cancer induced loss of life expectancy was 11.4 and 14.3 years for males and females respectively). However, this increase may partly be an artefact, since it is attributable to the assumption that radon-related deaths have the same age and gender specific incidence as lung cancer deaths in the general population, which might not necessarily be true, particularly since the exposure-response relationships have been derived from studies among mine workers, a predominantly male group.

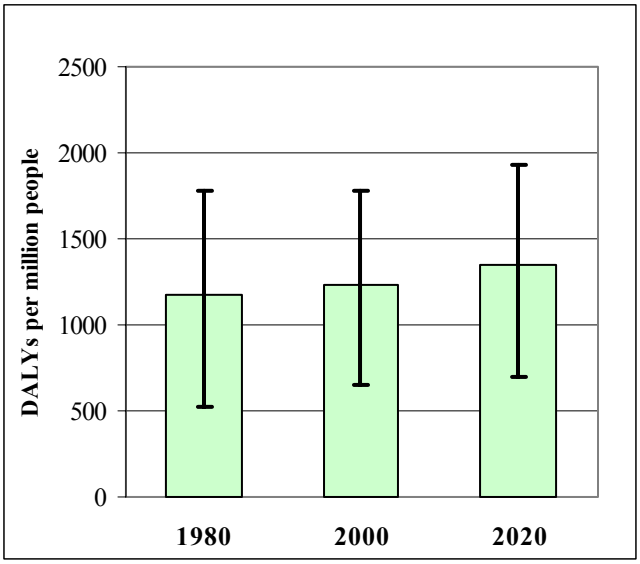


Figure 13 DALYs per 1.000.000 people for lung cancer mortality due to radon exposure, 1980 – 2020, with 90% prediction intervals.

The radon-related disease burden can be significant and may increase over the coming decades. Radon occurs naturally and the policy to build low-energy dwellings may lead to insufficient ventilation. Although the building of energy-efficient dwellings with adequate ventilation is possible, this involves extra costs. Finally, the behaviour of the occupant also affects the radon exposure. A covenant with the construction sector and a national call for adequate ventilation may reduce or reverse the increase.

5.4 UV

In the year 2000, around 130.000 people had a diagnosed form of some type of skin cancer in the Netherlands. Practically all of these cases are attributable to UV exposure. Since data about changing UV intensity or sunbathing behavior were not included in the calculations, mainly because of lack of information on sunbathing behavior and the latency time, this number is the same for 1980 and 2020 when imposed on the population of the year 2000. Based on our calculations it is not possible to make statements about trends in UV-related burden of disease that relate to actual changes in exposure.

Looking at trends in UV doses, the average dose in 2002 was 7% higher than in 1980, mainly due to degradation of the ozone layer ((RIVM MNP and CBS and Stichting DLO, 2003b). In time, this will probably lead to additional cases of skin cancer (Kelfkens, 2002). The increase in UV levels in the 90's are expected to lead to 2,000 to 3,000 additional skin cancer cases, of

which 40-50 are estimated to die from the skin cancer. When current policy measures are implemented and maintained, the ozone layer is expected to slowly recover. In that case, the extra number of skin cancer cases will probably be reduced to 1,500 to 2000 by the middle of the century (Pruppers *et al.*, 2001). These data, however, were not usable for DALY calculations, since effects are estimated to mainly take place beyond the time frame of our study.

Although we did not include potential trends in UV exposure in our calculations, we could investigate the effects of changes in population composition (age en gender) by not using the standard year 2000 population (as has been done for all other calculations) but instead using the actual population numbers for 1980 and 2020. Figure 14 shows the UV-related disease burden per million people, including effects of changes in population composition (age and gender), which leads to a significant increase in the number of DALYs per million people.

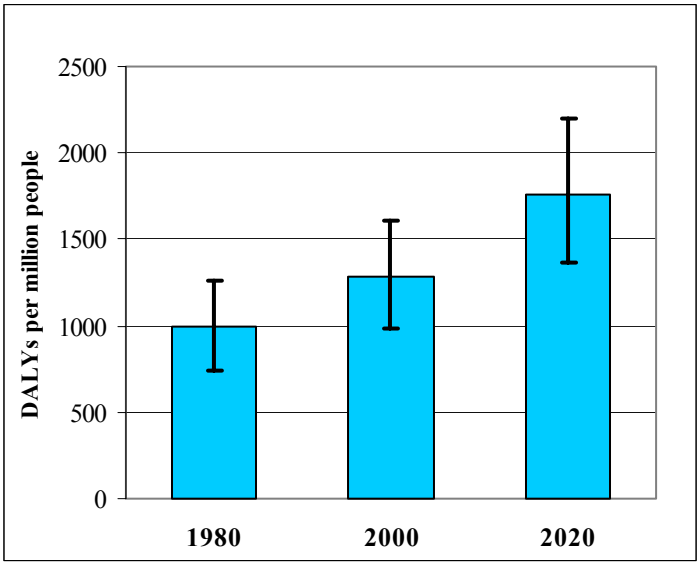


Figure 14 DALYs per 1.000.000 people for skin cancer mortality and morbidity due to UV exposure, 1980 – 2020, including demographic changes (not corrected for population composition), with 90% prediction intervals.

The burden of disease related to UV exposure lies somewhere between 900 and 1,600 DALYs per million people (year 2000). This could be an underestimation, since incidence data from the Dutch Cancer Society (KWF Kankerbestrijding, 2004) lie higher than incidence data from the Dutch Cancer Registry (the source of our prevalence data) (Table 8). This could lead to potential underestimation of 13 to 37%.

Table 8 Skin cancer incidence data from the Dutch Cancer Society and the Dutch Cancer Registry, year 2000

	Incidence	
	Dutch Cancer Society	Dutch Cancer Registry
Basal cell carcinoma	17,500	11,000*
Squamous cell carcinoma	5,000	3,168
Melanoma	2,500	2,168
Total	25,000	16,336

* Estimated by multiplying squamous cell carcinoma incidence with a factor 3.5 (factor based on data from the Dutch Cancer Society).

Additionally, we have made the DALY calculations using incidence instead of prevalence data (Table 9). DALY estimates based on incidence lie 29 – 39% higher than those based on prevalence data. This difference is caused by the fact that in the incidence based calculations, all incident cases that survive the year under study are considered skin cancer cases for the rest of their lives. Since some people will cure, this duration will be an overestimation, thereby overestimating the DALY outcomes.

Table 9 UV-related DALYs per million people, uncorrected for population composition (ageing), using prevalence and incidence data.

	DALYs per million people, uncorrected for population composition	
Based on:	**Prevalence**	**Incidence**
1980	998 (557 – 1,492)	1,385 (980 – 1,841)
2000	1,289 (757 – 1882)	1,728 (1,255 – 2,248)
2020	1,762 (1,054 – 2,550)	2,267 (1,686 – 2,951)

Even though it is difficult to calculate UV-related disease burden and trends are hard to assess, the continuing degradation of the ozone layer, potentially combined with more careless sunbathing behaviour and more frequent travels to exotic countries, will probably keep skin cancer on the public health agenda for the upcoming decades.

