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Burden of disease from environmental noise

Quantification of healthy life years lost in Europe

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Quantification of healthy life years lost in Europe

The WHO European Centre for Environment and Health, Bonn Office, WHO Regional Office for Europe coordinated the development of this publication.

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ABSTRACT

The health impacts of environmental noise are a growing concern among both the general public and policy-makers in Europe. This publication was prepared by experts in working groups convened by the WHO Regional Office for Europe to provide technical support to policy-makers and their advisers in the quantitative risk assessment of environmental noise, using evidence and data available in Europe. The chapters contain the summary of synthesized reviews of evidence on the relationship between environmental noise and specific health effects, including cardiovascular disease, cognitive impairment, sleep disturbance and tinnitus. A chapter on annoyance is also included. For each outcome, the environmental burden of disease methodology, based on exposure–response relationship, exposure distribution, background prevalence of disease and disability weights of the outcome, is applied to calculate the burden of disease in terms of disability-adjusted life-years (DALYs). With conservative assumptions applied to the calculation methods, it is estimated that DALYs lost from environmental noise are 61 000 years for ischaemic heart disease, 45 000 years for cognitive impairment of children, 903 000 years for sleep disturbance, 22 000 years for tinnitus and 654 000 years for annoyance in the European Union Member States and other western European countries. These results indicate that at least one million healthy life years are lost every year from traffic-related noise in the western part of Europe. Sleep disturbance and annoyance, mostly related to road traffic noise, comprise the main burden of environmental noise. Owing to a lack of exposure data in south-east Europe and the newly independent states, it was not possible to estimate the disease burden in the whole of the WHO European Region. The procedure of estimating burdens related to environmental noise exposure presented here can be used by international, national and local authorities as long as the assumptions, limitations and uncertainties reported in this publication are carefully taken into account.

LIST OF ACRONYMS AND ABBREVIATIONS

ADL	Activity of daily life
AF	Attributable fraction
AR	Attributable risk
CI	Confidence interval
CLAMES	Classification and Measurement System of Functional Health
DALY	Disability-adjusted life year
DW	Disability weight
EBD	Environmental burden of disease
EEA	European Environment Agency
EEG	Electroencephalogram
EMG	Electromyogram
END	Environmental noise directive (2002/49/EC)
EOG	Electrooculogram
ETC LUSI	European Topic Centre on Land Use and Spatial Information
EU	European Union
EUR-A	WHO epidemiological subregion in Europe: Andorra, Austria, Belgium, Croatia, Cyprus, the Czech Republic, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, the Netherlands, Norway, Portugal, San Marino, Slovenia, Spain, Sweden, Switzerland and the United Kingdom
GBD	Global burden of disease
HA	Highly annoyed people
HSD	Highly sleep disturbed people
ICD-9	International Statistical Classification of Diseases and Related Health Problems, ninth revision
ICD-10	International Statistical Classification of Diseases and Related Health Problems, tenth revision
$L_{Aeq,th}$ or $L_{eq,th}$	A-weighted equivalent sound pressure level over t hours
L_{den}	Day-evening-night equivalent sound level
L_{dn}	Day-night equivalent sound level
L_{night}	Night equivalent sound level
NIHL	Noise-induced hearing loss
NOISE	Noise Observation and Information Service for Europe
NYHA	New York Heart Association
OR	Odds ratio
OSAS	Obstructive sleep apnea syndrome
PAR	Population attributable risk
PSG	Polysomnography
REM	Rapid eye movement
SWS	Slow wave sleep
WHO	World Health Organization
YLD	Years lost due to disability
YLL	Years of life lost

FOREWORD

Public health experts agree that environmental risks constitute 24% of the burden of disease. Widespread exposure to environmental noise from road, rail, airports and industrial sites contributes to this burden. One in three individuals is annoyed during the daytime and one in five has disturbed sleep at night because of traffic noise. Epidemiological evidence indicates that those chronically exposed to high levels of environmental noise have an increased risk of cardiovascular diseases such as myocardial infarction. Thus, noise pollution is considered not only an environmental nuisance but also a threat to public health.

In 1999, WHO summarized the scientific evidence on the harmful impacts of noise on health and made recommendations on guideline values to protect public health in its *Guidelines for community noise*. The European Union (EU) enacted a directive on the management of environmental noise in 2002 and, accordingly, most EU Member States have produced strategic noise maps and action plans on environmental noise. The WHO European Centre for Environment and Health, Bonn Office, with the financial support of the European Commission, developed *Night noise guidelines for Europe* and provided expertise and scientific advice to policy-makers for future legislation in the area of night noise control and surveillance. Furthermore, a series of projects addressing the health burden of noise was implemented by the WHO Regional Office for Europe in 2005–2009.

At the Fifth Ministerial Conference on Environment and Health, in Parma, Italy in March 2010, the Member States urged WHO to develop suitable guidelines on environmental noise policy. This publication, developed by WHO with the support of the Joint Research Centre of the European Commission, responds to that request by assisting policy-makers in quantifying the health impacts of environmental noise. The evidence-base on burden of disease presented here will inform the new European health policy, Health 2020, which is being prepared by the WHO Regional Office for Europe for endorsement by the Member States in 2012.

The review of the scientific evidence supporting exposure–response relationships and case studies in calculating burden of disease was performed by a working group composed of outstanding scientists. The contents of this publication have been peer reviewed. The Regional Office is thankful to those who contributed to its development and presentation of this document and believe that this work will facilitate the implementation of the Parma Declaration and contribute to improving the health of the citizens of Europe.

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EXECUTIVE SUMMARY

Introduction

Urbanization, economic growth and motorized transport are some of the driving forces for environmental noise exposure and health effects. Environmental noise is defined as noise emitted from all sources except industrial workplaces. The EU Directive on the management of environmental noise (END) adds industrial sites as sources of environmental noise.

To estimate the environmental burden of disease (EBD) due to environmental noise, a quantitative risk assessment approach has to be used. Risk assessment refers to the identification of hazards, the assessment of population exposure and the determination of appropriate exposure–response relationships. The EBD is expressed as disability-adjusted life years (DALYs). DALYs are the sum of the potential years of life lost due to premature death and the equivalent years of "healthy" life lost by virtue of being in states of poor health or disability.

WHO estimated the global burden of disease (GBD) in the second half of the 1990s. The environmental burden of disease due to environmental factors such as lead, outdoor and indoor air pollution and water and sanitation was first published in 2002. The WHO European Centre for Environment and Health, Bonn Office, convened meetings of a working group to estimate the EBD due to exposure to environmental noise. The conclusions and recommendations of these meetings were synthesized to develop this guidance publication on risk assessment of environmental noise using evidence and data available in Europe.

The target audience for this publication is primarily policy-makers, their technical advisers and staff from supporting agencies, and other stakeholders who need to estimate the effects of environmental noise. It brings together evidence-based information on health effects of environmental noise and provides exemplary guidance on how to quantify these effects. In summary, the aims of the publication are to provide:

- guidance on the procedure for the health risk assessment of environmental noise;
- reviews of evidence on the relationship between environmental noise and health effects;
- exemplary estimates of the burden of the health impacts of environmental noise; and
- a discussion of the uncertainties and limitations of the EBD procedure.

The health end-points of environmental noise considered by the working group for the EBD estimation included cardiovascular disease, cognitive impairment, sleep disturbance, tinnitus and annoyance. Although annoyance was not addressed as a health outcome of the GBD project, it was selected for the EBD estimation in consideration of WHO's broad definition of health.

Exposure assessment

Assessment of exposure to noise requires consideration of many factors, including:

- the measured or calculated/predicted exposure, described in terms of an appropriate noise metric; and
- the distribution of the exposure of the population to noise.

Population noise exposure in this publication is based on the noise mapping mandated by the END, using the annual average metrics of L_{den} (day-evening-night equivalent level) and L_{night} (night equivalent level) proposed in the Directive.

$$L_{den} = 10 \cdot \lg \frac{1}{24}\left(12 \cdot 10^{\frac{L_{day}}{10}} + 4 \cdot 10^{\frac{L_{evening}+5}{10}} + 8 \cdot 10^{\frac{L_{night}+10}{10}}\right)$$

with $L_{day} = L_{eq,12h}$, $L_{evening} = L_{eq,4h}$

and $L_{night} = L_{eq,8h}$

with $L_{Aeq,th}$ the A-weighted equivalent sound pressure level over t hours outside at the most exposed facade.

Methods of environmental burden of disease assessment

The burden of disease is expressed in DALYs in the general population through the equation

$$DALY = YLL + YLD$$

In this equation, YLL is the number of "years of life lost" calculated by

$$YLL = \sum_i (N_i^m \cdot L_i^m + N_i^f \cdot L_i^f)$$

where $N_i^m (N_i^f)$ is the number of deaths of males (females) in age group i multiplied by the standard life expectancy $L_i^m (L_i^f)$ of males (females) at the age at which death occurs. YLD is the number of "years lived with disability" estimated by the equation

$$YLD = I \cdot DW \cdot D$$

where I is the number of incident cases multiplied by a disability weight (DW) and an average duration D of disability in years. DW is associated with each health condition and lies on a scale between 0 (indicating the health condition is equivalent to full health) and 1 (indicating the health condition is equivalent to death).

The EBD of each end-point was estimated using the following information and data:

- the distribution of environmental noise exposure within the population;
- the exposure–response relationships for the particular health end-point;
- the population-attributable fraction due to environmental noise exposure;
- a population-based estimate of the incidence or prevalence of the health end-point from surveys or routinely reported statistics; and
- the value of DW for each health end-point.

Cardiovascular diseases

The evidence from epidemiological studies on the association between exposure to road traffic and aircraft noise and hypertension and ischaemic heart disease has increased during recent years. Road traffic noise has been shown to increase the risk of ischaemic heart disease, including myocardial infarction. Both road traffic noise and aircraft noise increase the risk of high blood pressure. Very few studies exist regarding the cardiovascular effects of exposure to rail traffic noise.

Exposure–response relationships

Numerical meta-analyses were carried out assessing exposure–response relationships between community noise and cardiovascular risk. A polynomial function was fitted through the data points from the analytic studies within the noise range from 55 to 80 dB(A):

$$OR = 1.63 - 6.13 \cdot 10^{-4} \cdot L^{2}_{day,16h} + 7.36 \cdot 10^{-6} \cdot L^{3}_{day,16h}$$

Estimated burden in western Europe

Based on the exposure data from the noise maps of EU Member States, it is estimated that the burden of disease from environmental noise is approximately 61 000 years for ischaemic heart disease in high-income European countries.

Cognitive impairment in children

The case definition of noise-related cognitive impairment is: The Reduction in cognitive ability in school-age children that occurs while the noise exposure persists and will persist for some time after the cessation of the noise exposure. The extent to which noise impairs cognition, particularly in children, has been studied with both experimental and epidemiological studies.

Hypothetical exposure–response relationship

Based on available evidence, a hypothetical exposure–response relationship between noise level (L_{dn}) and risk of cognitive impairment was formulated: all of the noise-exposed children were cognitively affected at a level as high as 95 dB(A) L_{dn}, and no children were affected at a relatively low level, such as 50 dB(A) L_{dn}. A linear relationship in the range of these two limits was assumed as a basis for a conservative approximation of YLD.

Estimated burden in western Europe

If one extrapolates the exposure distribution and population structure of Sweden to western European countries, the estimated DALYs for the EUR-A countries are 45 000 years for children aged 7–19 years.

Sleep disturbance

Sleep disturbance can be measured electro-physiologically or by self-reporting in epidemiological studies using survey questionnaires. In epidemiological studies, "self-reported sleep disturbance" is the most easily measurable outcome indicator, because electro-physiological measurements are costly and difficult to carry out on large samples and may themselves influence sleep.

Exposure–response relationship

The percentage of "highly sleep disturbed" persons (HSD) as a function L_{night} was calculated with the equation:

$$HSD[\%] = 20.8 - 1.05 \cdot L_{night} + 0.01486 \cdot L_{night}^2$$

Estimated burden in western Europe

Conservative estimates applied to the calculation using exposure data from noise maps give a total of 903 000 DALYs lost from noise-induced sleep disturbance for the EU population living in towns of > 50 000 inhabitants.

Tinnitus

Tinnitus is defined as the sensation of sound in the absence of an external sound source. Tinnitus caused by excessive noise exposure has long been described; 50% to 90% of patients with chronic noise trauma report tinnitus. In some people, tinnitus can cause sleep disturbance, cognitive effects, anxiety, psychological distress, depression, communication problems, frustration, irritability, tension, inability to work, reduced efficiency and restricted participation in social life.

Exposure–response relationship

For tinnitus due to environmental noise, exposure to social/leisure noise such as personal music players, gun shooting events, music concerts, sporting events and events using firecrackers is most relevant for western Europe and North American countries. Population-based studies associating exposure to leisure noise with the risk of tinnitus are rare. From studies on people with tinnitus, a mean prevalence was calculated of those with slight, moderate and severe tinnitus.

Estimated burden in western Europe

Applying the mean prevalence data to the EUR-A population of 344 131 386 people aged 15 years and over in 2001, the prevalence of slight, moderate and severe tinnitus was estimated. DW of 0.01 was chosen for slight tinnitus and 0.11 for moderate and severe tinnitus. An educated guess of 0.03 was made for the population-attributable fraction of tinnitus caused by environmental noise exposure. DALYs for noise-induced tinnitus were estimated to be 22 000 years for the EUR-A adult population.

Annoyance

WHO defines health as a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity. Therefore, a high level of annoyance caused by environmental noise should be considered as one of the environmental health burdens. Standardized questionnaires are used to assess noise-induced annoyance at the population level. The percentage of highly annoyed is the most widely used prevalence indicator for annoyance in a population.

Exposure–response relationship

The percentage of "highly annoyed" persons (HA) due to road traffic noise was calculated with the equation:

$$HA[\%] = 0.5118 \cdot (L_{den} - 42) - 1.436 \cdot 10^{-2} \cdot (L_{den} - 42)^2 + 9.868 \cdot 10^{-4} \cdot (L_{den} - 42)^3$$

Estimated burden in western Europe

Conservative estimates applied to the calculation using exposure data from noise maps give a total of 654 000 DALYs lost from noise-induced annoyance for the EU population living in towns of > 50 000 inhabitants.

Conclusions

There is sufficient evidence from large-scale epidemiological studies linking the population's exposure to environmental noise with adverse health effects. Therefore, environmental noise should be considered not only as a cause of nuisance but also a concern for public health and environmental health.

This publication was produced by the working group convened by the Regional Office to provide policy-makers and their advisers in national and local authorities with exemplary practices of using WHO methods of quantifying the burden of disease for selected health end-points. Because of the uncertainties in exposure assessment, exposure–response relationships and health statistics, conservative assumptions were made as far as possible.

It is estimated that DALYs lost from environmental noise in the western European countries are 61 000 years for ischaemic heart disease, 45 000 years for cognitive impairment of children, 903 000 years for sleep disturbance, 22 000 years for tinnitus and 654 000 years for annoyance. If all of these are considered together, the range of burden would be 1.0–1.6 million DALYs.[1] This means that at least 1 million healthy life years are lost every year from traffic-related noise in the western European countries, including the EU Member States. Sleep disturbance and annoyance related to road traffic noise constitute most of the burden of environmental noise in western Europe. Owing to a lack of exposure data in south-east Europe and the newly independent states, it was not possible to estimate the disease burden in the whole of the WHO European Region.

The procedure of estimating the burden of selected health end-points related to environmental noise exposure presented here can be used by international, national and local authorities as long as the assumptions, limitations and uncertainties reported in this publication are carefully taken into account. This publication also provides an updated review of evidence for the future development of suitable guidelines on noise by WHO, as its urged by Member States in the Parma Declaration adopted at the Fifth Ministerial Conference on Environment and Health in 2010.

1 The extent to which years lost from different effects are additive across different outcomes is unclear. The different health outcomes might have synergistic rather than antagonistic effects when the combined effects occur in a person. Therefore, it would be a prudent approach to add the DALYs of different outcomes without considering synergistic effects.

1. INTRODUCTION

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Noise is a major environmental issue, particularly in urban areas, affecting a large number of people. To date, most assessments of the problem of environmental noise have been based on the annoyance it causes to humans, or the extent to which it disturbs various human activities. Assessment of health outcomes potentially related to noise exposure has so far been limited *(1)*.

According to preliminary results from the Environmental Burden of Disease (EBD) in Europe project in six European countries *(2)* reported at the WHO Ministerial Conference held in Parma in March 2010 *(3)*, traffic noise was ranked second among the selected environmental stressors evaluated in terms of their public health impact in six European countries. Further, the trend is that noise exposure is increasing in Europe compared to other stressors (e.g. exposures to second hand smoke, dioxins and benzene), which are declining.

In its *Guidelines for community noise (4)*, the WHO defined environmental noise as "noise emitted from all sources except for noise at the industrial workplace". European Union (EU) Directive 2002/49/EC on the management of environmental noise *(5)* defines environmental noise as "unwanted or harmful outdoor sound created by human activities, including noise from road, rail, airports and from industrial sites". The terms community, residential or domestic noise have also been applied to environmental noise, although these terms are not necessarily used consistently. This publication examines health risk assessment for these sources of environmental noise.

In recent years, evidence has accumulated regarding the health effects of environmental noise. For example, well-designed, powerful epidemiological studies have found cardiovascular diseases to be consistently associated with exposure to environmental noise. In order to inform policy and to develop management strategies and action plans for noise control, national and local governments need to understand and consider this new evidence on the health impacts of environmental noise. For this purpose, there should be a risk assessment to evaluate the extent of the potential health effects.

The process of risk assessment of environmental noise requires knowing:

- the nature of the health effects of noise;
- the levels of exposure at which health effects begin to occur and how the extent of the effect changes with increasing noise levels; and
- the number of people exposed to these hazardous levels of noise.