5.5 Indoor dampness

In the year 2000, 120 DALYs per million people were estimated due to indoor dampness (Figure 15). Although relatively similar numbers of children (<15 years old) and adults have

asthma, the fraction of cases that is potentially related to dampness is higher for children, so children contribute more to the total disease burden (see Appendix 1).

The indoor dampness-related disease burden (74-214 DALYs per million people in the year 2000) is expected to decrease in the future. However, in the long run, this trend might change due to increasing isolation influencing natural ventilation, but this trend is not yet visible (Van Veen *et al.*, 2001). Also, background asthma incidence is increasing, which, provided exposure-response relationships are constant, would lead to higher estimates of indoor dampness-related effects.

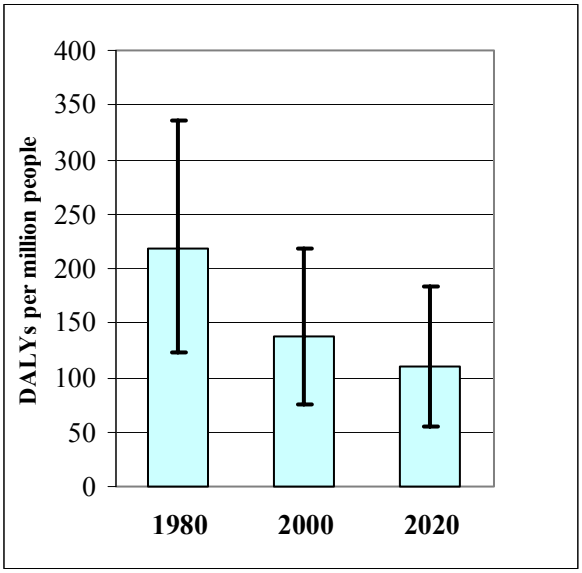


Figure 15 DALYs per 1.000.000 people for indoor dampness-related asthma, with 90% prediction intervals.

5.6 Traffic accidents

Traffic accidents cause a significant part of the burden of disease as calculated in this study. Even though traffic accidents might not be considered environmental, they are nonetheless useful to put the other environmental factors in perspective.

The traffic accident-related disease has declined in the past and will probably continue to decline in the future (8,000 DALYs in 1980 to less than half of that in 2020, Figure 16) because the decrease in risk (safer cars) will probably exceed the increase in mobility. This is based on historical trends and can partly be explained by the fact that the greatest increase in mobility will take place on the safest roads.

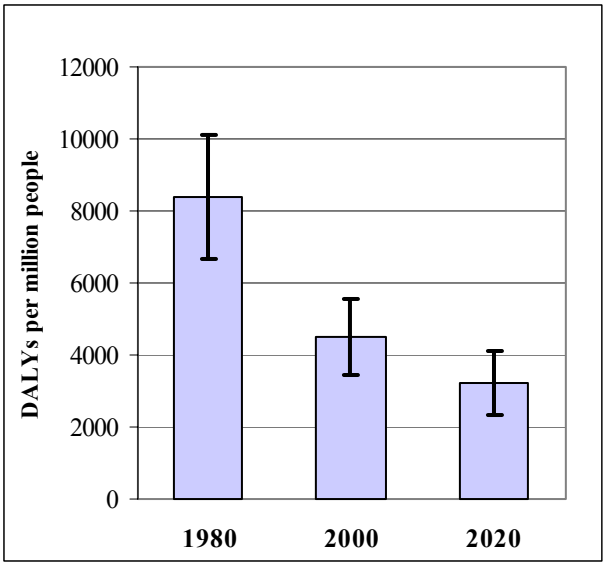


Figure 16 DALYs per 1.000.000 people for mortality and severe injuries (health consequences > one year) caused by traffic accidents, 1980 - 2020, with 90% prediction intervals.

The traffic accident-related disease burden is relatively high due to the fact that, although only a relatively small number of people are involved in traffic accidents, their average age is considerably lower than i.e. the age of people dying from a cardiovascular disease. Therefore, more years of healthy life are lost in traffic accidents, contributing to a greater disease burden. For example, in the year 2000, 73 people per million inhabitants died due to a traffic accident, which adds up to almost 2,700 DALYs per million for mortality alone. Including injuries, total disease burden equals almost 5,000 DALYs per million people.

The outcomes will probably be underestimates, since injury has only been included when persisting after one year. In the year 1994, 6,360 people were assumed to have such long lasting injury (*prevalence*). However, the total *incidence* of traffic injuries in that year was around 120,000 (Stichting Wetenschappelijk Onderzoek Verkeersveiligheid (SWOV). Personal communication. Bos N, 2004). Therefore, many injuries with less severe consequences could not be included due to a lack of appropriate data.

6. Overall results and discussion

6.1 Implications of the results

We have estimated the disease burden attributable to exposure to some environmental factors. In previous sections, results have been discussed per environmental factor. However, one of the reasons to calculate DALYs is to enable comparisons between these different factors. When we do so, it becomes clear that among the investigated factors, the (relatively uncertain) effects of long-term PM_{10} exposure currently generate most DALYs followed by traffic accidents, noise, UV and radon (Figure 17). The uncertainty interval around the long-term PM_{10}-related disease burden as presented in the figure is probably rather conservative, since we have used a standard 10% uncertainty interval for the number of years of life lost (10 years +/- 10%), which is relatively small given the overall uncertainty of the health effects of long-term PM_{10}. Using a greater and potentially more realistic uncertainty interval of 4 to 13 years results in a disease burden ranging from 4,100 to 29,000 DALYs. This alternative interval is based on varying assumptions regarding the age categories (whole population or only elderly people) that are affected by long-term PM_{10}-related mortality. However, these calculations are merely an illustration, as it is currently unclear what the uncertainty interval should look like.

The substantial impact of noise is due to the large number of people exposed to noise. Relatively small effects (not shown in Figure 17 because of the scale of the y-axis) can also be attributed to the short-term effects of PM_{10} and ozone exposure and to indoor dampness exposure (Figure 18). Because of the great variety in outcomes, results could not be displayed meaningfully in one graph using standard linear scaling. A graph with a logarithmic scale displaying all results together is presented in Appendix 2.

Trends

The number of DALYs related to noise and traffic accidents are likely to become more similar in the year 2020, due to an increasing disease burden for noise and a decreasing disease burden for traffic accidents. DALYs related to long-term exposure to PM_{10} are many times higher than DALYs related to short-term PM_{10} exposure (acute effects). Both are slowly decreasing over time because PM_{10} levels are expected to decrease. DALYs related to radon exposure slowly increase over time, because the overall level of radon and its decay products is expected to increase.

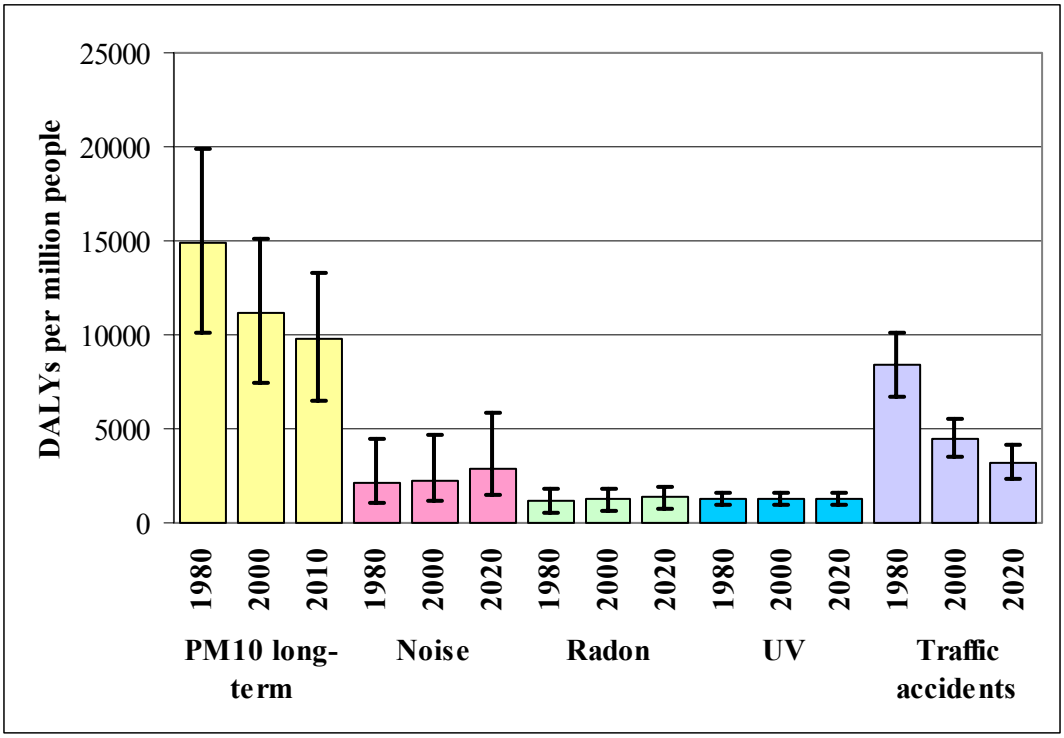


Figure 17 Estimation of the environment-related disease burden (in DALYs per million people) for the period 1980 – 2020: PM_{10} (long-term exposure; using a conservative (10%) uncertainty interval), noise, radon, UV and traffic accidents, with 90% prediction intervals. DALYs are corrected for population size and composition (standard population 2000).

PM_{10}, long-term based on mortality, using a conservative (10%) uncertainty interval for the number of years of life lost.
Noise based on severe annoyance (road/rail/air traffic), severe sleep disturbance (road/rail) and mortality due to hypertension (for all traffic sources)
Radon based on lung cancer mortality
UV based on skin cancer (morbidity and mortality)
Traffic accidents based on mortality and disability (>one year)

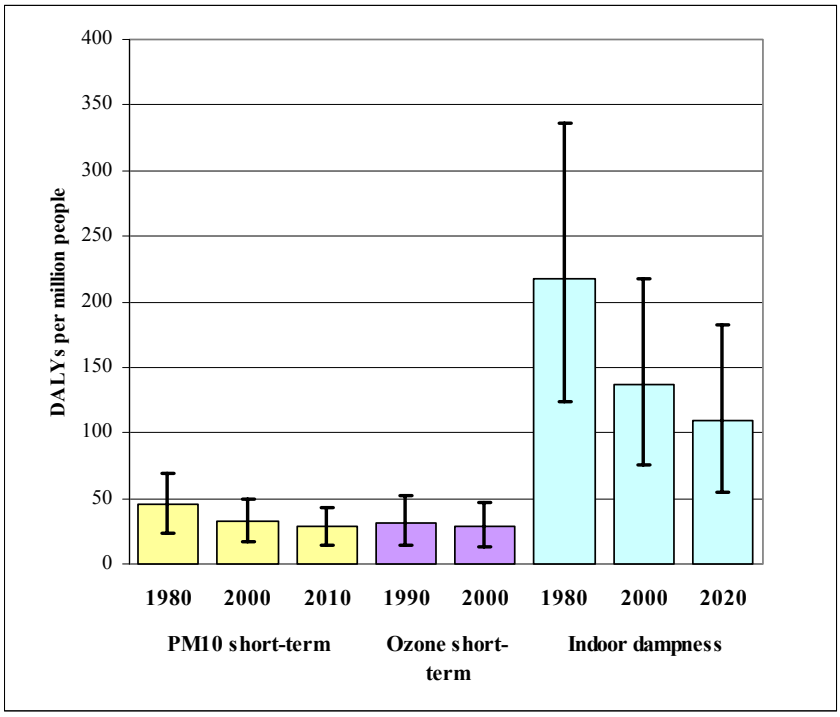


Figure 18 Estimation of the environment-related disease burden (in DALYs per million people) for the period 1980 – 2020: PM_{10} (short-term effects), ozone (short-term effects) and dampness in houses, with 90% prediction intervals. DALYs are corrected for population size and composition (standard population 2000)

PM_{10}, short-term effects based on mortality and hospital admissions for respiratory and cardiovascular diseases
Ozone, short-term effects based on mortality and 3-year mean annual 8 hour ozone concentrations.
Dampness in houses based on (aggravation of) asthma

Environmental disease burden

The 'total' disease burden in the Netherlands in the year 2000 is estimated to be at least 2.9 million DALYs (RIVM, 2004a). This estimate is based on data for 53 diseases that represent around half of the doctors diagnoses, 70% of mortality and 65% of the health care costs. For 7 of these diseases, no epidemiological evidence was available to calculate the disease burden (Hoeymans and Poos, 2002). Some other diseases have been split up in different subsets. In total, 49 (groups of) diseases are included in the calculation of this 'total' disease burden. Therefore, 2.9 million DALYs will be a significant underestimation of the total disease burden (based on the coverage of the estimate, the total might be over 4 million DALYs).