Quantitative risk assessments based on EBD methodology have been developed and used by WHO to help the Member States quantify several environment-related

health problems *(6)*. The EBD is usually expressed as the number of deaths and the metric disability-adjusted life year (DALY), which combines the concepts of (*a*) potential years of life lost due to premature death and (*b*) equivalent years of "healthy" life lost by virtue of being in a state of poor health or disability. An estimate for burden of disease due to noise exposure has been made in Germany and other European countries as well as by nongovernmental organizations.

In recent years, the Bonn Office of the WHO European Centre for Environment and Health has organized several meetings of experts to examine the current state of knowledge and to further develop approaches for quantifying the effect of noise on health. The outcomes of these meetings are summarized in this publication.

Aims of this publication

The target audience for this publication is primarily policy-makers and their technical advisers who need to evaluate the issue of environmental noise in their jurisdictions. Publication brings together information on the evidence base on the health effects of environmental noise and provides guidance on how to quantify these effects. It aims to provide:

- synthesized reviews of evidence on the relationship between environmental noise and health effects in order to inform policy-makers and the public about the health impacts of exposure to noise;

- exemplary estimates of the health impacts of environmental noise based on exposure–response relationships, exposure distribution, population-attributable fraction, background prevalence of disease and disability weights; and

- guidance on the process of health risk assessment of environmental noise consistent with the EBD methodology of WHO.

This publication has been prepared with a European focus in terms of policy, available data and legislation. Nevertheless, as long as the assumptions, limitations and uncertainties described in the various chapters are carefully taken into account, the processes of risk assessment illustrated here can also be applied outside Europe.

Risk assessment

The objective of risk assessment is to support decision-making by assessing risks of adverse effects on human health and the environment from chemicals, physical factors and other environmental stresses. There are several different frameworks available to guide risk assessment. The one used in this publication is the framework outlined in the WHO guideline publication *Evaluation and use of epidemiological evidence for health risk assessment (7)*. Other frameworks are used by other organizations *(8,9)*.

The WHO model splits health risk assessment into two activities: health hazard characterization and health impact assessment *(7)*. The results of risk assessment can be fed into risk management, including regulatory options. This publication focuses on health impact assessment aspect of risk assessment; the management of risk from environmental noise is not discussed here.

The process of risk assessment involves the synthesis and interpretation of the evidence from the available data, often across scientific disciplines. There are several limitations, challenges and uncertainties at each step. These include the availability and consistency of the evidence, chance and bias affecting the validity of studies, and the transparency, reproducibility and comprehensiveness of reviews.

Hazard identification (identification of effects of noise)

After reviewing the available scientific evidence supporting causal association, the following outcomes were selected for inclusion:

- cardiovascular disease
- cognitive impairment
- sleep disturbance
- tinnitus
- annoyance.

While a chapter on hearing impairment due to environmental noise would have been useful, it was found that the data available on the prevalence of leisure noise and the relationship between environmental noise and hearing impairment were not adequate for burden of disease calculations.

On the other hand it was thought to be important to include a chapter on the effect of environmental noise on high annoyance lasting (at least) one year. Although high annoyance is not classified as a disease in the International Classification of Disease (ICD-9; ICD-10), it does affect the well-being of many people and therefore may be considered to be a health effect falling within the WHO definition of health as being a "state of complete physical, mental and social well-being". More importantly, however, it is the effect of noise that most lay people are aware of and concerned about. It was believed that many jurisdictions would be interested in estimating the effects of noise on this outcome.

Exposure assessment

There are many different sources of environmental noise to which people are exposed including, for example:

- transport (road traffic, rail traffic, air traffic);
- construction and industry;
- community sources (neighbours, radio, television, bars and restaurants); and
- social and leisure sources (portable music players, fireworks, toys, rock concerts, firearms, snowmobiles, etc.).

Noise from all sources may be relevant to the assessment of risk, and hence it may be appropriate to assess the exposure of the population of interest to all of these sources. In practice, it is almost impossible to consider exposure to all sources in the risk assessment, because some exposures are difficult to estimate at the population level (for example, leisure noise through attending music concerts or listening to personal music devices). By contrast, considerable work has been done on assessing the exposure of populations to noise sources such as air traffic and road traffic.

Assessment of exposure to noise requires consideration of many factors, including:

- measured exposure or calculated/predicted exposure
- choice of noise indicator
- population distribution
- time-activity patterns of the exposed population
- combined exposures to multiple sources of noise.

The exposure of the population of interest to the noise source can be obtained by measurement or by using models that calculate noise exposure based on information about the source and on information about sound propagation conditions from source to receiver. Such calculation models can also be used to predict levels of noise exposure for some time in the future based on estimated changes in noise sources. Best-practice methods should be adopted for measurement or for calculation in the assessment of exposure, with a full understanding of the assumptions, limitations and potential errors associated with any approach to measurement or estimation. For example, a common approach to assessing the exposure of people to transport noise is to use, as a proxy, the exposure of the most exposed side of the dwelling in which they live. This may not always be a good approximation, however, because the rooms in which people spend most time may not be on the most exposed side of the dwelling.

Noise exposure mapping is a commonly adopted step in the process of estimating the noise exposure of a population. EU Directive 2002/49/EC on the management of environmental noise *(5)* mandated all EU Member States to produce strategic noise maps based on harmonized indicators by 2008 (see Box 1.1).

Box 1.1. EU Directive 2002/49/EC on the management of environmental noise

Noise has high priority on lists of environmental issues in Europe and noise reduction has increasingly become a focus for EU legislation and management. From the 1970s, successive directives have laid down specific noise emission limits for most road vehicles and for many types of outdoor equipment. Despite this increasingly stringent control of emissions, however, and despite the considerable effort and progress made in controlling noise from industry, there has been little improvement in the levels of noise exposure of people across Europe. The European Commission's 1996 Green Paper on future noise policy *(11)* marked the start of an extended "knowledge based" approach to the problem of noise, with a special emphasis on assessing and then managing the exposure of the population to environmental noise.

The European Commission developed a new framework for noise policy based on shared responsibility between the EU and national and local governments. It included a comprehensive set of measures to improve the accuracy and standardization of data to help improve the coherency of different actions:

- the creation of a Noise Expert Network *(12)*, whose mission is to assist the Commission in the development of its noise policy;

- EU Directive 2002/49/EC on the management of environmental noise *(5)*; and

- the follow-up and further development of existing EU legislation relating to sources of noise such as motor vehicles, aircraft and railway rolling stock, and the provision of financial support to noise-related studies and research projects.

The European Parliament and Council adopted Directive 2002/49/EC of 25 June 2002, whose main aim is to provide a common basis for tackling noise problems across the EU. The underlying principles of the Directive are similar to those for other environment policy directives:

- monitoring the environmental problem by requiring competent authorities in Member States to produce strategic noise maps for major roads, railways, civil airports and urban agglomerations, based on harmonized noise indicators;

- informing and consulting the public about noise exposure, its effects and the measures considered to address noise, in line with the principles of the Aarhus Convention *(13)*;

- addressing local noise issues by requiring competent authorities to draw up action plans to reduce noise where necessary and maintain environmental noise quality where it is good (the Directive does not set any limit value nor does it prescribe the measures to be used in the action plans, which remain at the discretion of the competent authorities); and

- developing a long-term EU strategy, including objectives to reduce the number of people affected by noise and providing a framework for developing existing EU policy on noise reduction from sources.

Detailed information is available on the authorities responsible for implementing the Directive in Member States and on the agglomerations, major roads, railways and airports to be covered by the noise maps and action plans.

Exposure assessment requires specification of the noise metric that is to be utilized. There is a wide variety of noise indicators and extensive discussion of these can be found in the WHO *Guidelines for community noise (4)*. This includes such matters as the type of physical scale and the period of the day over which exposure is to be integrated: for example, “night”, “evening” or “day”.

The EU has adopted harmonized noise metrics across all of its Member States, suggesting L_{den} (day-evening-night equivalent level) as an appropriate metric to assess annoyance and L_{night} (night equivalent level) as a metric to assess sleep disturbance *(5)*. While noise limits are set individually by each EU Member State, these suggested metrics are to be used for strategic mapping of exposure in all countries. They are common across all transport sources and other sources of environmental noise. Definitions of these metrics in Directive 2002/49/EC are paraphrased in Box 1.2 below. Strategic noise maps using these harmonized noise metrics are to be used throughout Europe to assess the number of people exposed to different levels of noise. This information on population exposure can be used in the risk assessment process for environmental noise. Directive 2002/49/EC also allows the use of supplementary noise metrics (other than L_{den} and L_{night}) to monitor or control special noise situations.

A key consideration is that risk assessment cannot be carried out (using an exposure-specific approach) unless both the exposure assessment and the exposure–response relationship utilize the matching noise indicators. This becomes an issue when there is evidence that the best relationship between a particular health effect and exposure may be based on one indicator, yet data on exposure are only available based on another. While the work required by Directive 2002/49/EC will increase the availability of exposure assessments using the harmonized noise indicators, available exposure–response relationships may be reported using other indicators. These matters are discussed within each of the chapters on the various health outcomes. Exposure–response relationships reported may utilize different noise indicators because the meta-analyses in which these relationships were derived relied on studies using other noise indicators, or because there is evidence that the relationship between a particular health outcome and noise exposure is better described using a different noise indicator.

The quality of exposure data is critical to the accuracy of risk assessment. Some of the difficulties in measuring noise and preparing noise maps are outlined in a good practice guide *(14)*. They include: coverage of all relevant sources; inaccuracies in the process of linking people to noise levels and thus obtaining exposure distributions; and accounting for the presence of a quiet side or special sound insulation of a house, in particular for effects related to sleeping.

Box 1.2. Harmonized noise indicators in EU Directive 2002/49/EC

The day-evening-night level L_{den} in decibels is defined by:

$$L_{den} = 10 \cdot \lg \frac{1}{24} \cdot \left(12 \cdot 10^{\frac{L_{day}}{10}} + 4 \cdot 10^{\frac{L_{evening}+5}{10}} + 8 \cdot 10^{\frac{L_{night}+10}{10}} \right)$$

- L_{day}, $L_{evening}$ and L_{night} are the A-weighted 12, 4, 8 hours average sound levels, respectively, as defined in ISO 1996-2:1987 *(15)*.
- The day is 12 hours, the evening 4 hours and the night 8 hours. Member States may shorten the evening period by 1 or 2 hours and lengthen the day and/or the night period accordingly (same for all the sources).
- The start of the day (and consequently the start of the evening and the start of the night) shall be chosen by the Member State (same for all sources); the default values are 07:00–19:00, 19:00–23:00 and 23:00–07:00 local time.
- The incident sound is considered, which means that no account is taken of the sound that is reflected at the facade of the dwelling under consideration.

The nighttime noise indicator L_{night} is the A-weighted long-term average sound level.

- The night is 8 hours as defined above.

Supplementary noise indicators. In some cases, in addition to L_{den} and L_{night}, and where appropriate L_{day} and $L_{evening}$, it may be advantageous to use special noise indicators and related limit values. Some examples (consult Directive 2002/49/EC for full advice) are:

- a very low average number of noise events in one or more of the periods (for example, less than one noise event an hour); a noise event could be defined as a noise that lasts less than five minutes, such as the noise from a passing train or aircraft;
- strong low-frequency content of the noise; and
- L_{Amax} or SEL (sound exposure level) for night period protection in the case of noise peaks.

Environmental burden of disease assessment

A detailed introduction to the calculation of EBD is available elsewhere *(16,17)*. In this section, we describe the main methods used to calculate EBD that are applied in the following chapters on each health outcome of environmental noise, and discuss some of the strengths and weaknesses of each approach.

In general, the number of deaths and cases of each of the outcomes is estimated in the initial process of EBD calculation. The burden of disease is expressed in deaths and DALYs. The DALY combines in one measure the time lived with disability (YLD) and the time lost due to premature mortality (YLL) in the general population:

$$DALY = YLL + YLD$$

The YLD is the number of incident cases (I) multiplied by a disability weight (DW) and an average duration of disability in years (L):

$$YLD = I \cdot DW \cdot L$$

The YLL essentially corresponds to the number of deaths (N) multiplied by the standard life expectancy at the age at which death occurs (L):

$$YLL = N \cdot L$$

These simple formulae can be further adjusted by discounting for the timing of the health effect (now or in the future) and by the relative value of a year of life lived at different ages using different assumptions (age weighting).

The approach to estimating total disease burden can be summarized in the following steps: (*a*) estimating the exposure distribution in a population; (*b*) selecting one or more appropriate relative risk estimates from the literature, generally from a recent meta-analysis; and (*c*) estimating the population-attributable fraction with the formula for population-attributable fraction. This is referred to in this volume as the exposure-based approach. In certain instances, the number of cases is also directly estimated on the basis of the exposure (outcome-based approach).

Exposure-based approach

This approach uses the distribution of noise exposure within the study population to estimate the fraction of disease in the population that is attributable to noise. This is then applied to the disease estimates. This approach requires the measurement or calculation of:

- the distribution of the exposure to environmental noise within the population (prevalence of noise exposure);
- the exposure–response relationship for the particular outcome;
- a population-based estimate of the incidence or prevalence of the outcome from surveys or routinely reported statistics; and
- a value of DW for each health outcome.

Prevalence of noise exposure

Estimates are required of the distribution of the exposure in the population of interest using the chosen noise metric.

Exposure–response relationship

Exposure–response relationships are usually obtained from epidemiological studies. The validity of any exposure–response relationship depends on the quality of the studies used to derive it, the choice of studies used and the modelling process used to pool the results. It is therefore very important that the process to derive the exposure–response relationships is well defined. In some cases, very well-designed studies can provide this information. In other cases, it is necessary to undertake a meta-analysis to combine a number of different studies. According to the WHO guidelines *(4)*, the process of meta-analysis should include, as a minimum:

- a systematic review of the available epidemiological information on exposure–response relationships;
- an inventory of studies that provide quantitative information on exposure or that allow linkage to such information;
- additional selection of studies according to clear inclusion criteria; and
- a meta-analysis of published results or pooling of original data.

The exposure–response relationship may be reported as a regression formula or as a relative risk measure for a given change in noise (or comparing noise-exposed to noise-unexposed). Important issues to consider in the meta-analysis are:

- the quality of studies that have been used in the meta-analysis and the selection criteria used;
- the completeness of the search for studies;
- the quality of the assessment of noise exposure;
- the temporality of the noise exposure (for example, nighttime noise exposure is relevant for sleep disturbance, while daytime noise exposure is important for annoyance and cognitive impairment); and
- the relevance of the published studies to the population for which the risk assessment is being carried out.

In addition, it may be necessary to extrapolate relationships beyond the range of exposure observed in the available epidemiological studies. The arguments for the validity of such an extrapolation must be stated.

Incidence (or prevalence) of outcome

The definition of health outcome in the exposure–response relationship should be consistently used when the incidence data are collected. Some outcomes are easily obtained from national health statistics. For example, deaths from cardiovascular disease in a population per year are routinely collected in most developed countries.

For other outcomes, such routine data may not be available and in these cases prevalence or incidence of outcomes may need to be determined by surveys of the population. The accuracy of the estimates of these outcomes depends on the questions used for each individual survey. Standardized and validated questionnaires are recommended. For example, asking people how often they take medication to overcome sleeping difficulties may differ according to the availability of medication and the definition of sleeping difficulties implicit in the question. The timing of the outcome is important, either reflecting lifetime prevalence ("Have you ever had ...?"), point prevalence ("Do you currently have ...?") or incidence ("Since the last survey have you developed new ...?"). Depending on the condition, severity may be important as different severities of the outcome may have different DWs (e.g. mild, moderate or severe hearing loss).

Attributable fraction

The attributable fraction is the proportion of disease in the population that is estimated to be caused by noise. The accuracy of the fraction of the outcome attributable to environmental noise may also be difficult to specify. If the distribution of exposure and the exposure–response relationship are known, the population-attributable risk percentage can be estimated for a population (see above). The following formulae can be used to calculate the attributable risk percentage (AR%), the population-attributable risk percentage (PAR%), and the population-attributable risk (PAR) for each noise category *(16)*:

$AR\% = (RR-1) / RR \cdot 100\ [\%]$

$PAR\% = P_e/100 \cdot (RR-1) / (P_e/100 \cdot (RR-1) + 1) \cdot 100\ [\%]$

$PAR = PAR\% / 100 \cdot N_d$

RR = relative risk (odds ratios are estimates of the relative risk)

P_e = percentage of the population exposed [%]

N_d = number of subjects with disease (disease occurrence).

A more generalized formula for the calculation of the population-attributable fraction (PAF) that better accounts for multiple comparisons for large relative risks may also be used:

$PAF = \{\Sigma(P_i \cdot RR_i) - 1\} / \Sigma (P_i \cdot RR_i)\}$

P_i = proportion of the population in exposure category *i*

RR_i = relative risk at exposure category *i* compared to reference level

$\Sigma P_i = 1$

$PAR = PAF \cdot N_d$

Disability weight

DWs allow non-fatal health states and deaths to be measured under a common unit *(15)*. DWs quantify time lived in various health states to be valued and quantified on a scale that takes account of societal preferences. DWs that are commonly used for calculating DALYs are measured on a scale of 0–1, where 1 represents death and 0 represents ideal health.

The values of DWs for various disease states have been the subject of considerable discussion and work. They are generally derived from expert panels. This work has been documented extensively *(17)* and will not be summarized further here. WHO has a reasonably comprehensive list of DWs *(17)* and these are recommended for use. If there is no appropriate DW, then an expert committee may be asked to find an appropriate DW by analogy with other known DWs.

Advantages and disadvantages of this method

The methods described above are the most common approach used in health risk assessments because the methodology has been established and accepted in comparative risk analysis of WHO's EBD projects *(16)*. They provide standardized estimates of the health risk due to noise that may be understood by workers in the field. However, as described above, these methods require detailed data on noise exposure, the outcome and the exposure–response relationship. Such data are not always easy to obtain and often have significant limitations. For example, the exposure–response relationships may be based on extrapolation from a small number of studies with few subjects and perhaps even a measure of noise exposure that is not available on a population basis. This means that the estimates usually suffer from a considerable degree of uncertainty. This uncertainty is very difficult to quantify, although it is sometimes possible to provide low and high limits using sensitivity analyses *(17)*.