If this minimal number is regarded as the total disease burden, the health impacts of air pollution, noise, radon, total UV and damp houses contribute to around 6 to 13% (assuming no thresholds for PM_{10} and ozone effects) of this 'total' disease burden in the Netherlands in

the year 2000. If the total disease burden is over 4 million DALYs, a similar calculation results in an environmental impact of 4 to 9% (again, assuming no threshold for PM_{10} and ozone effects). The effects of long-term exposure to PM_{10} contribute to almost 70% of the total environment-related disease burden. However, the evidence base for long-term PM_{10} effects is very limited to date (see paragraph 4.1) and this percentage is based on calculations made without reference levels. Assuming a 20 $\mu g/m^3$ reference level results in a 50% contribution of long-term PM_{10} to the environment-related disease burden, which then accounts for 3 to 9% of the total disease burden. When excluding the effects of long-term PM_{10} exposure – and therefore only including those environmental health relations for which scientific consensus is judged reasonable to good – the environment-related percentage of the 2.9 million DALYs is 2 – 5 %. Percentages are rough estimates that should be interpreted with caution.

The calculations above are based on the total effect each environmental factor has on public health and not only the human-induced portion of the effects. For instance, UV radiation is only for a small fraction human-induced (due to ozone layer degradation), but mainly occurs naturally. The most noticeable effects of ozone layer degradation on UV induced skin cancer prevalence will start to occur in 2030 to 2050 (RIVM. Personal communication. Kelfkens G. 2005), so UV-related disease burden to human activities is minimal in the timeframe of our study. PM_{10} also occurs naturally, although the greatest part is human induced, especially in urban areas. Noise is (almost) completely due to human activities. Housing conditions, which affect radon concentrations and indoor dampness, are partly structural and partly influenced by human behaviour. On most of these issues, consensus on the anthropogenic part is lacking, and therefore it is difficult to estimate the total effect of human activities on the environmental disease burden.

One of the main issues is that we have only included a limited set of environmental factors in our study (based on the priority areas of the MNP and availability of data). For example, we did not include the health impacts of certain chemicals (i.e. lead, cadmium, benzene, PAH, dioxins; the effects of which are probably very small), or potential annoyance caused by odours. Furthermore, for the effects of housing, we only investigated the potential effects of indoor dampness on asthma, thereby not including other housing factors, such as dust, dust mites, and environmental tobacco smoke. Other calculations (Ruwaard and Kramers, 1997) have estimated for the year 2000 that around 26,000 DALYs can be attributable to *total*

indoor environment-related asthma, which corresponds to more than 1,600 DALYs per million people. This might, however, be an overestimation, since 70% of all asthma cases are presumed to be housing-related, which seems a rather large percentage given the genetic and other possible causes of asthma. As for environmental tobacco smoke (ETS), an important indoor air pollutant, it is estimated by the Health Council (Gezondheidsraad, 2003) that passive smoking may annually lead to hundreds of lung cancer deaths, thousands of cardiovascular deaths, around ten cases of sudden infant death syndrome and several tens of thousands of cases of respiratory tract disorders in children.

6.2 Uncertainty

In our results, uncertainty can be caused by the (lack of) scientific consensus on the causal relationship between the environmental factor and the health effect; the choice of studies and numbers used in the calculations; and the uncertainty within these studies and numbers.

Scientific consensus

Within the complicated and only partly understood context of environmental pollution, and the equally complex matter of health and diseases, it is difficult to be certain about potential environment and health relationships. Epidemiological and toxicological research aims to elucidate these potential linkages. However, often evidence is insufficient, inconclusive or very diverse. Sometimes scientific consensus is not reached, in which case we have used expert judgements in order to decide whether a relationship is plausible enough and which exposure-response relationships would be best to use. We have based our calculations on authoritative consensus documents where possible (e.g. WHO, Dutch Health Council, peer reviewed analyses).

Of the investigated factors, the health effects due to long-term exposure to PM_{10} are probably most controversial. Although results of most studies point into the same direction (paragraph 5.1), the knowledge base for these effects is still relatively limited and relationships are based on American studies, which might not be completely transferable to the Dutch situation (Buringh and Opperhuizen, 2002). Also, the estimate of the number of years of life lost due to long-term PM_{10}-related mortality is not sufficiently clear yet. In our calculations, we have used an estimate of 10 years of life lost (Künzli *et al.*, 2001; WHO, 2005; AEA Technology, 2005), using a conservative uncertainty interval based on general uncertainty in life

expectancy tables. However, it is as yet unclear whether these estimations of (uncertainty around) the years of life lost are completely applicable to long-term PM_{10}-related mortality. Therefore, uncertainty is potentially greater, which is why we have made alternative calculations with a greater uncertainty interval around the duration estimate (4 to 13 years). This interval is based on alternative assumptions about the age-categories affected by mortality attributed to PM_{10}: only older age-categories or younger age groups as well. These alternative calculations indicate the great influence of assumptions on DALY output, and the necessity to interpret results with caution.

The effects of short-term exposure to PM_{10}, on the other hand, have been studied in numerous research projects (Buringh and Opperhuizen, 2002). Although one single best exposure-response relationships for short-term PM_{10} exposure for all countries and contexts does not exist, causality of the relationship may be assumed, based amongst other things on the Bradford Hill criteria for causality.

Short-term exposure to ozone has also been related to various health effects (Buringh and Opperhuizen, 2002). The fact that ozone exposure is associated with mortality implies that there probably also will be a relation between ozone exposure and some form of morbidity, assuming that people will be sick before they die. However, most recent Dutch studies do not yield significant relationships for morbidity effects of ozone exposure (Vonk and Schouten, 2002) so scientific evidence is inconclusive. Also, evidence for effects of long-term exposure to ozone is ambiguous (World Health Organization, 2003).

For noise, virtually no one denies people can be annoyed by noise. However, the opinions on whether annoyance or sleep disturbance can actually be considered health effects vary (De Hollander, 2004). Clinical effects, such as effects on the cardiovascular system, are more generally accepted as health effects. However, these effects have been investigated by only a small number of epidemiological studies.

Although the effects of UV are not easy to quantify, there is no debate on the causality of UV exposure and skin cancer, so scientific consensus is considerable in that respect. For radon, the other form of radiation we have studied, scientists agree that a relation between radon and lung cancer, especially in combination with smoking, is highly likely (Gezondheidsraad, 2000). The existing relationships, however, are based on American studies with mine

workers, who were exposed to extremely high radon concentrations. These data have been (linearly) extrapolated in order to gain exposure-response relations for low radon concentrations. Whether this assumption of linearity is valid, however, is not yet completely certain (Gezondheidsraad, 2000).

Finally, for indoor dampness scientific consensus exists for the causality with (aggrevation of) asthma; a relationship which has been shown in numerous studies (Van Veen *et al.*, 2001), however, robust exposure- response relationships have not been derived yet.

The disease burden that can be related to environmental factors for which the consensus on causality is at least reasonable (short-term PM_{10} and ozone exposure, noise annoyance and sleep disturbance, UV, radon and indoor dampness) is estimated to lie between 2 and 5 % (based on a 'total' disease burden of 2.9 million DALYs). When noise-related annoyance and sleep disturbance are not considered 'health effects', the contribution to the disease burden of the remaining environmental factors lies between 1 and 2 %.

Choices and assumptions

When starting a DALY exercise, many choices have to be made. Which environmental factors shall we include? Which health effects should be considered? Which data should be used: Prevalence or incidence? National or international exposure-response relationships? Modelled or measured concentrations? In this report, we have described our decisions regarding such choices. We have tried to maintain consistency across topics, but this could not always be achieved due to e.g. lack of relevant data.

We analysed the effects of such decisions on the DALY output by doing some alternative calculations. For example, using incidence instead of prevalence data for the UV calculations changed the DALY output by 29 to 39%. Using a different, international exposure-response relationship for the DALY calculation of some of the effects of short-term PM_{10} exposure resulted in up to 10 times higher output estimates for the short-term PM_{10} DALY's. Similarly, the use of noise annoyance prevalence data based on measured values produced DALY outcomes that were factors higher than those based on modelled estimates. Using a

2 component-model for PM_{10} and ozone instead of 1 component-models gave different DALY outcomes and we have also seen that the choice of a reference value for noise and for PM_{10} exposure greatly influenced the results.

Data uncertainty

Once it has been decided which studies and data to use for the calculations, it is important to consider the uncertainty *within* these values. We have accounted for this uncertainty by carrying out a Monte Carlo analysis, thereby being able to show the potential range of our outcomes. The input data for such an analysis are not always directly available. Data such as exposure-response relationships or weight factors usually come with an estimated confidence internal. However, for other input data, such as prevalences or duration (years of life lost), these type of intervals or not always available and sometimes need to be estimated by experts. Furthermore, once uncertainty ranges are clear, the form of the probability curve should fit with the conditions of the variable, which also has to be assessed. The uncertainty ranges given therefore only give a general indication of the uncertainty in the underlying data.

The variations in the input data have an equal impact on the output, since DALY calculations essentially consist of a series of multiplications. The relative variation in some input numbers can be much greater than in others. For example, changing a weight factor from 0.01 to 0.02 (which can be done for noise annoyance) means a 100% change in the output, while changing it from 0.6 to 0.7 for a health endpoint with a more severe impact on quality of life, only causes a 16% change. In general, variations in relatively small numbers such as severity weights have a bigger impact on the output than variation in relatively large numbers such as most prevalence and concentration data. Therefore, in order to decrease the variation in DALY outcomes, research should focus on reducing uncertainty in input data that have relatively large variation. Paragraph 6.6 will further comment on recommendations for future research, by which the limitations of DALYs can be reduced.

6.3 Other burden of disease studies

Several global as well as national and more regional burden of disease studies have been undertaken in recent years, some of which focused specifically on the potential environmental impact on this burden (Prüss *et al.*, 2001). Environmental health impact

assessment can be addressed in different ways, which can generally be divided into two approaches: exposure-based and outcome based (Prüss *et al.*, 2001). The exposure-based approach, which was used in our study, uses population exposure data and exposure-response relationships in order to estimate the number of disease cases attributable to certain environmental factors. In contrast, the outcome-based approach uses the disease burden as a starting point and estimates the fraction potentially attributable to the environmental factor. The latter approach is more suitable for diseases that are predominantly related to one specific risk factor, while the exposure-based approach can be used to assess broad, large scale and non-specific environmental health relationships (Prüss *et al.*, 2001).

We have used the exposure-based approach and focused on the most prominent environmental health problems in the Netherlands, using the most recent relevant data available. Some of our results vary somewhat from earlier calculations in the Netherlands (De Hollander *et al.*, 1999), since we had a slightly different aim (trend analysis) and we made other assumptions, based on newer insights. In some cases (such as the effects of short-term PM_{10}), the difference in outcomes was due to the use of a different relative risk or different prevalence estimates (i.e. for hospital admissions for respiratory diseases). Other reasons include different exposure levels (i.e. ozone), different overall calculation (i.e. mortality due to noise exposure) or different estimations of the duration of the health effects. These differences underline the difficulties in consistently calculating DALYs and comparing results from different studies, and the need for generally approved methods and baseline data (such as exposure-response relationships).

The same sorts of discrepancies exist between studies from different nations. For example, Belgian estimates (Torfs, 2003) would be expected to match relatively well with the Dutch estimates, since both countries have a lot in common with regard to the main environmental health problems. However, comparing results leads to some differences, with Dutch PM_{10}-related disease burden being more than twice as high as the Belgian estimates (mainly due to a different set of health end points and part of the Belgian calculations being based on $PM_{2.5}$), and ozone-related disease burden being higher in Belgium (the latter caused by the fact that Belgium included morbidity effects related to ozone, where we only included mortality). As long as DALY calculations are not standardised, it is probably more useful to compare exposure data instead of DALYs when comparing countries (for only one pollutant).

These consistency problems are not restricted to relatively small scale national studies. The variation in the various *global* burden of disease estimates is also considerable. Where Smith *et al.* (1999) estimated 25-33% of the global burden of disease to be attributed to environmental risk factors using an outcome-based approach, Murray and Lopez (1999) concluded, based on the data they used for the Global Burden of Disease study, that some 16% of the total worldwide DALYs could be attributed to malnutrition, almost 7% to poor water and sanitation and 0.5% to air pollution. Prüss *et al.* (2001) estimated the contribution of water, sanitation and hygiene to the global burden of disease to be around 5.7%, while Briggs *et al.* (2003) found environmental and occupational pollution (water, sanitation and hygiene; outdoor air pollution; indoor smoke; lead; and occupational carcinogens, airborne particles and noise) responsible for 8-9% of the total disease burden, based on data from Ezzati *et al.* (2002). An outcome-based environmental health impact assessment by Melse and Kramers (1998) concluded that up to 11% of the global burden of disease could be environment-related (narrowing it down to 2-5% in OECD countries).

Since each study investigated different aspects of the environmental disease burden, results are incomparable. Most of the differences in the outcomes can be traced back to the fundamentals of the research: which definitions are being used for ‘environment’ and ‘health’, which assumptions underlie the calculations, which models and which baseline data have been used? The outcomes can therefore not be interpreted as isolated numbers, but only make sense in view of the context and underlying assumptions of the calculations. Comparisons of numbers from different studies should be made with great caution.

A current research project (RIVM, not yet published) aims to assess which factors specifically have led to the variations in these various studies. Results of this project will be very useful for discussion and interpretation of burden of disease studies.

6.4 The DALY debate

Since the introduction of the DALY to measure the burden of disease, there has been a lot of discussion on its advances and limitations. The main issues are described in the following paragraphs.

Ethics of severity weights

The very concept of attributing severity weights to health states has been criticised. Critics say that severity weights assume that life of a disabled person has less value than that of a healthy person. Taken to the extreme, this could theoretically lead to reasoning that disabled people are less entitled to scarce health resources that would extend their lives (such as preventive medicine) than healthy people (Arnesen and Nord, 1999).