Outcome-based approach

For some noise-related outcomes, such as sleep disturbance and tinnitus, it is possible to estimate the burden directly through national or international surveys. This approach requires:

- an estimate of the prevalence of the outcome attributable to environmental noise; and
- a value of DW corresponding to this outcome.

The choice of questions in the survey needs to be carefully considered so as to be able to differentiate various severities of outcome and be compatible with the DWs. When the data on outcomes are not specific to environmental noise, attributable fractions should be applied to the data. When information on population exposure and/or the exposure–response relationship is not known, expert opinion may be

sought on what proportion of cases of an outcome is due to environmental noise. This approach was used for the chapter for tinnitus in this report, because exposure data on leisure noise and exposure–response relationships are not available for tinnitus.

The number of cases can then be multiplied by the DW to obtain the DALYs. When using this method, the attribution of the cause of the outcome tends to be more subjective than in exposure-based approaches.

Process of developing this publication

There is currently little information at the international level on the health impact of environmental noise in the WHO European Region. The WHO Regional Office for Europe has carried out an assessment study to provide methodological guidance for estimating the burden of disease related to environmental noise by calculating preliminary estimates of DALYs for the European Region.

The noise EBD project was started in 2005. An expert working group was convened in Stuttgart in June 2005 to review the health effects of noise and the selection of noise-related health outcomes for EBD estimation. Cardiovascular disorders, cognitive impairment, sleep disturbance, hearing loss, tinnitus and annoyance were selected as outcomes to be considered.

A second meeting was held in Bern in December 2005 to review the initial estimates of the burden of disease from environmental noise. Experts provided background documents and made presentations reviewing the detailed methods and preliminary results of EBD assessment for the selected noise-related outcomes. For each topic, a state-of-the-art review was made regarding the exposure data, exposure–response relationships, outcome data, DW and DALY calculation. WHO staff provided the topic-specific experts with methodological guidance based on previous global burden of disease experience. The meeting identified methodological constraints and informational gaps in quantification of DALYs due to environmental noise.

The methods and preliminary estimates were further elaborated in Berlin in April 2006 and in Bonn in December 2006. It was noted that calculation of DALYs is not possible for more than a few countries owing to the limited availability of data in most European countries. Because of this difficulty, the working group had to focus on providing methodological guidance on risk assessment rather than on estimating the EBD of environmental noise. Because EU Directive 2002/49/EC provides exposure data in many countries, it was also decided that the exposure metrics should use the Directive indicators as much as possible. With these aims in mind, a meeting of experts was convened in Bonn in May 2008.

Subsequent to the Bonn meeting, the authors of this chapter edited the final document. All chapters have been peer-reviewed, both within the working group and externally. At the final compilation of the chapters on health outcomes, the chapter on hearing loss was excluded because of a lack of epidemiological data pointed out by the reviewers. All other chapters were revised by the authors, taking into account the comments of the reviewers.

In 2010, exposure data on urban areas of > 250 000 inhabitants in the EU Member States became available through the EEA with the enforcement of EU Directive 2002/49/EC *(18)*. Accordingly, the WHO secretariat decided to include the EBD calculations for the EU population using the available data. In every step of the calculation that involved uncertainties, the working group made conservative assumptions in filling the information gap in order to avoid any possibility of overestimation.

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2. ENVIRONMENTAL NOISE AND CARDIOVASCULAR DISEASE

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This chapter examines the burden of cardiovascular diseases related to environmental noise. It is a common experience that noise is unpleasant and affects the quality of life. It disturbs and interferes with activities of the individual, including concentration, communication, relaxation and sleep *(1,2)*. Besides the psychosocial effects of community noise, there is concern about the impact of noise on public health, particularly regarding cardiovascular outcomes *(3–5)*.

According to the WHO Global Burden of Disease 2000 study, ischaemic heart disease is the leading cause of death in developed and developing countries (22.8% and 9.4% of total deaths, respectively *(6,7)*. Worldwide, 12.6% of deaths are caused by ischaemic heart disease, 9.6% by cerebrovascular disease and 1.6% by hypertensive heart disease *(8)*. High blood pressure and high levels of blood lipids, including cholesterol and triglycerides, are major (biological or endogenous) risk factors for ischaemic heart disease. Endogenous risk factors can be affected by exogenous risk factors (e. g. nutrition, environmental factors). Worldwide, 13.5% of deaths are attributable to high blood pressure (hypertension) and 6.9% to high (total) cholesterol levels. 1.4% of deaths are attributed to urban air pollution according to the WHO Global Burden of Disease 2000 study *(6,8)*.

The auditory system is continuously analysing acoustic information, which is filtered and interpreted by different cortical and sub-cortical brain structures. Arousal of the autonomic nervous system and the endocrine system is associated with repeated temporal changes in biological responses. In the long run, chronic noise stress may affect the homeostasis of the organism due to dysregulation, incomplete adaptation and/or the physiological costs of the adaptation *(9–17)*. Noise is considered a nonspecific stressor that may cause adverse health effects in the long run. Epidemiological studies suggest a higher risk of cardiovascular diseases, including high blood pressure and myocardial infarction, in people chronically exposed to high levels of road or air traffic noise. This chapter collates the available evidence regarding risk estimation for the burden of cardiovascular disease attributable to environmental noise in European regions.

Definition of outcome

Cardiovascular disease includes ischaemic heart disease, hypertension (high blood pressure) and stroke. There is no evidence available on the relationship between noise and stroke, so it will not be considered further here.

Ischaemic heart diseases (ICD 10 codes I20–I25) include angina (I20), acute myocardial infarction (I21), subsequent myocardial infarctions and complications of infarctions (I22 and I23), other acute forms of ischaemic heart disease (I24) and chronic ischaemic heart disease (I25). Essential hypertension is classified as I10 with further codes for hypertensive heart failure (I11), hypertensive renal disease (I12) and hypertensive heart and renal disease (I13).

Summary of evidence linking noise and cardiovascular disease

Epidemiological studies on the relationship between transportation noise (particularly road traffic and aircraft noise) and cardiovascular effects have been carried out on adults and on children, focusing on mean blood pressure, hypertension and ischaemic heart diseases as cardiovascular end-points. The evidence, in general, of a positive association has increased during recent years *(18–20)*. While there is evidence that road traffic noise increases the risk of ischaemic heart disease, including myocardial infarction, there is less evidence for such an association with aircraft noise because of a lack of studies. However, there is increasing evidence that both road traffic noise and aircraft noise increase the risk of hypertension. Very few studies on the cardiovascular effects of other environmental noise sources, including rail traffic, are known. Numerical meta-analyses were carried out assessing exposure–response relationships in quantitative terms *(21,22)* and the issue has been addressed in various WHO projects. The exposure–response curves presented here refer to the data collected for these projects, to illustrate the processes of a quantitative risk assessment.

Biological model of causation

Non-auditory health effects of noise have been studied in humans and animals for several decades, using laboratory and empirical methods. Biological reaction models have been derived, based on the general stress concept *(17,23–30)*. Noise is a nonspecific stressor that arouses the autonomous nervous system and the endocrine system *(9,11–14,31,32)* (C. Maschke & K. Hecht, unpublished data, 2005). A neuro-endocrinological definition of stress is that it is a state that threatens homeostatic or adaptable systems in the body *(16,33,34)*. Increased allostatic load is associated with various diseases, including ischaemic heart disease *(35)*. The epidemiological reasoning is based on three facts. First, experimental studies in the laboratory have been carried out for a long time and revealed an increased vegetative and endocrine reactivity during periods of exposure *(1,36–70)*. However, the question regarding long-term effects of chronic noise exposure cannot be answered from short-term experiments. Second, animal studies have shown manifest disorders in species exposed to high levels of noise for a long time *(71–83)*. However, effects in humans and animals cannot be directly compared, particularly because two pathways may be relevant – the direct effect due to nervous innervation and the indirect effect due to the cognitive perception of the sound; the latter is certainly different in humans. Furthermore, noise levels in animal studies were higher than in ambient situations. Third, occupational studies have shown health disorders in workers chronically exposed to noise for many years *(20,84–98)*. However, noise levels were higher than in the ambient environment. Epidemiological research has therefore been carried out with respect to community noise levels to test the hypothesis and to quantify the risk.

Among other non-auditory health end-points, short-term changes in circulation, including blood pressure, heart rate, cardiac output and vasoconstriction, as well as stress hormones (epinephrine, norepinephrine and corticosteroids), have been studied in experimental settings for many years *(32,99)*. Classical biological risk factors have been shown to be elevated in subjects that were exposed to high levels of noise *(44,54,79,100–111)*.

From this, the hypothesis emerged that persistent noise stress increases the risk of cardiovascular disorders, including hypertension and ischaemic heart disease. According to the noise/stress reaction model, the arousal of the endocrine and autonomic nervous system affects classical biological risk factors (e.g. blood pressure, blood lipids, glucose regulation, blood flow, haemostatic factors and cardiac output). Chronic metabolic changes or dysfunction due to noise increase the risk of manifest diseases, including hypertension, arteriosclerosis and myocardial infarction.

Exposure–response relationship

For a quantitative risk assessment and the derivation of guidelines for public health noise policy, a common exposure–response curve is required. The risk estimates obtained from different noise studies can be summarized using the statistical approach of meta-analysis.

Definition of exposure

Energy-based indicators of exposure (L_{eq}) are adequate and sufficient for assessing the relationship between long-term exposure to community noise and chronic diseases such as cardiovascular disorders. While single event noise indicators can be useful predictors (as additional information) for assessing the effects of acute noise (e. g. sleep disturbance) *(112)*, integrated noise indicators (e.g. a year's average noise level) are suitable predictors in epidemiological studies for assessing the long-term effects of chronic noise exposure. Such indicators should measure noise during certain periods of the day. Examples include $L_{day,16h}$ (day-noise indicator 7:00 to 23:00), $L_{day,12h}$ + $L_{evening,4h}$ (day-noise indicator 7:00 to 19:00 and evening-noise indicator 19:00 to 23:00) and $L_{night,8h}$ (night-noise indicator 23:00 to 7:00). $L_{day,16h}$ is a useful indicator for estimating health impacts according to the method proposed here. When information on noise for the various periods of the day, i.e. day/evening/night, is available, weighted and non-weighted indicators can easily be calculated for use in health studies and related quantitative risk assessment. This includes the indicators L_{den} (weighted day-evening-night noise indicator) and L_{night} according to Directive 2002/49/EC *(113)*, which are considered in noise mapping.

If only one figure is anticipated to describe the noise situation, a single noise indicator may be a useful factor to be considered in noise studies (e.g. L_{24h}, L_{dn} or L_{den}). However, since night noise is assessed separately according to Directive 2002/49/EC, it does not appear reasonable when daytime noise and nighttime noise exposures are then combined in a weighted 24-hour indicator. With respect to health effects, it would make much more sense to clearly distinguish between real day and night indicators. An optimal noise study would try to distinguish between the exposure of the living room during the day (L_{day}) and the exposure of the bedroom during the night (L_{night}). Further, the concept of L_{den} is annoyance-based. From a cardiovascular point of view, there is no rationale known for weighing factors such as +5 dB(A) or +10 dB(A) for the evening and night periods of the day. It would be a better approach to consider day and night exposures separately with respect to its effects, particularly for noise sources other than road traffic noise (where the day and night noise levels are usually highly correlated). Studies should also try to distinguish between the exposure of the living room (during daytime) and the exposure of the bedroom (during nighttime). However, such information is often not available.

When comparing study results for the meta-analyses, problems arise from the fact that different noise indicators (including even more complex national noise indices) have been used in different studies. However, conversion formulas are available for approximation. For example, with respect to road traffic noise the following empirical formula can be used for conversions between $L_{day,16h}$ and L_{den} *(114)*:

$$L_{den} \approx L_{day,16h} - 2 \cdot \ln((L_{day,16h}-L_{night,8h})/22.4))$$

However, this conversion can, per se, not be applied to other noise sources such as aircraft noise and railway noise. Nevertheless, as long as particular studies referring to Directive 2002/49/EC indicators L_{den} and L_{night} are largely missing, exposure–response relationships (regression coefficients) based on other noise indicators could approximately be considered for assessing the relative increase in risk with increasing noise level.

For the meta-analyses, noise exposure was divided into 5-dB(A) categories for the daytime outdoor average A-weighted sound pressure level ($L_{day,16h}$). This was considered in most studies. Information on nighttime exposure ($L_{night,8h}$) was seldom available. Newer studies used non-weighted or weighted averages of the 24-hour exposure (L_{eq}, L_{dn}, L_{den}) *(113)*. Some aircraft noise studies used national calculation methods (e.g. Dutch Kosten Units). Some of the studies considered subjective ratings of the noise, including noise annoyance, as indicators of noise exposure. Sound levels were converted on the basis of best-guess approximations to $L_{day,16h}$ for comparison and pooling.

In urban settings, average nighttime noise levels for road traffic tend to be approximately 7–10 dB(A) lower than average daytime levels and are relatively independent of the traffic volume of the street (except motorways) *(115–117)*. Measurements showed that L_{den} was approx. 1–3 dB(A) higher than $L_{day,16h}$ where the difference between $L_{day,16h}$ and $L_{night,8h}$ ranged from 10 to 5 dB(A) *(114)*.

In the conversion formula given above, if the difference between day and night sound levels is of the order of 7–8 dB(A), then this accounts for approximately 2 dB(A) higher L_{den} values compared to $L_{day,16h}$. This is commonly found for road traffic noise in urban streets with the 24-hour noise levels tending to be only slightly lower than daytime levels *(118)*. A conversion factor of 2 dB(A) was also suggested based on Norwegian data (T. Gjestland, personal communication, 2006). Another study found the difference range $L_{den} - L_{dn}$ to be between 0 and 1.5 dB, depending on whether the noise level L_{Aeq} dropped in the evening *(119)*.

To summarize, because the differences between L_{den} and L_{dn} are usually small, in epidemiological studies in which the relative effects of road traffic noise are studied, sound emission during the daytime can be taken as an approximate relative measure of the overall sound emission, including at night. This is further justified by the fact that existing noise regulations usually accept a 10-dB(A) difference between the day and the night. However, this approximation can only be made with respect to road traffic noise. For train and aircraft noise, no such approximation can be made. Approximate formulae for the conversion of different noise indicators are also given in the *Good practice guide for strategic noise mapping (120)*.

Meta-analysis – road traffic noise and myocardial infarction

To determine the most up-to-date and accurate exposure–response relationship between community noise and myocardial infarction, a meta-analysis was carried out *(21,121)*. By 2005, a total of 61 epidemiological studies had been recognized as having either objectively or subjectively assessed the relationship between transportation noise and myocardial infarction. Nearly all of the studies referred to road traffic noise or (commercial) aircraft noise, and a few to military aircraft noise. Most of the studies were of the cross-sectional type (descriptive studies) but observational studies such as case-control and cohort studies (analytical studies) were also available. The study subjects were children and adults. Confounding factors were not always adequately considered in some older studies. Not many studies provided information on exposure–response relationships, because only two exposure categories were considered.

All epidemiological noise studies were evaluated with respect to their feasibility for inclusion in a meta-analysis. The following criteria for the inclusion in the analysis/synthesis process were applied: (*a*) peer-reviewed in the international literature; (*b*) reasonable control of possible confounding (stratification, model adjustment, matching); (*c*) objective assessment of exposure (sound level); (*d*) objective assessment of outcome (clinical assessment); (*e*) type of study (analytical or descriptive); and (*f*) multi-level exposure–response assessment (not only dichotomous exposure categories).

Based on the above criteria, five analytical (prospective case-control and cohort) and two descriptive (cross-sectional) studies were suitable for derivation of a common exposure–response curve for the association between road traffic noise and the risk of myocardial infarction. Two separate meta-analyses were undertaken by considering the analytical studies and descriptive studies separately. The analytical studies comprised those that were carried out in Caerphilly and Speedwell with a pooled analysis of 6 years follow-up data *(122,123)* and the three Berlin studies *(124,125)*. The descriptive studies comprised the cross-sectional analyses that were carried out on the studies in Caerphilly and Speedwell *(126)*. All studies referred to the road traffic noise level during the day ($L_{day,16h}$) and the incidence (analytical studies) or prevalence (descriptive studies) of myocardial infarction as the outcome. The study subjects were men. In all analytical studies the orientation of rooms (moderator of the exposure) was considered for the exposure assessment (at least one bedroom or living room facing the street or not). In all descriptive studies the traffic noise level referred to the nearest facades that were facing the street and did not consider the orientation of rooms/windows (source of exposure misclassification). The individual effect estimates of each study were adjusted for the covariates given in these studies. This means that different sets of covariates were considered in each study. Nevertheless, this pragmatic approach accounts best for possible confounding in each study and provides the most reliable effect estimates derived from each study.

The common set of covariates considered in the descriptive studies were age, sex (males only) social class, body mass index, smoking, family history of ischaemic heart disease, physical activity during leisure time and prevalence of pre-existing diseases. The common set of covariates considered in the analytical studies were

age, sex (males only), social class, school education, employment status, shift work, smoking and body mass index. Some of the analytical studies also considered physical activity during leisure time, family history of ischaemic heart disease or myocardial infarction, prevalence of pre-existing diseases, work noise and marital status. In one study, the effect estimates were further adjusted for hypertension and diabetes mellitus. This may be a conservative approach owing to over-controlling, because these biological (risk) factors may be mediators along the pathway from exposure (noise stress) to disease.