Limitations of severity weights

Severity weights value a certain health state in relation to an ideal state of good health, with 0 being perfectly healthy, and 1 being dead. The weights, however, do not represent the experience of an individual in a certain health state or the ability of that person to cope with the disability (Anand and Hanson, 1997). Some diseases/disabilities can give a higher burden of disease to certain people than to others. For example, missing an arm will be worse to a farmer than to a college professor (Groce and Cheney, 1998). Allotey *et al.* (2003) described these contextual factors related to the burden of disease, such as culture, development, environment (urban/rural), gender and social-economic status. Their conclusion was that the experience of a health condition is an interaction between a person and their social, cultural and environmental context and the experience of the disease is greatly influenced by all of these factors. They proposed to include a 'development gradient' in the severity valuation that would lessen the severity as the development of a country improves. However, this would not cover all contextual factors and also, such severity weights do not currently exist.

Ways to value disability

Severity weights are used to value the time lived in a certain health state. In most studies, the various health states are valued by a team of experts. Experts may represent a skewed sample of the population (Arnesen and Nord, 1999), although the Dutch disability weights study (Stouthard *et al.*, 1997) did not find much difference between the average values assigned by a lay panel compared to those assigned by an expert panel. The Andhra Pradesh burden of disease study (Indian Institute of Health Systems, 2005) however did show that the general public rated severity weights for the same health state higher than experts. Also, people living in a particular health state tend to value severity weights for that state lower than other people (AbouZahr and Vaughan, 2000). Depending on the purpose of the study, input from those suffering from a certain health state, people with direct experience of the health state (family,

friends), health care providers, and the general public could be considered, next to the opinion of experts (Schneider, 2001). Further research is needed to find out what the best method is to value disability. It has been suggested (Indian Institute of Health Systems, 2005) to do repeated measures to help the valuers clarify their value sets. The realization of such a new or improved valuation set went beyond the scope of this study.

Age-weighting

When age-weighting is used in DALY calculations, different values are assigned to (healthy) time lived at different ages. This procedure has been justified by arguing that 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. Age-weighting is one of the most controversial aspects of DALY calculations. 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). Age-weights are not used in the calculations in this study, so a healthy life year is valued equal at all ages.

Discounting

In discounting, future years of healthy life lived are valued less than present years. Therefore, this is not favourable for children and future generations (Anand and Hanson, 1997; Arnesen and Nord, 1999). Preventive measures are devalued, as they cost money now while benefits will become apparent later (Schneider, 2001). For these reasons, no discounting has been done in this study. Other studies, such as the Australian burden of disease study (Mathers *et al.*, 1999), do use discounting. A recent Belgian burden of disease study (Torfs, 2003) has calculated results with and without 3% discounting. The discounting factor resulted in a 12% decrease of the total number of DALYs (not statistically significant).

Comorbidity

In industrialised countries, older people often have more than one disease. Severity weights do not take account of these comorbid 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).

The Global Burden of Disease study (Murray and Lopez, 1996) did not attempt to deal with the effects of comorbidities. In the Australian burden of disease study (Mathers *et al.*, 1999), adjustments were made for the effects of comorbidity between mental disorders and between physical disorders at older ages. A multiplicative model was used to estimate weights for comorbid conditions and the change in total weight was attributed back to the weight for the milder of the conditions. However, because of the complexity and the fact that there is currently no general approach, we have not accounted for co-morbidity in the current study.

Burden to society

DALYs only reflect the time of healthy life lost for patients themselves. However, society shares the burden of disease in supporting the patient in different ways (Pang *et al.*, 2000). An example of the burden to society is support provided through public services, family and friends, and private income. If the objective of DALY calculation is to measure the actual burden of disease, the burden to society should be included. However, it is currently impossible to quantitatively account for these effects in a coherent manner. These aspects should however be considered.

6.5 Policy relevance

As addressed in previous chapters, outcomes of various DALY studies vary, the method is being debated, and results are relatively uncertain. With all this ambiguity, one could question the usefulness of these types of calculations for policy makers, who generally want relatively certain information. One should realise, however, that a substantial part of the uncertainties is always involved in quantitative assessment of the impact of environmental pollutants, even when restricted to estimates of the number of people with a certain health effect, without aggregation into a DALY.

However, in spite of the various criticisms, the DALY has been adopted internationally and is increasingly used in various national and global burden of disease studies. We think that, within the proper context, results can give a useful -though crude- indication of the dimensions of environment-related health loss.

DALYs can also be used as summary measure to evaluate policy measures, instead of using multiple endpoints. Although more research is needed, our study has shown some examples of these types of evaluations. For instance, implementing policy measures to reduce PM_{10} levels to the EU target threshold value of 20 $\mu g/m^3$ in 2010 could reduce the PM_{10}-related disease burden. For noise, reducing exposure levels by 5dB in 2020 could reduce the disease burden by almost half (1800 DALYs, mainly attributable to noise from road traffic). For radon, policy could aim at a stand-still situation for radon levels (having them the same in 2020 as in 2000), which could prevent over 100 DALYs. By summarizing these effects in one integrated measure, the impact of different environmental factors can be compared. The policy relevance of these types of calculations, even though they are only rough estimates, is evident, since these numbers can help to focus policy on the aspects of the environment with the largest public health impact. Calculating source-specific DALYs (such as could, with certain assumptions, be attempted for i.e. traffic, industry or housing) can provide added value to policy makers, since policy is preferably aimed at the source.
For further usefulness, estimates need to be combined with cost-benefit analysis. This would also provide a better grip on the numbers, since it is difficult for most people to judge whether, for example, preventing 500 DALYs is a lot or a little.

Overall, if quantitative health impact assessment is useful, which it is in many (policy-related) circumstances, DALYs offer the most complete way of denominating health. However, DALYs should not be used in isolation, but in context with information about public engagement, acceptance of the risk by the exposed population, current policy and its effectiveness, special risk groups, etc. One way to consider these issues is by using the so-called Appraisal Framework Environment and Health (Van Bruggen and Fast, 2003), which has been set up to serve as a useful checklist, including the various issues that may play a role, separately or in unison, in policy decisions on health-related environmental problems.

As a supporting tool, DALYs certainly have added value as compared to underlying data such as concentrations, exposed population, etc, which can also be used as a basis for policy making. This is because DALYs integrate information on the number of people affected and the severity and duration of the disease, while also standardizing health effects, thereby allowing comparison between different (environmental) health problems. The usefulness of DALYs is a balance between these extra useful dimensions on the one hand and the extra uncertainty on the other hand. Uncertainty should be analyzed and described, in order to

make DALY results meaningful and interpretable. When using DALYs as guidelines instead of definite priorities, the approach can be very useful.