The odds ratios calculated for the different 5-dB(A) noise categories ($L_{day,16h}$) within a single study were then pooled between studies for each noise category. Since higher exposure categories usually consist of smaller numbers of subjects than the lower categories, regression coefficients across the whole range of noise levels within a study tend to be largely influenced by the lower categories. This may lead to an underestimation of the risk in higher noise categories. The multi-level approach pooled the effect estimates of single studies within each noise category, thus giving more weight to the higher noise categories and accounting for possible non-linear associations.

The results from the two meta-analyses (descriptive studies and analytical studies) are shown in Table 2.1 *(121)*. For each meta-analysis we include the odds ratios (OR) and 95% confidence intervals (CI) for the original studies (with the weights used in the pooled analysis), the pooled OR and CI and the Laird Q-test of heterogeneity between studies. If the *P*-value from the Q-test is < 0.05, the studies are too heterogeneous and should not be combined.

The pooled estimates and CIs are shown graphically in Fig. 2.1 (descriptive studies) and Fig. 2.2 (analytical studies). The descriptive (cross-sectional) studies (Fig. 2.1) cover the sound level range of $L_{day,16h}$ from > 50 to 70 dB(A), while the cohort and case-control studies (Fig. 2.2) cover the range from ≤ 60 to 80 dB(A). The two curves together can serve as a basis for estimating the exposure–response relationship. From Fig. 2.1, it can be seen that below 60 dB(A) for $L_{day,16h}$ no noticeable increase in myocardial infarction risk is to be detected. For noise levels greater than 60 dB(A), the myocardial infarction risk increases (Fig. 2.1 and 2.2).

A polynomial function was fitted through the data points from the analytical studies (Fig. 2.2), to generate a continuous exposure–response curve that can be applied to categorized noise data and also to continuous noise data. The data points were weighted by the number of subjects (N-weighting) *(21,121)*. Mean category values of the decibel-axis are considered for the calculation. For the reference category "≤ 60 dB(A)", a value of 55 dB(A) was used because this category also includes a large number of noise levels below 55 dB(A). Using alternative values for this reference category (e.g. 52.5 or 57.5) had only a very marginal effect on the coefficients and the fit statistics. According to the empirical German noise assessment model (*Lärmbelastungsmodell*), daytime noise levels tend to be equally distributed across the categories > 45–50, > 50–55 and > 55–60 (127). In urban settings, background levels during the day do not often fall below 50 dB(A).

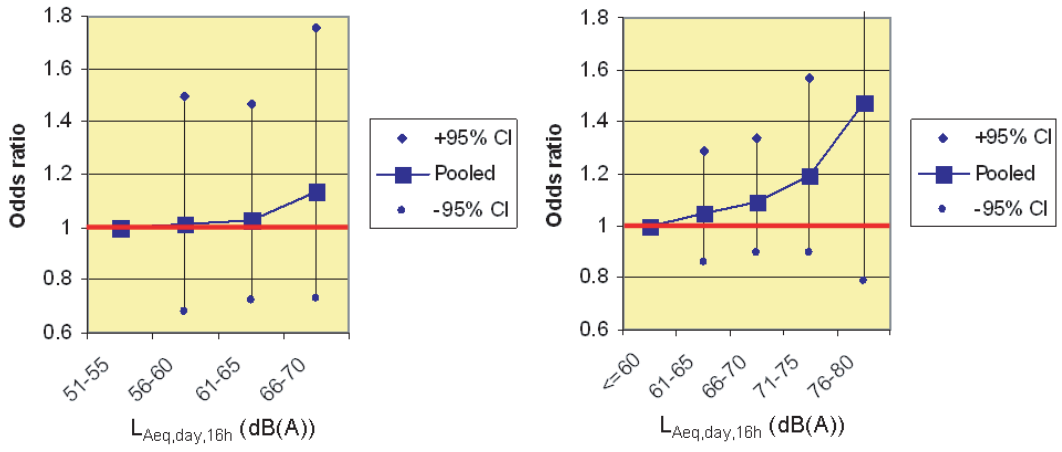
Table 2.1. Odds ratios and 95% confidence intervals from descriptive and analytical studies on the relationship between road traffic noise level and the incidence/prevalence of myocardial infarction

Descriptive studies	Road traffic noise level, $L_{day,16h}$ (dB(A))					N
	51–55	56–60	61–65	66–70		
Caerphilly	1.00	1.00 (0.58–1.71) [13.29]	0.90 (0.56–1.44) [17.23]	1.22 (0.63–2.35) [8.98]		2512
Speedwell	1.00	1.02 (0.57–1.83) [11.19]	1.22 (0.70–2.12) [12.62]	1.07 (0.59–1.94) [10.94]		2348
Pooled	1.00	1.01 (0.68–1.50)	1.02 (0.72–1.47)	1.14 (0.73–1.76)		
Q-test		P = 0.96	P = 0.41	P = 0.77		
Analytical studies	< 60	61–65	66–70	71–75	76–80	N
Caerphilly & Speedwell	1.00	0.65 (0.27–1.57) [4.95]	1.18 (0.74–1.89) [17.48]	—	—	3950
Berlin I	1.00	1.48 (0.57–3.85) [4.21]	1.19 (0.49–2.87) [4.94]	1.25 (0.41–3.81) [3.09]	1.76 (0.11–28.5) [0.50]	243
Berlin II	1.00	1.16 (0.82–1.65) [31.43]	0.94 (0.62–1.42) [22.76]	1.07 (0.68–1.68) [18.92]	1.46 (0.77–2.78) [9.27]	4035
Berlin III	1.00	1.01 (0.77–1.32) [54.42]	1.13 (0.86–1.49) [50.87]	1.27 (0.88–1.84) [28.24]	—	4115
Pooled	1.00	1.05 (0.86–1.29)	1.09 (0.90–1.34)	1.19 (0.90–1.57)	1.47 (0.79–2.76)	
Q-test		P = 0.57	P = 0.87	P = 0.84	P = 0.90	

Source: Babisch 2006 (121).

Note: Numbers are odds ratios; 95% confidence intervals are given in round brackets; weights are given in square brackets; N = sample size; Pooled = pooled estimates from meta-analysis of the studies shown; P = probability of the Q-test for heterogeneity.

Fig. 2.1 & 2.2. Pooled effect estimates (meta-analysis) of the association between road traffic noise and the prevalence (Fig. 2.1, left) and incidence (Fig. 2.2, right) of myocardial infarction (odds ratio +/- 95% confidence interval)



Source: Babisch (21).

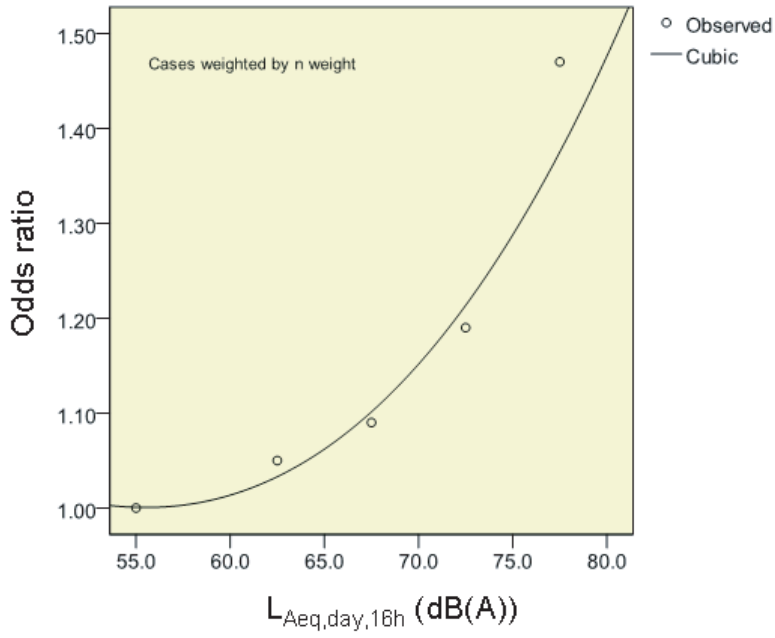
The result is shown graphically in Fig. 2.3 and mathematically below. This polynomial function explains 96% of the variance (R^2) in the meta-analytical results. Because of the data used to derive this function, the exposure–response function refers to road traffic noise and to the daytime noise indicator $L_{day,16h}$. It is defined for noise levels ranging from 55 to approximately 80 dB(A):

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

The analytical studies were chosen for the risk curve because of their generally accepted higher credibility with respect to causal inference. However, when both descriptive and analytical studies were considered together for one polynomial fit, the results were almost identical. This exposure–effect curve will regularly be updated with respect to information from new studies. For practical application, the odds ratios for different noise levels are given in Appendix 1 to this chapter.

Alternatively, a fixed-effect meta-analysis of a linear trend was carried out *(21)*. It revealed an OR of 1.17 (95% CI 0.87–1.57, $P = 0.301$, $P(Q) = 0.943$).

Fig. 2.3. Polynomial fit of the exposure-response relationship for road traffic noise and the incidence of myocardial infarction



Source: Babisch (21).

Meta-analysis: road traffic noise and hypertension

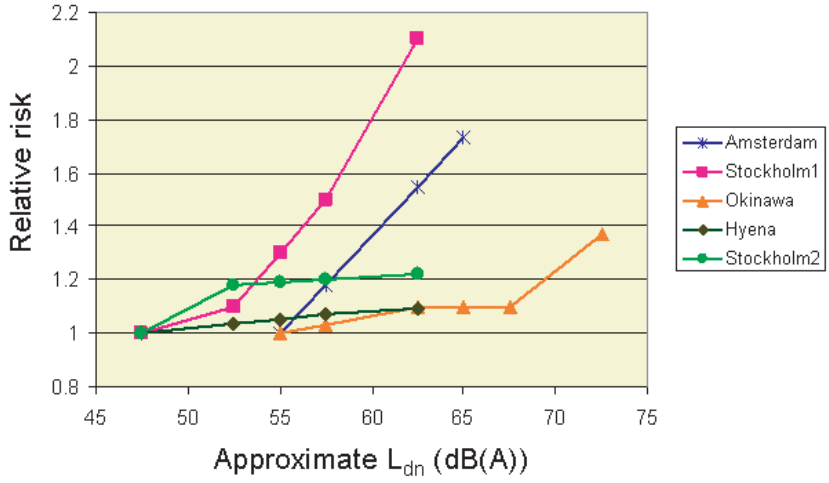
Regarding hypertension, a pooled estimate of the relative risk of 0.95 (95% CI 0.84–1.08) per 5-dB(A) increase in noise level during the day ($L_{day,16h}$ < 55–80 dB(A)) was calculated for the association between road traffic noise and hypertension based on a meta-analysis published in 2002 *(20)*. This estimate was recently updated based on new study results, and a pooled estimate of 1.12 (95% CI 0.97–1.30) was reported *(22)*. Significant results were found in two recently published studies, showing increases in the risk of hypertension of 1.05 (95% CI 1.00–1.10) per 5-

dB(A) increase in noise level (L_{24h} = 45–75 dB(A)) *(128)* and 1.38 (95% CI 1.06–1.80) per 5-dB(A) increase in the 24-hour noise level (L_{24h} ≈ 40–70 dB(A)) *(129)*, respectively. In a study looking at the combined effects of road traffic noise and air pollution on the prevalence of hypertension, the odds ratios for noise did not wane after adjustment for air pollution *(130)*.

Meta-analysis: aircraft noise and hypertension

The results of five studies on the relationship between aircraft noise and high blood pressure are shown in Fig. 2.4 *(128,131–135)*. The study subjects were men and women. A noise-level-related data pooling (categorical approach) was difficult to perform owing to the fact that different (national) exposure indices were used. Furthermore, different definitions of hypertension were applied. Individual odds ratios and confidence intervals were taken from summary reports and the original publications for this purpose to calculate regression coefficients of individual studies and odds ratios with respect to the weighted day/night noise indicator L_{dn}, which is supposed to be very similar to L_{den}. When the coefficients of a linear trend from the five studies were taken together ("regression approach"), the pooled estimate of the relative risk was 1.13 (95% CI 1.00–1.28) per 10 dB(A) for aircraft noise levels ranging between approximately 47 and 67 dB(A) *(136)*. The statistical test for heterogeneity of the studies was significant (P(Q) = 0.002). However, fixed and random effect estimates were the same. Owing to the results of new studies, this pooled effect estimate was smaller than that obtained from an earlier meta-analysis where the estimate of the relative risk was 1.59 (95% CI 1.30–1.93) per 10-dB(A) increase in the noise level *(20)*.

Fig. 2.4. Association between aircraft noise and the prevalence or incidence of high blood pressure



Source: Babisch & Van Kamp (136).

Disability weight

Different values of DW are used in the WHO comparative risk assessment reports by the different categories of epidemiological subregion that were defined based on geographical location and the level of infant and adult mortality (*7*).

The DW for acute myocardial infarction in the WHO EUR-A epidemiological subregion[2] is 0.405 *(7)*. However, disability weights of 0.108 and 0.186 are given for angina pectoris and congestive heart failure. No DW is given for ischaemic heart disease as a group. Hypertensive heart disease for the EUR-A epidemiological subregion is 0.201 but no DW is given for hypertension alone. In the literature, however, disability weights of 0.350 and 0.352 are reported for ischaemic heart disease as a group and for hypertension, and one year was considered for the duration of ischaemic heart disease and hypertension *(137)*.

EBD calculations

Two examples are given for calculating EBD from noise for cardiovascular disease. First, the exposure-specific approach is used to estimate the DALYs from myocardial infarction due to road traffic noise in Germany. Second, different noise exposure prevalence data are used to estimate the attributable fraction of myocardial infarction due to noise in Berlin.

Exposure-based approach for road traffic noise and myocardial infarction in Germany

An example is given for Germany regarding road traffic noise and myocardial infarction. These EDB calculations use an exposure-based approach. The country-specific population-attributable fraction (impact fraction) and the attributable cases can be calculated based on the distribution of the population in different exposure categories and the respective relative incidence of disease. This approach requires:

- a population-based estimate of the prevalence of the outcome in Germany obtained from surveys or national statistics;
- an estimate of the attributable fraction of the outcome caused by environmental noise, calculated from German estimates of exposure prevalence and Fig. 2.3; and
- a value of DW for each case of the outcome caused by environmental noise.

Prevalence of noise exposure

According to the older German noise exposure model (*Lärmbelastungsmodell*), it was estimated (reference year 1999) that approximately 16% of the German population were exposed to road traffic noise levels (taken at the facades of their houses) exceeding 65 dB(A) during the day ($L_{day,16h}$), that some 15% were exposed to 60–65 dB(A) and that approximately 69% were exposed to levels below 60 dB(A) *(138)*. The noise distribution is shown in Table 2.2. During the night, noise levels tend to be 7–10 dB(A) lower.

Attributable fraction calculation

By applying the polynomial equation of the exposure–response function (Fig. 2.3) to the noise exposure distribution of the German population, it is possible to calculate an attributable fraction (AF) for each exposure group, that is, the proportion of cases of myocardial infarction due to noise exposure.

2 The WHO EUR-A epidemiological subregion comprises Andorra, Austria, Belgium, Croatia, Cyprus, the Czech Republic, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, the Netherlands, Norway, Portugal, San Marino, Slovenia, Spain, Sweden, Switzerland and the United Kingdom.

The risk ratios attributed to the exposure categories are taken from Fig. 2.2. Using the formula of the population-attributable fraction (PAF) provides the following results:

$$PAF = \frac{(1.031 \cdot 0.153 + 1.099 \cdot 0.090 + 1.211 \cdot 0.051 + 1.372 \cdot 0.015 + 1 \cdot 0.691) - 1}{1.031 \cdot 0.153 + 1.099 \cdot 0.090 + 1.211 \cdot 0.051 + 1.372 \cdot 0.015 + 1 \cdot 0.691} = 0.0291$$

The resulting attributable fraction of myocardial infarction due to road traffic noise for the German population in the year 1999 is therefore 2.9%.

Table 2.2. Example: attributable fraction for myocardial infarction due to road traffic noise, estimated from the noise exposure pattern in Germany

Road traffic noise 1999, $L_{day,16h}$ (dB(A))	Percentage exposed	Relative risk	Attributable fraction
< 60	69.1	1.000	0.00
60–64	15.3	1.031	3.03
65–69	9.0	1.099	9.03
70–74	5.1	1.211	17.44
> 75	1.5	1.372	27.13

Cases of and deaths from myocardial infarction due to noise

According to the national health statistics, 849 557 cases of ischaemic heart diseases (ICD 9, No. 410–414), including 133 115 cases of acute myocardial infarction (ICD 9, No. 410), were diagnosed in 1999 *(139)*. The number of deaths due to myocardial infarction in Germany in 1999 was 76 961. So as not to double count cases when DALYs are calculated, the number of deaths was subtracted from the number of cases, leaving 56 154 new cases that did not result in death.

To calculate the cases due to traffic noise, the number of cases of myocardial infarction is multiplied by the attributable risk. Since there is no reason to believe that cases resulting in death should differ from those that do not with respect to noise exposure, the same attributable risk is applied to both groups of myocardial infarction cases.

The number of cases of non-fatal myocardial infarction (56 154) multiplied by 2.9% results in approximately 1629 new cases per year of non-fatal myocardial infarction in Germany attributable to traffic noise.

In addition, a proportion of deaths from myocardial infarction may also be attributable to traffic noise. Each of these deaths includes future YLL. Life expectancy at each age in 2002–2004 was used *(139)*. For each age group, the number of deaths due to myocardial infarction was multiplied by the life expectancy at that age separately for males and females. The total YLL for each sex was multiplied by 2.9% to give the YLL attributable to noise. This results in approximately 29 488 YLL.

Calculation of DALYs

To gain a rough estimate of the DALYs lost due to noise-related myocardial infarction for one year, the formulae in the previous chapter can be used:

DALY = YLL + YLD

where YLD = I · DW · L and YLL = number of deaths · average loss of life per death due to myocardial infarction.

Assuming one year of disability for each non-fatal case of myocardial infarction, the total DALYs are equal to:

29 488 + (1 629 · 0.405 · 1) = 30 147

This does not include ongoing morbidity after the first year.

Exposure-based approach for road traffic noise and myocardial infarction in Berlin

Another example, referring to the city of Berlin, is based on recent noise exposure data (L_{den} and L_{night}) derived from the strategic noise maps according to Directive 2002/49/EC *(113,140)*. The noise distribution is shown in Table 2.3 and it can be seen that the prevalences of exposure are lower than those in Table 2.2. Since Berlin is a metropolitan city where the noise exposure is likely to be higher than in smaller communities and rural areas, the data suggest that the traffic noise exposure in Germany, in general, is lower than estimated by the old *Lärmbelästigungsmodell (138)*. However, one has to consider that only the primary road network was assessed. On the other hand, traffic volumes of more than about 12 000 vehicles during the day (6:00–22:00) – corresponding to approximately L_{Aeq} = 65 dB(A) – are not very likely for the secondary road network. Applying the formula given above, the attributable fraction for Berlin is 0.0107, meaning that approximately 1.1% of all myocardial infarctions would be attributable to the road traffic noise in Berlin.

Table 2.3. Estimated road traffic noise exposure for the city of Berlin

Average sound pressure level, L_{den} (dB(A))	Number of citizens exposed [a]	Percentage exposed [b]	Relative risk of myocardial infarction [c]
Approx. < 55	2 683 449	80.53	1.000
> 55–59	220 200	6.61	1.000
60–64	155 000	4.65	1.015
65–69	140 200	4.21	1.067
70–74	112 600	3.38	1.161
> 75	20 800	0.62	1.302

[a] Numbers refer to the primary road network of Berlin.

[b] Total population of Berlin: 3 332 249 (2005).

[c] Odds ratios are derived from the polynomial risk equation for $L_{day,16h} = L_{den}$ - 2 dB(A).

Estimation of ischaemic heart disease burden from road traffic noise in the EU Member States

There is no international database on noise exposure of the European population covering the whole European Region. However, the Noise Observation and Information Service for Europe (NOISE) maintained by the European Environment Agency (EEA) and the European Topic Centre on Land Use and Spatial Information (ETC LUSI) on behalf of the European Commission provide noise exposure data that can be used for calculating disease burden in the western European countries. It contains data related to strategic noise maps delivered in accordance with EU Directive 2002/49/EC relating to the assessment and management of environmental noise (*141*). As for road traffic noise, the dataset covers the exposure distribution in approximately 20% of the total EU population as of January 2010. Bearing in mind that there are uncertainties and assumptions involved in using the exposure data based on strategic noise maps by the Member States (see below), we can use this official data to estimate burden of disease in the EU Member States.[3]

Table 2.4 summarizes the distribution of the population exposed to road traffic noise in agglomerations with more than 250 000 inhabitants, and relative risks and attributable fractions for respective exposure categories. The risk ratios attributed to different L_{den} categories are taken from Appendix 1 of this chapter. Applying the formula given above, the attributable fraction is 0.018, meaning that approximately 1.8% of all myocardial infarctions would be attributable to road traffic noise in these western European countries.

Table 2.4. Road traffic noise exposure for the European countries reporting noise maps

Road traffic noise within agglomeration L_{den} (dB(A))	Percentage exposed [a]	Relative risk [b]	Attributable fraction
< 55	50	1.000	0.00
55–59	17	1.000	0.00
60–64	19	1.015	1.48
65–69	9	1.067	6.29
70–74	4	1.161	13.87
> 75	1	1.302	23.20

Source: Noise Observation and Information Service for Europe *(141)*.

[a] The population size is 110 million living in agglomerations with > 250 000 inhabitants.

[b] The risk ratios attributed to different L_{den} categories are taken from Appendix 1 of this chapter.

3 Austria, Bulgaria, the Czech Republic, Denmark, Estonia, Finland, France, Germany, Hungary, Ireland, Italy, Latvia, Lithuania, the Netherlands, Norway, Poland, Romania, Slovakia, Slovenia, Spain, Sweden, Switzerland and the United Kingdom.

In 2008, WHO published an updated report on global burden of disease *(142)*. In this report, the DALYs for disease cluster categories were reported by different subregions based on income levels. High-income European countries[4] correspond to the EUR-A subregion with very low child and adult mortalities in the previous reports. DALYs of cardiovascular diseases are reported in the categories of rheumatic heart disease, hypertensive heart disease, ischaemic heart disease, cerebrovascular disease and inflammatory heart diseases. The total burden of ischaemic heart disease is 16 826 000 DALYs out of 883 million people in the WHO European Region, of which 3 376 000 DALYs are out of 407 million people in the high-income European countries. As DALYs for myocardial infarction were not published, we applied the above attributable fraction to the category of ischaemic heart disease. In other words, for the sake of DALY calculation, we assumed that road traffic noise has the similar impact on all ischaemic heart disease as on myocardial infarction. In high-income European countries, DALYs attributable to transport noise were estimated to be 60 768 years (1.8% of 3 376 000 DALYs) *(142)*.

Uncertainties, limitations and challenges

Biological plausibility of association

The biological plausibility of the hypothesis of noise effects is well-documented (see previous section summarizing the evidence). Acute noise effects have been studied extensively over the past 50 years, and a general noise reaction model was well-established before research moved from the laboratory to test hypotheses with respect to the long-term effects of noise in epidemiological studies.

The auditory system is continuously analysing acoustic information, which is filtered and interpreted by different cortical and sub-cortical brain structures causing acute responses of the autonomic nervous and the endocrine system, even during sleep. Long-term noise stress can adversely affect biological risk factors due to chronic dysregulation. Considering this pathway, noise must be viewed as an environmental risk factor. In epidemiological noise studies, higher risk estimates were found when length of exposure was considered (years in residence). The same accounts for room orientation and window opening habits (higher risks when rooms were facing the street with windows open). This is in accordance with the noise hypothesis and the effects of chronic noise stress (exposure effect).

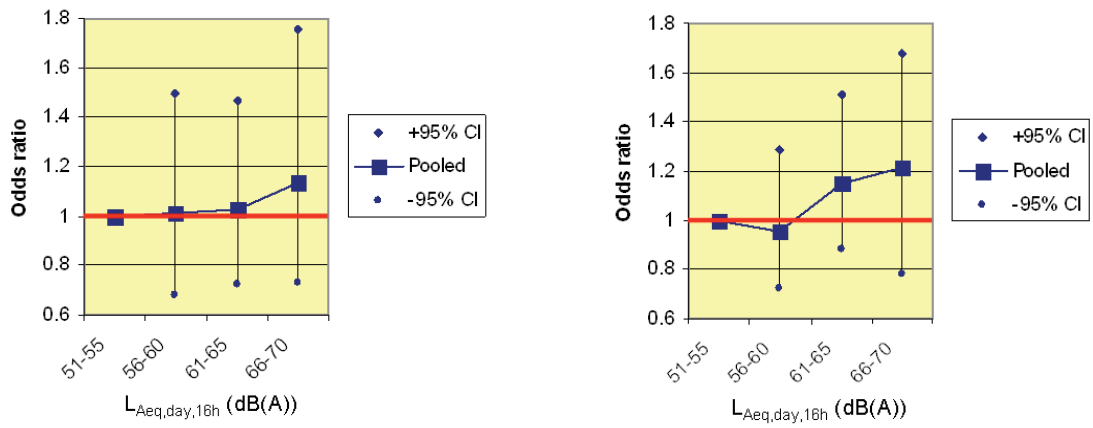
Generalization of myocardial infarction to other ischaemic heart diseases

Myocardial infarction was considered for the meta-analysis because it was the outcome most commonly assessed in the studies that met the inclusion criteria for the review. The noise impact on myocardial infarction may have been easier to detect by epidemiological studies, because misclassification in the diagnosis of myocardial infarction is less likely than for all ischaemic heart diseases. Ischaemic heart disease comprises: acute myocardial infarction, other acute and sub-acute forms of ischaemic heart disease, old myocardial infarction, ischaemic signs in the electrocardiogram, angina pectoris, coronary atherosclerosis and chronic ischaemic heart disease.

4 High-income European countries are: Andorra, Austria, Belgium, Cyprus, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, the Netherlands, Norway, Portugal, San Marino, Slovenia, Spain, Sweden, Switzerland and the United Kingdom.

Because there is no exclusive causal mechanism postulated specifically for myocardial infarction, it has been suggested that the impact fraction of traffic noise could be applied to all types of ischaemic heart disease. Therefore, the exposure–response curve for myocardial infarction could be generalized to all ischaemic heart diseases for the calculation of DALYs. This is supported by Fig. 2.5 and Fig. 2.6, which shows the association between road traffic noise level during the day ($L_{day,16h}$) and the prevalence of myocardial infarction and ischaemic heart diseases based on two studies, where all detailed information was assessed within each study *(126)*. It can be seen that the associations with the noise level look quite similar. The point estimate of pooled effect estimates for noise levels higher than 60 dB(A) are slightly higher for (all) ischaemic heart diseases than for myocardial infarction.

Fig. 2.5 & 2.6. Exposure-response curve for road traffic noise and the prevalence of myocardial infarction (Fig. 2.5, left) and all ischaemic heart diseases (Fig. 2.6, right)



Source of the data: Babisch et al. 1993 *(126)*

Specificity of hypertension as an outcome

Pooling of data is difficult when different criteria and assessment methods for the disease end-points were used in different studies. For example, with respect to hypertension, some aircraft noise studies refer to the former WHO criterion of a measured blood pressure of 160/100 mmHg, while others refer to the current WHO criterion of 140/90 mmHg. Perhaps more importantly, different determinants of high blood pressure were used, including self-reported doctor-diagnosed hypertension, anti-hypertensive drug medication, actual blood pressure measurements, or combinations of the three. The heterogeneity of the studies may be less of a problem with respect to the slope of the pooled exposure–response curve. However, decisions must be made regarding the onset (threshold) of the increase in risk. For the calculation of the attributable fraction, estimates of different scenarios can be made.

Generalization of evidence to both sexes

The exposure–response curve derived from male study subjects was generalized to women. The subjects in the noise studies were mostly men, owing to considerations of statistical power in the study design. Cardiovascular diseases are more frequent in middle-aged males *(143)*. For reasons of homogeneity, the relatively small number of females was excluded from the calculation of the pooled effect estimates.

The available results of noise studies do not allow for a distinction between the sexes. There is some indication that males may be more affected by road traffic noise *(125,128,144,145)* but contradictory results have also been found *(129)*. Studies on the association between environmental noise and high blood pressure showed no consistent pattern with respect to higher relative risks in either men or women *(18)*. In studies where females were considered, the hormonal/menopausal status was not assessed, which could act as a confounder (falsely showing differences between the sexes) *(146)*.

In laboratory studies, the focus was primarily on "before-after" effects of noise exposure in the same test subjects rather than on gender differences. In occupational noise studies, gender was often considered as a confounding factor but not as a potentially effect-modifying factor in the statistical analyses. Male blue collar workers were predominantly found in high-noise workplaces. Studies on the association between environmental noise and high blood pressure showed no consistent pattern with respect to higher relative risks in either men or women *(121)*.

Although there are differences in the absolute risk between males and females, it seems reasonable to assume that, in relative terms, females may be just as affected by noise stress as males. Nevertheless, in future noise studies, potential gender differences should be addressed.

Issues of statistical significance

The confidence intervals of the effect estimates shown in Fig. 2.1 and 2.2 for the association between traffic noise and myocardial infarction include relative risks of 1.0. The purpose of the meta-analysis was to derive a "best guess" pooled relationship for the calculation of population-attributable risks. Individual studies showed significant ($P < 0.05$) or borderline significant ($P < 0.10$) results when the highest exposure categories were combined and/or subsets of subjects with long years in residence were considered *(124,125)*. When the meta-analysis is carried out for subsamples of subjects that had lived for at least 10 or 15 years in their dwellings, larger effect estimates were also obtained in the meta-analysis *(21)*. For example, when the upper two noise categories of the exposure–response curve are combined, the pooled effect estimate is OR = 1.25 ($P = 0.068$) in the total sample, and OR = 1.44 ($P = 0.020$) in the sub-sample, the latter being statistically significant. Regarding linear trend, the odds ratio in the sub-sample of subjects with many years of residence was 1.44 per 10-dB(A) increase in the noise level (CI 0.97–2.12, $P = 0.067$), which was borderline significant. However, for the calculation of population-attributable risk percentages, the weaker effect estimates were considered to apply to the entire study populations, because information about modifiers of exposure such as length of residence or window/room orientation will not be available for general populations. Depending on the results of new studies, the current risk curves must be regularly updated.

Lack of exposure data

The lack of accurate exposure data is a major hindrance in estimating actual burden of disease. How can exposure data from countries and subregions be obtained? EU Member States have just started to systematically assess the environmental noise due

to road, rail and air traffic and commercial/industrial activities in their communities according to EU Directive 2002/49/EC *(113)*. The noise mapping data for Directive 2002/49/EC can be used as shown above. It should be noted that the application of the exposure data for the urban population to the total population in the EU may lead to overestimation of burden. To avoid this possibility, we extrapolated only to agglomerations with > 50 000 inhabitants (57% of the EU population). The accuracy and representativeness of exposure data will improve when the second round of noise mapping produce data from agglomerations with 100 000–250 000 inhabitants in 2012. Exposure data will be still sparse from the WHO EUR-B[5] and EUR-C[6] epidemiological subregions. Extrapolation of exposure data from EUR-A to the EUR-B and EUR-C epidemiological subregions might be problematic because the level of noise exposure of the population might be quite different between these subregions.

Road traffic is a key environmental noise source. However, results from epidemiological studies with respect to the association of other environmental noise sources (such as air traffic noise, railways or even leisure noise) with myocardial infarction are rarely available. For the time being, the exposure–response curve derived for road traffic noise could be used, considering that at the same average noise level, aircraft noise tends to be more annoying and conventional railway noise less annoying than road traffic noise *(119,147)*. Furthermore, exposure misclassification diluting the true effects is less of a problem with respect to aircraft noise because all sides of the house are equally exposed. (*Note*. According to Directive 2002/49/EC, noise levels refer to the most exposed side of a dwelling.) The characteristics of road traffic noise and its effects can be quite different from rail and aircraft noise, which is an additional source of uncertainty when applying road noise curves to other noise sources and vice versa.

Confounding with air pollution

Air pollutants have also been shown to be associated with cardiovascular end-points *(148–155)*. In real life, individuals exposed to road noise are also likely to be exposed to air pollution arising from road traffic. It is not yet clear whether the impact of noise on ischaemic heart disease is independent, additive or synergistic to the impact of outdoor air pollution. Air pollution studies have not controlled for noise and vice versa. Air pollution epidemiology carried out in the last century focused primarily on respiratory illness, which was not an issue in noise research. However, cardiopulmonary mortality was also identified as a key outcome of acute and chronic exposure to air pollutants.

Most information on hospital admissions due to acute changes (increases) in levels of air pollutants come from time-series studies *(150)*. Studies on short-term exposure to elevated concentrations of fine particulate matter are associated with acute changes in cardiopulmonary health. However, since traffic volume does not show

5 The WHO EUR-B epidemiological subregion comprises Albania, Armenia, Azerbaijan, Bosnia and Herzegovina, Bulgaria, Georgia, Kyrgyzstan, Montenegro, Poland, Romania, Serbia, Slovakia, Tajikistan, the former Yugoslav Republic of Macedonia, Turkey, Turkmenistan and Uzbekistan.

6 The WHO EUR-C epidemiological subregion comprises Belarus, Estonia, Hungary, Kazakhstan, Larvia, 6 Lithuania, the Republic of Moldova, the Russian Federation and Ukraine.

considerable day-to-day variations, the changes in air pollution in these studies are due to other factors that affect the concentration of air pollutants, mainly changes in weather conditions. Noise levels in urban environments, on the other hand, are primarily determined by the relatively constant traffic volume per day, and much less by weather conditions when the distance of houses from the street is short (urban noise). In this respect, confounding between noise and air pollution is not likely with respect to short-term effects in time-series studies.

The health effects of noise in general refer to long-term chronic noise stress. Confounding can be an issue in long-term effects observed by cross-sectional, case-control and cohort studies. Epidemiological studies have shown strong associations of mortality and life expectancy with long-term exposure to fine particulate matter and sulfates *(156)*. However, the study designs of cohort studies on the association between air pollutants and cardiopulmonary mortality differ considerably from those of noise exposure. In air pollution studies, the spatial exposure is often considered on an ecological basis. Subjects from different metropolitan areas with different mean (background) concentrations of air pollutants have been compared with respect to disease occurrence. No distinction is usually made between busy streets and side streets *(148,149,152,157)*. In noise studies, the exposure in front of a study participant's house was assessed on an individual level with respect to nearby sound sources, along with individual confounding factors. Differences of 1:100 (20 dB(A)) in terms of sound intensity are common for people living in different streets or even only a few yards away from one another, because shielding is highly effective for noise. The sound level can diminish from the front to the back of a house by 30 dB(A) or more (sound intensity 1:1000). To some extent, one could say that major air pollution studies refer to macro-scale exposures while noise studies refer to micro-scale exposures.

Further, cardiovascular effects of noise (hypertension) were also found for noise sources where air pollutants are less likely to be co-varying factors, e.g. occupational noise *(20)* and aircraft noise *(121)*. It was shown that the relative contribution of airport operations to the emission levels of nitrogen oxides, carbon monoxide, sulfur dioxide, volatile organic compounds and black smoke was small compared to the background concentrations in the vicinity of an airport *(158)*. In spite of this obvious co-exposure, there was a lack of interaction between the scientific community dealing with the health impacts of noise and that dealing with air pollution. However, this has changed in recent years and studies on their combined effects are currently under way *(130,159,160)*. Some studies have used the distance to major roads as a surrogate for exposure to air pollutants. However, noise would be as good an explanation for the observed effects *(161–165)*.