6.6 Methodological conclusions and recommended future research

Several burden of disease studies have been completed or are currently being carried out. These types of health impact assessments are useful for a (crude) comparison of the overall public health impact of different environmental factors and for assessing policy options. Therefore, as already mentioned in the introduction, it is useful to link general burden of disease studies to actual causes of disease (whether environmental or otherwise). This helps to identify main sources of (environmental) health loss, which can than be targeted with specific policy measures. It is therefore also helpful to try and identify a specific segment of the disease burden that can be attributed to a certain source, such as, for instance, the traffic- or housing-related disease burden. A further useful step is to calculate trends over time, as we have undertaken in this study, which allows evaluation of past policy measures and anticipation of the future. Generation of alternative (policy) scenarios can also help in this context. For example, in this study, we have experimented with alternative noise scenarios. The outcomes of our exercise show the possible health improvements of policy measures that reduce noise levels by 5 dB.

Furthermore, geographical trends can be informative, and can help evaluate regional policy efforts. There is an increasing request for integrated (environmental) health information on a more local scale. Research and discussion is needed on the applicability and validity of the DALY concept on such smaller (sub-national) scales.

DALYs can be expressed in terms of money, thereby being compatible for cost-benefit analysis, which is useful for policy makers. Such an analysis can help to evaluate which environmental health problem can be reduced most efficiently and effectively. This involves assembling potential policy measures, calculating their potential effects on the problem, assessing the costs of these measures and also evaluating the costs of the health effects (such as the costs of absence due to illness, medical costs, etc). These costs can than be compared

in a cost benefit analysis. Such an analysis was beyond the scope of this project. RIVM is currently starting a project on cost benefit analyses of transport policy measures.

When presenting results, it is important to provide the context of the calculations. As stated before, DALYs can only be interpreted properly when considering the framework, assumptions and uncertainties. Even though this context will always remain necessary, uniformity of burden of disease studies would certainly be of use. It will be very useful to acquire internationally consistent methodologies to calculate the (environmental) burden of disease, based on "good practise". These methods could specify ways to assess exposure for each factor and population, ways to obtain prevalence data, etc, and could also supply a model that processes all variables in an internationally consistent way (Prüss *et al.*, 2001). The World Health Organization has already supplied several other guideline documents and continues to try and standardize all sorts of methodologies (such as on the use of epidemiological evidence for environmental HIA, (World Health Organization, 2000)).

In addition to setting up these good practise guidelines, an attempt should be made to streamline the data that are being used, such as exposure-response relationships. Careful analysis of the relationships between risk factors and health outcomes for different populations could potentially provide an internationally accepted core set of exposure-response relationships (including guidelines on how and when to use national research instead). However, these relationships might differ in different context, so applicability might be limited. Furthermore, existing data should constantly be updated, trying to minimize uncertainty and maximize validity and applicability.

Different types of uncertainty underlie DALY calculations. Some data could theoretically be almost 100% precise, such as prevalence data for specific populations. Other factors simply cannot be completely accurate. In many cases, expert judgements were needed to estimate input values and/or uncertainty of input values. More formalized procedures need to be adopted to elicit such expert opinions.
Severity factors will always be estimates and the exact exposure of a population can only be modelled. Even though DALYs will therefore never be fixed numbers and will always be surrounded with considerable uncertainty ranges, it is worthwhile to at least try to limit these. The focus should lie on limiting uncertainty that affects the output most. Since DALYs are based on multiplications, relatively small values with relatively great uncertainty ranges, such

as severity weights, affect outcomes most. One of the weakest links in the DALY calculations is therefore the absence of a complete and validated set of severity weights. Combining all previous efforts, filling in the blanks and validating all weights by expert panels as well as lay people, could potentially lead to one internationally recognized set of severity weights. However, this set would only be valid for countries with similar cultural and social backgrounds and values, as different cultures appraise health states differently.

Although it will be difficult to limit uncertainty in DALY issues to a great extent, thinking about these issues and trying to tune ones own research to other studies might help to streamline all burden of disease studies, adding value to each individual one.

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Appendix 1 Data

Environmental factors and associated health outcomes with the corresponding number of people affected, duration and severity of the health state, and DALYs *per million people* for the year 2000.