Method of calculating the exposure–response relationship

Different approaches have been used to calculate pooled effect estimates and exposure–response relationships. These include the "regression approach" and the "categorical approach". In the regression approach, the slopes (regression coefficients) across all noise categories of each noise study are pooled to assess a common regression coefficient. In the categorical approach, the relative risks found for the same

noise category in each noise study are pooled and considered for the calculation of an exposure–response curve. The regression approach has the advantage that regression coefficients can be pooled regardless of actual noise levels; only the slope (regression coefficient) of the exposure–response relationship is taken into account. The categorical approach is noise-level oriented. Possible thresholds of effects can be determined, and it is less likely to obscure possible non-linear associations, but it requires comparable exposure indicators of the studies considered in the meta-analysis. Often both, trend and categorical contrast analyses are carried out simultaneously (128).

Conclusions

The noise indicators used for noise mapping in the EU can – in principle – be used for a quantitative risk assessment regarding cardiovascular risk if exposure–response relationships are known. Only two end-points – hypertension and ischaemic heart disease – should be considered at this stage. If necessary, different exposure–response curves could be used for different exposures. Some studies showed that associations between noise level and cardiovascular outcomes were stronger with respect to noise exposure at night *(128,166,167)*. In this respect, it can be useful to consider different exposure–response relationships for day and night noise, particularly if the exposed side of the house is considered for exposure assessment. For practical reasons, attempts should be made to reduce the set of necessary exposure–response curves to a minimum. The noise indicator L_{den} may be useful for assessing and predicting annoyance in the population. However, non-weighted day and night noise indicators may be more appropriate for health-effect-related research and risk quantification. It is a matter for future research to determine how the integrated noise indicator L_{den} performs in noise studies, particularly with respect to noise sources (railways, aircraft) other than road traffic where the differences between day and night noise are less uniform and depend on location and other circumstances (e. g. night noise regulations).

We adopted conservative assumptions whenever necessary. One exception was to extrapolate the exposure data from urban population to the whole population of the EU. This was necessary because of a lack of exposure data for the rural population as of 2010. Considering the advanced level of urbanization in western Europe and the bias toward the null in the estimation of relative risks due to random misclassification of exposure, the overall impact of overestimation due to extrapolation might be minimal. Nevertheless, it is desirable to use exposure data for the whole population when it is available.

We have to learn to live with uncertainties *(168,169)*. Nevertheless, "no exposure data" does not mean "no exposure" and "no scientific evidence" does not mean "no effect" *(170)*. Using the precautionary principle, decisions can be made based on best available data *(171,172)*. Future epidemiological noise research will need to focus on vulnerable groups, effect modifiers, sensitive hours of the day, coping mechanisms, differences between noise sources, possible confounding with air pollution, differences between objective (noise level) and subjective (noise perception) exposure, and multiple exposures (home, work and leisure environments).

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Appendix 1. Exposure-response curve (polynomial fit) of the association between road traffic noise and incidence of myocardial infarction

$$OR = 1.629657 - 0.000613 \cdot (L_{day,16h})^2 + 0.000007357 \cdot (L_{day,16h})^3$$

$L_{day,16h}$	L_{den}*	OR
55	57	1
55.5	57.5	1
56	58	1
56.5	58.5	1
57	59	1
57.5	59.5	1.002
58	60	1.003
58.5	60.5	1.005
59	61	1.007
59.5	61.5	1.009
60	62	1.012
60.5	62.5	1.015
61	63	1.019
61.5	63.5	1.022
62	64	1.027
62.5	64.5	1.031
63	65	1.036
63.5	65.5	1.042
64	66	1.047
64.5	66.5	1.054
65	67	1.06
65.5	67.5	1.067
66	68	1.074
66.5	68.5	1.082
67	69	1.091
67.5	69.5	1.099
68	70	1.108
68.5	70.5	1.118
69	71	1.128
69.5	71.5	1.138
70	72	1.149
70.5	72.5	1.161
71	73	1.173
71.5	73.5	1.185
72	74	1.198
72.5	74.5	1.211
73	75	1.225
73.5	75.5	1.239
74	76	1.254
74.5	76.5	1.269
75	77	1.285
75.5	77.5	1.302
76	78	1.318
76.5	78.5	1.336
77	79	1.354
77.5	79.5	1.372
78	80	1.391
78.5	80.5	1.411
79	81	1.431
79.5	81.5	1.452
80	82	1.473

*Approximation: $L_{den} = L_{Aeq,16h} + 2$ dB

3. ENVIRONMENTAL NOISE AND COGNITIVE IMPAIRMENT IN CHILDREN

Staffan Hygge
Rokho Kim

It has been suspected for many years that children's learning and memory are negatively affected by noise. Over 20 studies have shown negative effects of noise on reading and memory in children *(1,2)*: epidemiological studies report effects of chronic noise exposure and experimental studies report acute noise exposure. Tasks affected are those involving central processing and language, such as reading comprehension, memory and attention *(3–6)*. Exposure during critical periods of learning at school could potentially impair development and have a lifelong effect on educational attainment.

Evidence from recent well-controlled epidemiological studies with representative samples of children has also made it possible to start to quantify the magnitude of noise-induced impairment on children's cognition and identify the relative contribution of different sources of noise. Children may be exposed to noise for many of their childhood years and the consequences of long-term noise exposure on reading comprehension and further cognitive development remain unknown. Such quantifications, albeit initially crude, will in the long run help to estimate and quantify how much cognitive development individual children could be expected to lose because of noise, and the economic impact of this for learning in schools. In turn, such estimates will be also of value for making projections on the societal level, including political decision about any sociodemographic redistribution of noise exposure. On the other hand, exposure–response curves can also be used for social engineering decisions about how much of an improvement, and for whom, can be expected from a reduction in noise levels.

This chapter attempts to contribute to this general goal by placing the negative effects of noise on children's cognition into the risk assessment context.

Definition of outcome

Cognitive impairment is not an outcome of a clinical diagnosis; it is therefore not possible to derive a conventional exposure–risk relationship suitable for calculating burden of disease. Lopez et al. *(7)* defined cognitive impairment as "delayed psychomotor development and impaired performance in language skills, motor skills, and coordination equivalent to a 5- to 10-point deficit in IQ". Contemporaneous cognitive deficit is defined as "reduction in cognitive ability in school-age children, which occurs only while infection persists".

These definitions are not helpful and not readily applicable to the studies reported on noise and cognition in children. None of the studies has explicitly employed IQ as an end-point and the confining of any reduction in cognitive ability to the duration of the noise exposure is too restrictive. Therefore, our case definition of noise related cognitive impairment is:

> Reduction in cognitive ability in school-age children that occurs while the noise exposure persists and will persist for some time after the cessation of the noise exposure.

A notable characteristic of this definition is that the cognitive impairment is assumed to show itself during the noise exposure as well as some time after the exposure has stopped.

Summary of evidence linking noise and cognitive impairment in children

The extent to which noise impairs cognition, particularly in children, has been studied with both experimental and epidemiological designs. The epidemiological studies report effects of chronic noise exposure and the experimental studies of acute noise exposure. The studies relevant to children's cognition are not many and do not always meet strict methodological criteria. Nevertheless, there are three recent studies that meet basic methodological quality criteria and are also comparable with each other in terms of the cognitive functions measured.

One of the most compelling studies in this field is the naturally occurring longitudinal quasi-experiment reported by Evans and colleagues, examining the effect of the relocation of Munich airport on children's (9–10 years, N = 326) health and cognition *(8–10)*. In 1992, the old Munich airport closed and was relocated. Prior to relocation, high noise exposure was associated with deficits in long-term memory and reading comprehension. Two years after the closure of the airport, these deficits disappeared, indicating that effects of noise on cognition may be reversible if exposure ceases. Most convincing was the finding that deficits in the very same memory and reading comprehension tasks developed over a two-year follow-up in children who became newly exposed to noise near the new airport.

The recent large-scale RANCH study, which compared the effect of road traffic and aircraft noise on children's (9–10 years, N = 2844) cognitive performance in the Netherlands, Spain and the United Kingdom, found a linear exposure–effect relationship between long-term exposure to aircraft noise and impaired reading comprehension and recognition memory, after taking a range of socioeconomic and confounding factors into account *(11)*. No associations were observed between long-term road traffic noise exposure and cognition, with the exception of episodic memory, which surprisingly showed better performance in high road traffic noise areas. Neither aircraft noise nor road traffic noise affected attention or working memory.

A study of ambient noise exposure (predominantly road and rail sources) of fourth-grade children living in the Tyrol mountain region compared three cognitive measures for schoolchildren (mean age 9–7 years, N = 123) exposed to 46 or 62 dB(A) L_{dn}. The two sociodemographically homogeneous samples differed only in their noise exposure range (M = 46.1 L_{dn} vs M = 62 L_{dn}). Long-term noise exposure was significantly related to both intentional and incidental memory. The improvement in cognitive performance in the quieter group was estimated at 0.5% (recall prose and recognition) to 1% (free recall) per dB. The authors note that the magnitude of the effects shown was smaller than those uncovered in earlier airport noise studies.

Both the RANCH and Tyrol studies indicate that aircraft noise may be worse for cognition than road traffic noise. For aircraft noise, exposure evidence from the Munich study seems to indicate that L_{Aeq} = 60 may be a dividing line, but the RANCH study results suggest more of a linear association between aircraft noise exposure and impairment of reading comprehension. For ambient road and rail noise, the Tyrol study suggests that effects occur around L_{dn} = 60.

Other field studies of children have had some methodological limitations, which make them less relevant as evidence. For example, the testing of cognitive capacities took

place in noisy conditions for the noise-exposed and in quieter conditions for the children in the control groups. Testing in silent conditions would have been preferred, in order to compare the noise effect on memory and learning between exposure and control groups *(12–16)*. Also, for some studies, the sociodemographic variables and different reading curricula between the schools were not fully adjusted or controlled for.

Experimental studies of the impact of acute noise exposure on reading and memorizing new material are generally not as vulnerable to selection biases as epidemiological studies. Memory tests are made in silence of material that was read in noise. Participants are randomized to exposure and control groups, and children are sampled from sociodemographically comparable schools. To a certain extent, there is comparability between the memory and reading tests employed in the experimental studies and the field studies (the Munich and RANCH studies), even though the field studies concern chronic noise exposure and the second set acute noise exposure.

Exposure–response relationship

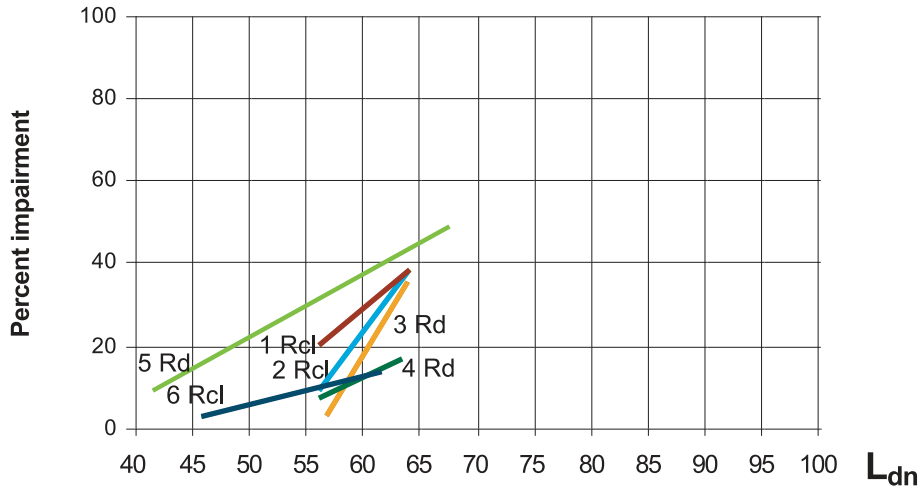
Only the Tyrol study *(17)* has used the noise indicator L_{dn}. The Munich study used $L_{eq,24h}$ and the RANCH study predominantly used $L_{eq,16h}$. The L_{dn} and L_{eq} metrics are not directly equivalent: L_{dn} is always equal to or larger than L_{eq}, with the following differences between L_{dn} and L_{eq} (T. Gjestland, personal communication, 2006):

- evenly distributed traffic flow, + 6.4 dB
- evenly distributed 07:00–22:00, no night traffic, + 1.9 dB
- 10% of traffic during 22:00–07:00, + 2.9 dB.

Although it is not clear which noise metric is the most adequate, L_{dn} may be more appropriate for the measurement of noise effects on cognition for some specific noise sources. For example, for aircraft noise exposure, the RANCH study found that both school $L_{eq,16h}$ and home $L_{eq,8h}$ (so a comparison of daytime noise exposure at school and nighttime noise exposure at home) had a similar detrimental effect on reading comprehension scores. These findings suggest that a measure such as L_{dn}, which combines daytime and nighttime exposure, would be appropriate for examining the effects of aircraft noise on cognition. However, this issue may be more complicated for other noise sources. For cognition, the fact that children spend the daytime at school and the nighttime at home needs to be taken into consideration. Aircraft noise exposure at school and home were highly correlated in the RANCH study, which could account for the similar effect on cognition for the daytime and nighttime measures. Road traffic noise at home and school were less highly correlated, suggesting that exposure measures that cover the 24-hour period may be less reliable in detecting cognitive effects and could be associated with error.

Fig. 3.1 shows the exposure–response curves from the different epidemiological studies. This can be summarized in quantitative terms: for the field studies in Fig. 3.1, memory recall and reading have average slopes of around 2% per L_{dn}, as calculated by the mean of the slopes of the six lines. Thus, for recall and reading, it is expected that a reduction of the chronic noise level by 5 L_{dn} would result in improved performance by 10%. As noted above, the only available road traffic noise study *(17)* had a less steep slope. The fact that we do not have much data from road traffic noise exposure set a limit to the generality of our conclusion, but the results of studies on aircraft noise, albeit few, are nevertheless consistent.

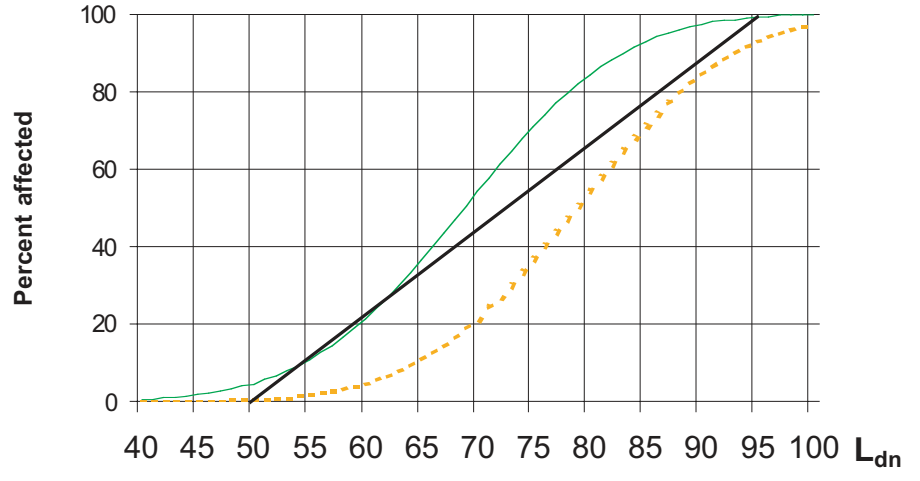
Fig. 3.1. Exposure-response curves from different epidemiological studies



Notes. Rd = reading; Rcl = memory, recall
1 = recall, children, old airport *(10)*.
2 = recall, children, new airport *(10)*.
3 = reading, children, old airport *(10)*.
4 = reading, children, new airport *(10)*.
5 = reading, children *(11)*.
6 = free recall, children *(17)*.

To obtain the exposure–response relationship, we need to use the information above to determine an approximate curve. Assuming that 100% of those exposed to noise are cognitively affected at the very high noise levels, e.g. 95 L_{dn}, and that none are affected at a safely low level, e.g. 50 L_{dn}, a straight line (linear accumulation) connecting these two points, as in Fig. 3.2, can be used a basis for approximations. This straight line is an underestimation of the real effect, since for theoretical reasons based on an (assumed) underlying normal distribution, the true curve should have the same sigmoidal function form as the two curves in Fig. 3.2. Within the noise exposure bracket 55–65 L_{dn}, the straight line and the solid line sigmoidal distribution agree on approximately 20% impairment. In the bracket 65–75 L_{dn}, the number should be in the range of 45–50% and above 75 L_{dn} in the range of 70–85%.

Fig. 3.2. Hypothetical exposure-risk curves and estimated percentage of affected people



Disability weight

Lopez et al. *(7)* suggested DWs for different cognitive impairments ranging from 0.468 (e.g. Japanese encephalitis) or 0.024 (e.g. as a result of iron deficiency anaemia). Contemporaneous cognitive deficit was given a DW of 0.006. Thus, this is a very conservative choice to go with the definition of contemporaneous cognitive deficit and a DW of 0.006 in estimates of the noise-related impairment of children's cognition.

There would be no mortality due to cognitive impairment, so estimation of YLD per year will be sufficient to estimate the total DALYs.

EBD calculations

Two examples are given. First, the exposure-specific approach is used to calculate the burden of disease from cognitive impairment due to noise in children aged 7–19 years in Sweden. And second, the values estimated in the first example are extrapolated to all of the WHO EUR-A epidemiological subregion *(7).*

Note that the calculations rest on the assumption that the noise effects are there only when people are exposed. There is no assumption made that the inflicted noise-induced disability lasts longer than the noise exposure. It would not be unreasonable to set a case also for lasting cognitive effects of noise after the cessation of exposure, but that has explicitly not been done here.

Exposure-specific approach to environmental noise and cognitive impairment in Swedish children

For the first example, the exposure-specific approach is used to calculate the burden of cognitive impairment due to environmental noise in children aged 7–19 in Sweden. This approach requires:

- the distribution of the prevalence of exposure to environmental noise within the population from EU data;
- the exposure–response relationship between noise and the outcome from Table 3.1; and
- a value of DW for each case of the outcome caused by environmental noise.

Prevalence of noise exposure

There are no relevant figures for how many children are exposed to different noise levels. What are available are estimates of the percentage of people exposed to noise at different levels in the EU. For instance, Roovers et al. *(18)* stated that around 68% are exposed to L_{dn} levels < 55, 19% to 55–65, 11% to 65–75 and 2% to > 75. This is shown in Table 3.1, although statistics for the specific countries within geographical regions such as the EU may vary *(19)*.

The noise exposure distribution shown in Table 3.1 is for adults, but there is no reason to believe that the exposure distribution for children is very different. If there is a difference in noise exposure levels, children are more likely than adults to be exposed to noise.

To calculate the number of children exposed to the noise levels that meet the criterion of cognitive impairment, the age distribution in the population must be consid-

ered. In Sweden, 23.9% of the population are aged under 20 years and 16.53% were in the age range of the mandatory school system in 2004. In 2004, there were 1 489 437 school-aged children in Sweden. It can be noted that the proportion of the population up to 19 years (23.95%) fits closely with the 24.2% for the EU in 1998 *(19)*.

Table 3.1. Percentage of the population exposed to various levels of noise (L_{dn}) and calculated number of exposed children aged 7–19 years

Noise level (L_{dn})	Population exposed	Number of children exposed
< 55	68%	1 012 817
55–65	19%	282 993
65–75	11%	163 838
> 75	2%	29 789
Total	100%	1 489 437

Source: Roovers et al. *(18)*.

Number of cases of and YLD from cognitive impairment caused by environmental noise

Combining the number of children exposed (Table 3.1) with the likelihood of cognitive impairment if exposed (Fig. 3.2), the number of children with noise-induced cognitive impairment can be calculated. To estimate YLD due to the cognitive impairment, this number is multiplied by the DW of 0.006 (Table 3.2).

Table 3.2. Estimated number of children aged 7–19 years in Sweden with noise-induced cognitive impairment and DALYs per year due to noise-induced cognitive impairment (NICI)

Age group and noise exposure level	No. of children aged 7–19 exposed	Percentage of children who will develop NICI	No. of children with NICI	DALYs lost for NICI
7–19 years, < 55 L_{dn}	1 012 817	0	0	0.0
7–19 years, 55–65 L_{dn}	282 993	20	56 599	339.6
7–19 years, 65–75 L_{dn}	163 838	50	81 919	491.5
7–19 years, > 75 L_{dn}	29 789	75	22 342	134.1
Total	1 489 437		160 859	965.2

According to our estimates, there are 160 859 Swedish children aged 7–19 (point prevalence) who could be cognitively impaired to the extent of DW 0.006. This can also be considered equivalent to 160 859 years lived with this disability in 2004. This amounts to 965 YLD for noise-induced cognitive impairment in Swedish children aged 7–19 years. This estimate is based on the conservative assumption that noise effects on cognitive impairment and childhood learning are temporary.

Exposure-specific approach for environmental noise and cognitive impairment in children in the EUR-A epidemiological subregion

The noise exposure figures in Table 3.1 were taken to be representative for Europe, and the distribution of children aged 7–19 years of age in Sweden is close to that reported for Europe as a whole. Therefore, the number of DALYs per million children aged 7–19 in the EUR-A countries can be calculated (Table 3.3). The absolute DALY for the EUR-A countries, with an estimated total population of 420 503 million, is therefore 45 036.

Table 3.3. Estimated DALYs per year per million children aged 7–19 in the EUR-A epidemiological subregion

Age group and noise exposure level	Percentage of population exposed to noise level	Percentage of population who will develop cognitive impairment	Number impaired per million	DALYs lost per million
7–19 years, < 55 L_{dn}	11.24	0	0	0.0
7–19 years, 55–65 L_{dn}	3.14	20	6 281	37.7
7–19 years, 65–75 L_{dn}	1.82	50	9 090	54.5
7–19 years, > 75 L_{dn}	0.33	75	2 475	14.9
All other age groups	83.47	0	0	0.0
Total	100.00		17 846	107.1

Uncertainties, limitations and challenges

Source of noise

The slopes reported in Fig. 3.1 are for aircraft noise only. In contrast to the Munich study, which focused on aircraft noise, the RANCH study also included road traffic noise. But for road traffic noise, there was no indication of a significant impairment of children's cognition. As an explanation, the authors pointed out that aircraft noise, because of its intensity, the location of the source, and its variability and unpredictability, is likely to have a greater effect on children's reading than road traffic noise, which might be of a more constant intensity. Thus, it is conceivable that aircraft noise is more damaging than road traffic noise for children's cognition. This may also be true when the L_{dn} level is controlled for, which has been reported for children's memory in an experimental acute noise study *(20)*.

Even though there may be a degree of difference between aircraft and road traffic noise, acting on the safety principle would suggest treating them as equally damaging to children's cognition and to assume that there is approximately the same response effect regardless of noise source. This may, however, tend to overestimate the effects of road traffic noise.

Design of epidemiological studies

It should be noted that the RANCH study was a cross-sectional study in contrast to the prospective, longitudinal Munich study. This may make the Munich study more powerful in picking up unconfounded cause–effect relationships between noise exposure and outcomes.

Possibility of long-term cognitive impairment from chronic noise exposure

The DALYs calculated in Table 3.2 have not taken into account any lasting or long-standing impairment of cognitive functioning that could occur as a result of long-term noise exposure. Our calculations are restricted to the period in children's life when they attend primary school, assuming that the impacts of noise are negligible on the cognitive function of adults. This assumption is very conservative, however, because it is more likely that children who have passed through the mandatory school system in a noisy environment would live with a long-term consequence of

cognitive impairment. They are also more likely to live in a noisy environment even after the schooling period, which is more likely for children who go to school in areas exposed to aircraft noise. It would be realistic to assume that the impaired cognitive function will carry over to the years after the schooling period. If future studies provide an estimation of the severity and the duration of such chronic effect of noise on cognitive function, the calculation of DALYs should be updated.

Assumption of the duration of the impact

There is some evidence from the Munich study *(10)* that after the cessation of exposure to aircraft noise, children (age 9–11 years) recover within 18 months to the cognitive performance levels of their year-mates who were not exposed to much aircraft noise. Thus, it is possible that, at least for young children, chronic noise effects are reversible and that the DWs will diminish with increasing age. However, we assumed in our calculation that the effects are temporary and recovery is quicker, yielding YLD values that are conservative.

Assumption of the exposure–risk relationship

As pointed out above, with reference to the linear and sigmoidal accumulation of effects in Fig. 3.2, we have most likely not overestimated the fractions of children affected in the noise exposure ranges 65–75 L_{dn} (50%) and > 75 L_{dn} (75%). Further, we might have underestimated the average DW (0.006) for those affected by the higher level of noise. These two conservative assumptions may have led to a significant underestimation of the real DALYs in the EUR-A epidemiological subregion given in Table 3.3. For example, if DW doubles and quadruples to 0.012 and 0.0024 in the exposure brackets 65–75 L_{dn} and > 75 L_{dn}, respectively, the DALYs will be much greater than shown in Table 3.3.

Policy considerations

An alternative to viewing the noise-induced cognitive impairment of children from a burden-of-disease perspective is to analyse the impairment in terms of wasted learning units. The learning units could be given a monetary value in wasted teaching hours in schools – wasted for the teachers, the pupils and society. Therefore, the societal impact will probably be larger than the impact reflected by DALYs, which solely estimate the impact on specific cognitive impairment. A calculation of wasted learning units instead of DALYs is probably a more complicated task, with many more uncertain parameters. For the time being, DALYs from noise-induced impairment of cognition in children, together with DALYs from other environmental risks, may provide evidence for prioritizing policy options, such as lowering recommended noise levels in control guidelines for schools and learning.

Conclusions

Reliable evidence indicates the adverse effects of chronic noise exposure on children's cognition. There is no generally accepted criterion for quantification of the degree of cognitive impairment into a DW. However, it is possible to make a conservative estimate of loss in DALYs using the methods presented in this chapter. It is important to consider the assumptions, uncertainties and limitations in the methods when interpreting the estimated values of EBD.

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4. ENVIRONMENTAL NOISE AND SLEEP DISTURBANCE

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Sleep disturbance is one of the most common complaints raised by noise-exposed populations, and it can have a major impact on health and quality of life. Studies have shown that noise affects sleep in terms of immediate effects (e.g. arousal responses, sleep stage changes, awakenings, body movements, total wake time, autonomic responses), after-effects (e.g. sleepiness, daytime performance, cognitive function deterioration) and long-term effects (e.g. self-reported chronic sleep disturbance).

Sufficient undisturbed sleep is necessary to maintain performance during the day as well as for general good health *(1)*. The human organism recognizes, evaluates and reacts to environmental sounds even while asleep *(2)*. These reactions are part of an integral activation process of the organism and express themselves as, for example, changes in sleep structure or increases in heart rate. Although they are natural (and even necessary) reactions to noise, it is assumed that a substantial increase in the number of such effects constitutes a health issue. Environmental noise may reduce the restorative power of sleep by means of repeatedly occurring activations (so-called sleep fragmentation). Acute and chronic sleep restriction or fragmentation has been shown to affect, among other things, waking psychomotor performance *(3)*, memory consolidation *(4)*, creativity *(5)*, risk-taking behaviour *(6)*, signal detection performance *(7)* and risks of accidents *(8,9)*.

There is an ample number of laboratory and field studies that provide sufficient evidence to conclude that traffic noise causally and relevantly disturbs sleep and, depending on noise levels, may impair behaviour and well-being during the subsequent period awake *(10–22)*. Although clinical sleep disorders (e.g. obstructive sleep apnoea, which is a sleep disorder characterized by pauses in breathing during sleep) have been shown to be associated with increased risks for cardiovascular disease, little is known about the long-term effects of noise-disturbed sleep on health. However, recent epidemiological studies do suggest that nocturnal exposure to traffic noise increases the risk of cardiovascular disease *(23–25)*.

In this chapter, available exposure–response relationships for various sleep disturbance indicators are discussed. Subsequently, a method for estimating the burden of self-reported sleep disturbance due to noise is proposed and illustrated.

Definition of outcome

Sleep disturbances can be measured electrophysiologically, using so-called polysomnography (PSG), or with self-reporting in epidemiological studies using survey questionnaires. PSG, i.e. the simultaneous recording of the electroencephalogram (EEG), the electrooculogram (EOG), the electromyogram (EMG)

and other physiological variables, remains the gold standard for measuring and evaluating sleep. According to specific conventions *(26,27)*, the night is usually divided into 30-second epochs. Depending on EEG frequency and amplitude, specific patterns in the EEG, muscle tone in the EMG and the occurrence of slow or rapid eye movements in the EOG, different stages of sleep are assigned to each epoch. Wake, superficial sleep stages S1 and S2, deep sleep stages S3 and S4, and REM (rapid eye movement) sleep are differentiated. Current knowledge assumes that sleep stages differ in their function and in their relevance for sleep recuperation, where continuous periods of deep sleep and REM sleep seem to be especially important for sleep recuperation *(4)*. Shorter activations in the EEG and EMG, so-called arousals, can also be detected with polysomnography *(26,28)*. These arousals are usually accompanied by activations of the autonomic nervous system (e.g. increases in heart rate and blood pressure) and they may contribute to sleep fragmentation *(29,30)*. Further, motility (i.e. body movement during sleep) has been found to be a relatively easy to use and sensitive measure for sleep disturbance, and has been shown to be a predictor of effects such as awakening and self-reported sleep quality *(22)*. Depending on their frequency, acute noise effects on sleep (arousals, awakenings, body movements) cause a general elevation of the organism's arousal level that consequently leads to a redistribution of time spent in the different sleep stages, with an increase of the amounts of wake and stage S1 and a decrease of slow wave sleep (SWS) and REM sleep *(16,31–33)*.

In epidemiological studies, "self-reported sleep disturbance" is the most easily measurable outcome indicator, because physiological measurements are costly and difficult to carry out on large samples and may themselves influence sleep. However, since during most of the night the sleeper is not aware of himself or his surroundings, the process of falling asleep and longer wake periods during the night contribute disproportionately to subjective estimates of sleep quality and quantity, which may therefore differ substantially from objective measures *(34)*. Nevertheless, self-reported sleep disturbance may have validity in its own right by reflecting the impact on sleep as perceived by the subject over a longer period of time.

In surveys asking about sleep disturbance, responses can be graded on a scale from 0 to 100. On this scale, similar to definitions of noise annoyance, cut-off values were chosen of 50 and 72 to determine the percentage of people sleep-disturbed and highly sleep-disturbed by transportation noise, respectively *(35)*. In the case study included in this chapter, high sleep disturbance is used as the sleep disturbance indicator. Using a lower cut-off value (i.e. sleep-disturbed) would give higher prevalence but would be associated with a lower DW, resulting in either a higher or a lower estimate of the burden caused by sleep disturbance due to noise. An important reason for using high sleep disturbance is that this is closer to the case definition used in studies associating a DW to sleep disturbance based on the comparison to other health states (see below).

Noise exposure

Appropriate exposure indicator

In the position paper on dose–effect relationships for nighttime noise *(36)*, as well as in the EU's Directive 2002/49/EC *(37)*, L_{night} was proposed as the nighttime noise indicator for sleep disturbances (see Chapter 1). L_{night} is defined as the "A-weighted long-term average sound level as defined in ISO 1996-2: 1987", determined over all night periods of a typical year. Noise events in the period between 23:00 and 7:00 contribute to the calculation of L_{night}. In WHO's *Night noise guidelines for Europe (38)*, several $L_{night,outside}$ exposure categories are linked with sufficient scientific evidence to health and sleep disturbance outcomes, and can accordingly be used to assess the degree of sleep disturbance associated with transportation noise (see Table 4.1). Additionally, it is possible to derive exposure–response relationships between L_{night} and instantaneous reactions to noise (such as the number of additionally induced EEG awakenings or behaviourally confirmed awakenings) to assess the expected degree of sleep fragmentation. However, L_{night} is an equivalent continuous sound pressure level summarizing complex time patterns of exposure into a single value. This necessarily leads to information loss: noise scenarios, which differ in number, acoustical properties and placement of noise events, may calculate to the same L_{night} but differ substantially in their effects on sleep. In contrast to daytime traffic, where high traffic densities may lead to more or less constant and continuous noise levels, low traffic densities during the night often go along with intermittent exposure to single noise events. Hence, traffic-noise-induced alterations in sleep structure depend crucially on the number of noise events, the acoustical properties (such as maximum sound pressure levels) of single noise events, the placement of noise events within the night, and noise-free intervals between noise events *(11,19,39)*. Indeed, the *Night noise guidelines for Europe (38)* still support the validity of the recommendation of the WHO *Guidelines for community noise (40)* that, in order to prevent sleep disturbances, one should consider the equivalent sound pressure level and the number and level of sound events. Also, Directive 2002/49/EC *(37)* states that it may be advantageous to use maximum sound pressure level L_{Amax} or sound exposure levels as supplementary noise indicators for night period protection. However, predicting after-effects such as self-reported sleep disturbance or long-term health effects may require information on the long-term average sound level.

Exposure data for estimating the burden of sleep disturbance due to noise

Since road traffic noise accounts for the larger proportion of people exposed in most European countries (based on data from France, the Netherlands, Switzerland and the United Kingdom), road traffic noise exposure data are chosen here to estimate the burden of disease. As an example, exposure data from the Netherlands are used (Table 4.2). The exposure assessment was based on most exposed facade at dwellings, not on individuals. The total population was 15.864 million in the Netherlands in 2000. Assuming that household size does not differ between the noise exposure categories, these data may be extrapolated to the whole population. It should be noted that, because of the method of calculation used (25-metre grid), the higher levels tend to be underestimated.

Table 4.1. Ranges for the relationship between nocturnal noise exposure and health effects in the population

$L_{night,outside}$	Health effects observed in the population
< 30 dB(A)	Although individual sensitivities and circumstances differ, it appears that up to this level no substantial biological effects are observed.
30 – 40 dB(A)	A number of effects are observed to increase: body movements, awakenings, self-reported sleep disturbance and arousals. The intensity of the effect depends on the nature of the source and the number of events. Vulnerable groups (for example, children and chronically ill and elderly people) are more susceptible. However, even in the worst cases, the effects seem modest.
40 – 55 dB(A)	Adverse health effects are observed among the exposed population. Many people have to adapt their lives to cope with the noise at night. Vulnerable groups are more severely affected.
> 55 dB(A)	The situation is considered increasingly dangerous for public health. Adverse health effects occur frequently, and a sizable proportion of the population is highly annoyed and sleep-disturbed. There is evidence that the risk of cardiovascular disease increases.

Source: Night noise guidelines for Europe (38).
Note. The guidelines assume an average attenuation of 21 dB(A) between inside and outside noise levels.

Table 4.2. Percentage of dwellings per environmental noise class in the Netherlands, 2000

L_{night} levels dB(A)- source	<39	40–44	45–49	50–54	>54
Motorways	70.2	16.2	9.1	3.1	1.4
Regional roads	93.8	3.4	1.6	0.8	0.3
City roads	57.9	17.7	15.2	8.0	1.3
All roads	21.9	37.3	25.9	11.9	3.0
Railways	76.6	12.4	6.3	2.7	1.9
Amsterdam Airport	98.1	1.4	0.5	0.0	0.0
All types of traffic	18.6	24.7	31.3	18.6	6.8

Source: Unpublished data from the Netherlands National Institute for Public Health and the Environment (RIVM), method described in Dassen AGM, Jabben J, Janssen PMH. [*Development of the environmental model for population annoyance and risk analysis. Partial validation and risk analysis*.] (abstract in English). Bilthoven, RIVM, 2001 (RIVM report 2001 725401001/2001).