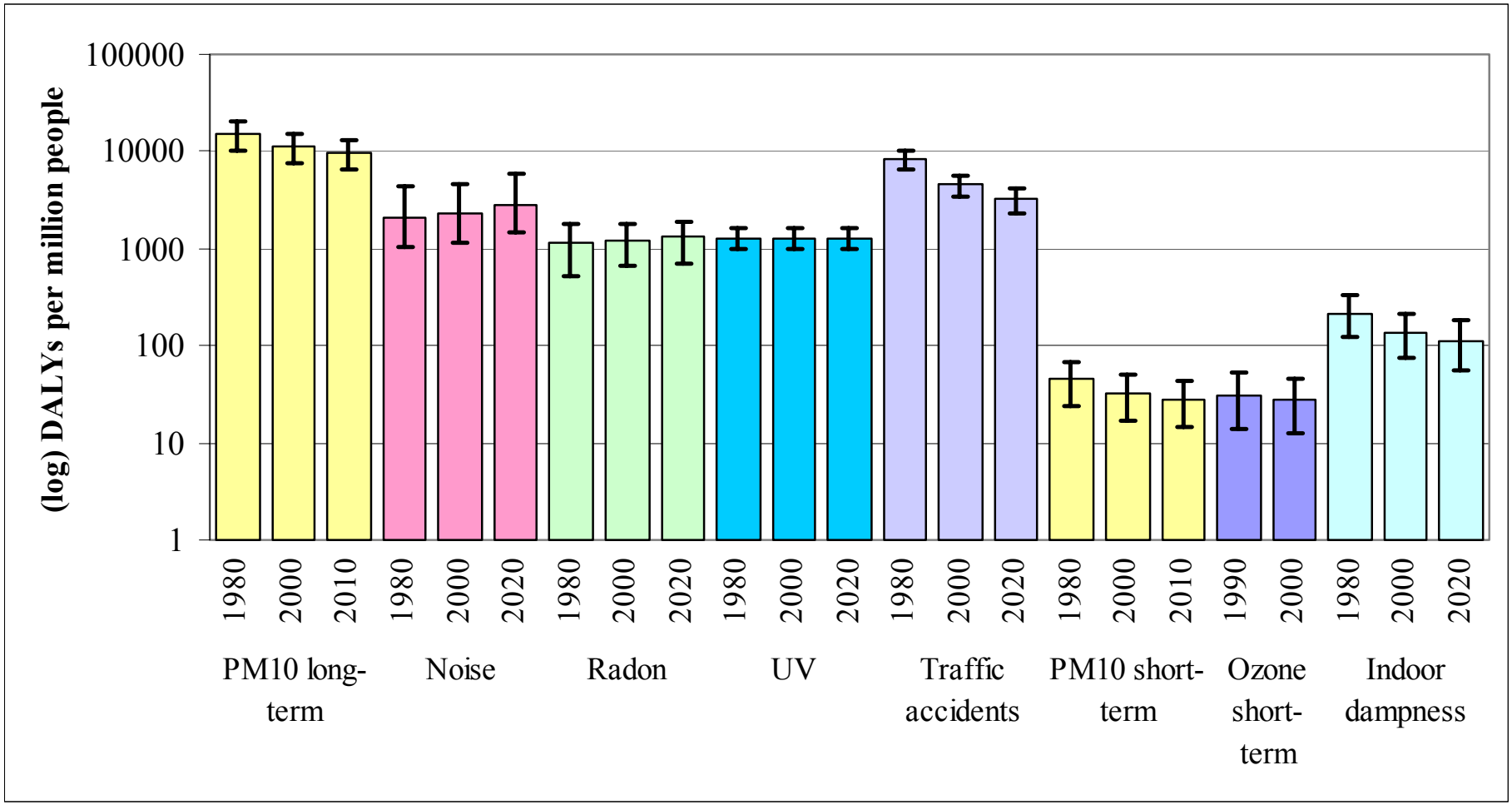
Environmental Factor	**Health Outcome**	**Total number of people affected**	**Duration (+ sd)**	**Severity (+ sd)**	**DALYs per million people Ж**
PM_{10} (short-term)	Mortality (total)	1,700 (1,200- 2,200) *based on individual PM_{10} model.* 2,800 (2,200 – 3,500) *based on 2 component-model with ozone.*	3 months (+/1 month)	1	27 (13-44) *based on individual PM_{10} model.* 45 (24 – 70) *based on 2 component-model with ozone.*
	Cardiovascular disease mortality	420 (190 – 660)	3 months (+/1 month)	1	7 (2-12)
	Respiratory disease mortality	580 (430 – 750)	3 months (+/1 month)	1	9 (4-15)
	COPD mortality	240 (160 – 340)	3 months (+/1 month)	1	4 (2-6)
	Hospital admissions cardiovascular disease	2,800 (1,900 – 3,900)	2 weeks *	0.71 (+/- 0.2)	5 (1-10)
	Hospital admissions respiratory disease (total)	700 (430- 990)	2 weeks *	0.64 (+/- 0.17)	1 (0-2)
	Hospital admissions COPD	500 (340- 670)	2 weeks *	0.53 (+/- 0.08)	1 (0-1)
	Hospital admissions asthma	*not sign*	-	-	-
	Total				*33 (17- 50)* Ж
PM_{10} (long-term)	Mortality (total)	18,100 (12,400 – 23,800)	10 years (+/- 10%) Ψ 10 (4 – 13) years	1	*11,200 (7,400 – 15,000)* Ψ 11,200 (4,100 – 29,000)
Ozone (short-term)	Mortality (total)	1,800 (1,200 – 2,400) *based on individual ozone model.* 2,400 (1,600 – 3,100) *based on 2 component-model with PM_{10}.*	3 months (+/1 month)	1	28 (13-47) *based on individual ozone model.* 38 (17 – 61) *based on 2 component-model with PM_{10}.*
	Cardiovascular disease mortality	500 (130 – 870)	3 months (+/1 month)	1	8 (2-16)
	Respiratory disease mortality	*not sign*	-	-	-
	COPD mortality	*not sign*	-	-	-
	Hospital admissions cardiovascular disease	*not sign*	-	-	-
	Hospital admissions respiratory disease (total)	*not sign*	-	-	-
	Hospital admissions COPD	*not sign*	-	-	-
	Hospital admissions asthma	*not sign*	-	-	-
	Total				*28 (13 – 47)* Ж

Noise	Mortality (through stress, hypertension and cardiovasc. disease)	620 (300-1,000)	10.5 years (+/- 10%)	1	420 (200-700)
	Severe annoyance	727,000 (564,000 -910,000)	P	0.02 (+/- 0.02)	1,200 (470 – 2,900) ξ
	Severe sleep disturbance (excl air traffic)	337,000 (227,000 – 468,000)	P	0.02 (+/- 0.02)	560 (200 – 1,400) ξ
	Total				*2,300 (1100 – 4700)* Ж
Radon	Lung cancer mortality	800 (420 – 1100)	12.3 years (+/- 10%) Δ	1	*1,200 (650 – 1,800)*
UV	Basal cell carcinoma morbidity	164,000 ф	-	0.05 (+/- 0.03)	280 (64 – 520)
	Squamous cell carcinoma morbidity	23,500 ф	-	0.07 (+/- 0.04)	110 (24 – 200)
	Squamous cell carcinoma mortality	80 ф	9.0 years (+/- 10%Δ	1	44 (39 – 49)
	Melanoma morbidity	24,000 ф	-	0.19 (+/- 0.07)	290 (130 – 470)
	Melanoma mortality	470 ф	19.1 (+/- 10%) Δ	1	570 (500 – 640)
	Total				*1,300 (1,000 – 1,600)* Ж
Traffic	Mortality	1,170 (1,120-1,210)	36.3 years (+/- 1 year) Δ	1	2,700 (2,500-2,800)
	Injury (>1 year)	67,000 (61,600-72,500)	P	0.43 (+/- 0.15)	1,800 (770 – 2,900)
	Total				*4,500 (3,500 – 5,600)* Ж
Dampness	Asthma children	4,100 (2000-6,600)	P	0.078 (+/-0.0125)	110 (49-180)
	Asthma adults	4,500 (3,100-6,200)	P	0.078 (+/-0.0125)	27 (16-40)
	Total				*140 (75 – 220)* Ж

Total of PM10 (short-term), ozone (short-term), noise, radon, UV, and dampness *(whole population)*	77,000 (45,000 – 130,000)
Total of PM10 (short-term and long-term), ozone (short-term), noise, radon, UV, and dampness *(whole population)*	250,000 (160,000 – 370,000)

Ж 90% prediction intervals around health-effect-specific DALYs are based on health-effect-specific Monte Carlo analyses. The sums of these effect-specific 90% prediction intervals per environmental factor are not necessarily exactly the same as the 90% prediction interval given for the total number of DALYs per environmental factor. This is due to the fact that the latter is based on a separate Monte Carlo analysis on the total values (excluding double counts; more information: see paragraph 3.4)
* mean value: 2 weeks (min: 4 days, max: 2 months, most likely value: 1 week)
ξ Total for road traffic, air traffic (annoyance only) and rail traffic, not corrected for overlaps.
Ψ Conservative estimate of uncertainty (10%) around years of life lost
Δ Duration differs per year (1980, 2000 or 2020), value for 2000 is given
ф Prevalence data; no exposure-response relationship and therefore no uncertainty range
P = Prevalence data, duration is standard one year

Appendix 2 Results on a logarithmic scale



PM_{10} long-term based on calculations using a conservative (10%) uncertainty interval for the number of years of life lost.