Exposure–response relationship

Exposure–response relationships from experimental and field studies

Experimental and field studies have shown clear exposure–response relationships between single noise events and instantaneous arousals, EEG awakenings, behavioural awakenings or motility *(12,14,19,22,38,42–44)*. Exposure–response relationships between L_{night} or similar integrated measures and instantaneous sleep disturbance are rare *(45,46)*. This may in part be attributed to the fact that L_{night} as a whole-night indicator can only be directly related to whole-night sleep parameters. In principle, exposure–response relationships on the single event level can be used to

predict the expected degree of sleep fragmentation depending on L_{night}, given the fact that the number and loudness of noise events are positively correlated with L_{night}. However, the variance in the number of noise-induced awakenings, and therefore the imprecision of the prediction, increases with increasing L_{night}, as many different exposure patterns can lead to the same L_{night} in the higher exposure categories. Therefore, it may be advantageous for assessing sleep disturbance to gather information on the number of noise events contributing to L_{night} additional to L_{night}.

Although instantaneous effects such as arousals, EEG awakenings, behavioural awakenings and elevated motility all reflect relevant aspects of the complex concept of sleep disturbance, it is not clear how they could be used to assess the burden of disease. Their occurrence is not pathological per se, as these reactions are also a physiological part of sleep in the absence of noise-induced sleep disturbance. They only reach pathological significance once a certain physiological frequency is exceeded, i.e. once sleep fragmentation reaches a relevant degree. However, inter-individual variability in the sensitivity to noise exposure is high, and it is not clear to what extent the exposure–response relationships that were derived from field study subject samples with limited representativeness can be extrapolated to the population. Furthermore, although new research is under way, at the moment relationships are almost exclusively available for aircraft noise, whereas an assessment of the burden of sleep disturbance due to noise requires an assessment of the risk of other main sources as well.

Exposure–response relationships from epidemiological studies

Miedema et al. *(47)* presented synthesis curves for self-reported sleep disturbance from aircraft, road traffic and railway noise. These curves were based on the pooled data from 15 original data sets (more than 12 000 individual observations) obtained from 12 field studies (*a*) where L_{night} was included in the dataset or there was the possibility to calculate/estimate this metric on the basis of information regarding the included sites; and (*b*) where questions regarding waking up or being disturbed by transportation noise during the night were answered. Studies using questions that included disturbance of rest were excluded because resting is different from sleeping and does not necessarily take place during the night only. A more extensive analysis was recently completed *(35)*. It was based partly on the same data but included pooled data from 28 original data sets obtained from 24 field studies (23 000 participants) carried out since 1970. This analysis yielded very similar curves and included 95% confidence intervals that took into account the variation between individuals and studies.. However, no polynomial approximations were published for these curves, and therefore the functions from Miedema et al. *(47)* were used for the present purpose. The percentage of "highly sleep-disturbed" persons (%HSD) as a function of noise exposure indicated by L_{night} was found to be as follows.

Aircraft: $\%\ HSD = 18.147 - 0.956\ (L_{night}) + 0.01482(L_{night})^2$

Road traffic: $\%\ HSD = 20.8 - 1.05\ (L_{night}) + 0.01486(L_{night})^2$

Railways: $\%\ HSD = 11.3 - 0.55\ (L_{night}) + 0.00759\ (L_{night})^2$

The curves are based on data in the L_{night} (outside, maximally exposed facade) range 45–65 dB(A). Low exposure levels (L_{night} < 45 dB(A)) were excluded from the analyses because the assessment of those noise levels was relatively inaccurate and other sources may be more important in situations with these low levels. High exposure levels (L_{night} > 65 dB(A)) were also excluded, because in the areas of very high ex-

posure levels there may also have been self-selection of persons with low sensitivity to noise. Therefore, the extrapolation of the presented functions is expected to give a better indication of sleep disturbance at low and very high levels than using the data at these levels. The polynomial functions are close approximations of the curves in this range and their extrapolations to lower exposure (40–45 dB(A)) and higher exposure (65–70 dB(A)).

Although cumulative effects of simultaneous exposure to noise from different types of traffic should ideally be taken into account, knowledge on the effects of simultaneous exposure to different noise sources is limited *(48)*. A pragmatic way would be to calculate a single L_{night} value for all modes of transportation and base the risk assessment on this combined exposure measure, or preferably to use the methodology established earlier for determining the relationship between exposure to multiple noise sources and annoyance *(49)*.

Disability weight

The WHO DW for primary insomnia is 0.100 and is defined *(50)* as:

> *… difficulty falling asleep, remaining asleep, or receiving restorative sleep for a period [of] no less than one month. This disturbance in sleep must cause significant distress or impairment in social, occupational, or other important functions and does not appear exclusively during the course of another mental or medical disorder or during the use of alcohol, medication, or other substances.*

This definition of primary insomnia excludes the sleep disturbances that appear during the use of "other substances" or outside factors such as light or noise. When sleep is permanently disturbed by environmental factors and becomes a sleep disorder, it is classified in the International Classification of Sleep Disorders *(51)* as "environmental sleep disorder". Environmental sleep disorder (of which noise-induced sleep disturbance is an example) is a sleep disturbance due to a disturbing environmental factor that causes a complaint of either insomnia or daytime fatigue and somnolence *(38)*. While noise-induced sleep disturbance is not to be considered as a case of primary insomnia, the "burden of disease" of primary insomnia and noise-induced environmental sleep disorder may be similar. Van Kempen, cited in Knol & Staatsen *(41)*, reported a mean DW of 0.100 for severe sleep disturbance due to noise, based on a pilot study among 13 medical experts working according to a protocol by Stouthard *(52)*. De Hollander *(58)* expanded the study to 35 environmental physicians, epidemiologists and public health professionals and also found a mean DW of 0.10 (median DW: 0.08; standard deviation: 0.10; range: 0–0.45) using the same protocol. Although an earlier study published by de Hollander et al. *(53)* used a DW of only 0.010 for the same condition, no DW was available at that time so the weight of the least severe category of the first GBD study by Murray et al. *(59)* was used.

Müller-Wenk *(54)* found a mean DW of 0.055 (median DW: 0.04; range: 0.02–0.31) for those highly sleep-disturbed by nighttime road noise, based on a survey of 42 Swiss physicians who were asked to interpolate this type of sleep disturbance into a list of health states with existing DWs. In 2005, Knoblauch &

Müller-Wenk *(55)* interviewed a sample of 14 general practitioners recently admitting patients with obstructive sleep apnoea syndrome (OSAS) to the sleep clinic in St Gallen in Switzerland. They were asked to compare the relative mean severity of the health state of contacted persons with OSAS, with primary insomnia or with sleep disturbance due to increased exposure to road noise in the bedroom. This case definition of sleep disturbance is comparable to that of "highly sleep disturbed" on which the exposure–response relationship was based. Based on their own professional experience, 9 of the 14 respondents considered noise-related sleep disturbance to be less serious on average than primary insomnia, and 11 of the 14 considered noise-related sleep disturbance to be less serious on average than OSAS; the mean judgement of the 14 respondents was that noise-related sleep disturbance has a mean severity of 0.9 times the severity of primary insomnia (range: 0–2.1), which resulted in a DW of 0.09 (CI 0.06–0.12). As in the previous studies, the distribution was rather skewed; the median severity ratio was 0.63, which corresponds to a DW of 0.063.

Following the *Night noise guidelines for Europe (38)*, 0.07 was chosen as the DW of noise-related sleep disturbance in the calculation of DALYs. This value takes into account both the medians and the means of the DW observed in the above studies. Given the rather skewed distributions of the reported DWs, the median of the study with the lowest DW *(54)* was chosen as a low estimate, whereas the highest observed mean value *(41,58)* was chosen as a high estimate, yielding the uncertainty interval (0.04–0.10). The uncertainty in the exposure–response relationship was not factored in for this analysis.

EBD calculations

This section provides methodological guidance to two approaches to calculating the burden of sleep disturbance related to environmental noise. The first method is the exposure-based approach using the exposure–response relationship and exposure data. The second method is the direct estimation of the burden using a population survey.

Exposure-based assessment

The exposure-based approach estimates the prevalence of high sleep disturbance (reporting 72 or higher on a 100-point scale) due to noise by combining the exposure data with the exposure–response relationships for high sleep disturbance. One year of nighttime exposure to road traffic noise is proposed as the duration causing high sleep disturbance, since people with a bedroom exposed to a road with a high level of night traffic are subject to more or less stationary noise levels at night. Therefore, it can be assumed that their sleep disturbance exists all year round.

DALYs for sleep disturbance were calculated using the road traffic noise exposure distribution in L_{night} as assessed in the Netherlands in 2000 (see Table 4.2), the total population of the Netherlands in 2000 (15 864 000), the exposure–response relationships presented above for sleep disturbance due to road traffic noise (using the expected percentage of highly sleep-disturbed people at the midpoint of the category as a function of L_{night} in the range 45–65 dB(A)) and the DWs (see Table 4.3). This calculation suggests that there are about 24 669 DALYs lost in the Netherlands due to road traffic noise-induced sleep disturbance. Taking 0.04 and 0.10 as the extremes of the range for the weights, the credible range for the DALYs is from 14 096

to 35 242. This is a very conservative estimate, derived only for the exposure–response and exposure data for road traffic noise and not including the impacts of aircraft and railway noise. However, although the impact at a given exposure level is expected to be higher for aircraft noise (but slightly lower for railway noise) *(35)*, far fewer people are exposed to aircraft (and railway) noise than to road traffic noise.

Table 4.3. Exposure-based approach to estimating DALYs for highly sleep-disturbed people due to environmental noise, using exposure data from the Netherlands

Exposure category L_{night} (dB(A))	Percentage of population exposed	Percentage of people highly sleep-disturbed	Number of cases in the Netherlands	DALYs		
				DW = 0.04	DW = 0.07	DW = 0.10
45 – 49	25.9	4.3	176 677	7 068	12 367	17 668
50 – 54	11.9	6.4	121 009	4 840	8 471	12 101
>54	3.0	11.5	54 730	2 188	3 831	5 473
Total				14 096	24 669	35 242

Source: Unpublished data from the Netherlands National Institute for Public Health and the Environment (RIVM), method described in Dassen AGM, Jabben J, Janssen PMH. [*Development of the environmental model for population annoyance and risk analysis. Partial validation and risk analysis*.] (abstract in English). Bilthoven, RIVM, 2001 (RIVM report 2001 725401001/2001).

Burden of sleep disturbance from road traffic noise in western Europe

As mentioned in Chapter 2, the Noise Observation and Information Service for Europe (NOISE) provides noise exposure data that can be used for calculating disease burden in western European countries. Following the same method used in Chapter 2, the percentage of people highly sleep-disturbed can be calculating using the mid–level values of the exposure categories in the NOISE dataset. Because the NOISE dataset does not provide data on the categories of < 45 dB(A) and 45–49 dB(A), the percentages for these two categories were calculated conservatively by assuming the same percentages between the two categories of 45–49 dB(A) and 50–54 dB(A). The mid-level value of the category was used in the application of exposure–response functions specific to the noise sources. Because the L_{night} was the annual average of exposure level by definition, the duration of effects was also considered to be one year.

Tables 4.4, 4.5 and 4.6 summarize the distribution of population exposed to road, rail and air traffic noise, respectively, during the night in agglomerations with more than 250 000 inhabitants, and exposure-based DALY calculation using the exposure–response function presented above. Owing to a lack of exposure data covering the rural population, it was not possible to estimate DALYs for the whole EU population including rural areas without extrapolation. Assuming that the observed exposure distributions using the strategic noise maps may apply to approximately 285 million people living in cities or agglomerations with more than 50 000 inhabitants (57% of the total EU population), we can cautiously infer that the DALYs are approximately 903 000 years for urban population in the EU assuming DW = 0.07 (Table 4.7). Taking 0.04 and 0.10 as the extremes of the range for DWs, the credible range for the DALYs is 0.52–1.29 million. It should be noted that the burden in rural areas or small town with less than 50 000 inhabitants is not included here, and that we did not count the burden in the exposure range below 45 dB(A).

Table 4.4. DALYs for highly sleep-disturbed people due to road traffic noise in the EU

Exposure category L_{night} (dB(A))	Percentage of population exposed [a]	Percentage of people highly sleep-disturbed [b]	Number of cases per million [b]	DALYs lost in the urban population [c]		
				DW = 0.04	DW = 0.07	DW = 0.10
< 45	44 [d]	NA	NA	NA	NA	NA
45–49	20 [d]	4.5	8 906	101 526	177 670	253 814
50–54	20	6.6	13 266	151 230	264 652	378 074
55–59	10	9.6	9 556	108 937	190 640	272 342
60–64	5	13.2	6 611	75 365	131 888	188 412
65–69	1	17.6	1 763	20 099	35 174	50 248
Total	100		40 102	457 156	800 023	1 142 890

[a] The source of exposure data is the Noise Observation and Information Service for Europe (NOISE) as of June 2010.
[b] The percentage and number of cases were calculated with the polynomial equation, using the mid-level values of exposure categories.
[c] DALYs were calculated for the 285 million persons living in agglomerations with > 50 000 inhabitants.
[d] Noise maps do not provide data for the categories of < 45 dB(A) and 45–49 dB(A) for L_{night}. Therefore, the percentages of population in these categories were interpolated using a very conservative assumption: the percentage for the 45–49 dB(A) is the same as that for 50–54 dB(A).

Table 4.5. DALYs for highly sleep-disturbed people due to rail traffic noise in the EU

Exposure category L_{night} (dB(A))	Percentage of population exposed [a]	Percentage of people highly sleep-disturbed [b]	Number of cases per million [b]	DALYs lost in the urban population [c]		
				DW = 0.04	DW = 0.07	DW = 0.10
< 45	93 [d]	NA	NA	NA	NA	NA
45–49	3 [d]	2.3	690	7 866	13 765	19 664
50–54	3	3.3	1 003	11 440	20 019	28 599
55–59	1	4.8	477	5 437	9 515	13 593
60–64	0	6.6	0	0	0	0
65–69	0	8.8	0	0	0	0
Total	100		2 170	24 743	43 300	61 857

[a] The source of exposure data is the Noise Observation and Information Service for Europe (NOISE) as of June 2010.
[b] The percentage and number of cases were calculated with the polynomial equation, using the mid-level values of exposure categories.
[c] DALYs were calculated for the 285 million persons living in agglomerations with > 50 000 inhabitants.
[d] Noise maps do not provide data for the categories of < 45 dB(A) and 45–49 dB(A) for L_{night}. Therefore, the percentages of population in these categories were interpolated using a very conservative assumption: the percentage for the 45–49 dB(A) is the same as that for 50–54 dB(A).

Table 4.6. DALYs for highly sleep-disturbed people due to air traffic noise in the EU

Exposure category L_{night} (dB(A))	Percentage of population exposed[a]	Percentage of people highly sleep-disturbed[b]	Number of cases per million[b]	DALYs lost in the urban population[c]		
				DW = 0.04	DW = 0.07	DW = 0.10
<45	96[d]	NA	NA	NA	NA	NA
45–49	2[d]	6.2	1 235	14 078	24 637	35 195
50–54	2	8.8	1 761	20 075	35 130	50 186
55–59	0	12.2	0	0	0	0
60–64	0	16.3	0	0	0	0
65–69	0	21.1	0	0	0	0
Total	100		2 996	34 153	59 767	85 382

[a] The source of exposure data is the Noise Observation and Information Service for Europe (NOISE) as of June 2010.
[b] The percentage and number of cases were calculated with the polynomial equation, using the mid-level values of exposure categories.
[c] DALYs were calculated for the 285 million persons living in agglomerations with > 50 000 inhabitants.
[d] Noise maps do not provide data for the categories of < 45 dB(A) and 45–49 dB(A) for L_{night}. Therefore, the percentages of population in these categories were interpolated using a very conservative assumption: the percentage for the 45–49 dB(A) is the same as that for 50–54 dB(A).

Table 4.7. DALYs for highly sleep-disturbed people due to all traffic noise in the EU

Source of traffic noise	DALYs[a]		
	DW = 0.04	DW = 0.07	DW = 0.10
Road	457 156	800 023	1 142 890
Rail	24 743	43 300	61 857
Air	34 153	59 767	85 382

[a] For the 285 million population living in agglomerations with > 50 000 inhabitants.

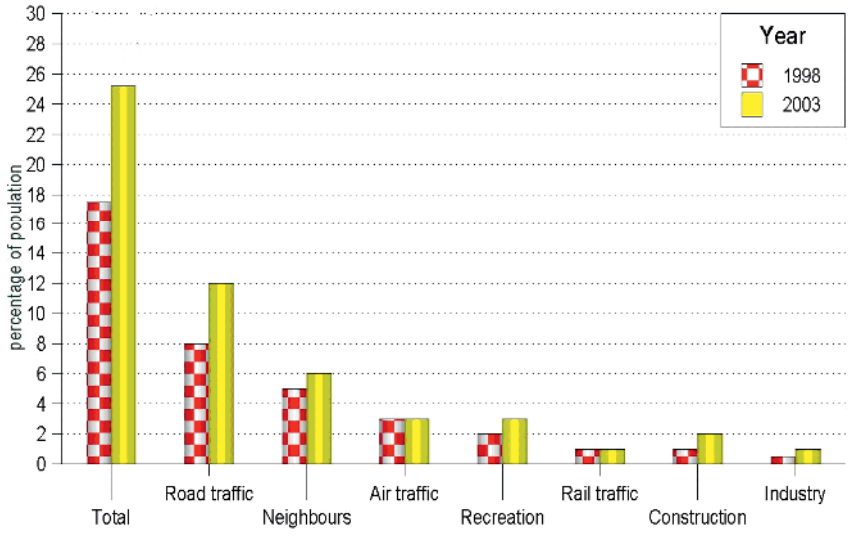
Outcome-based assessment

The burden of highly disturbed sleep due to nighttime noise in terms of DALYs may also be directly estimated on the basis of survey data in the population concerned. Survey data from the Netherlands were used as an example in this section. Fig. 4.1 shows the relative contributions to overall sleep disturbance caused by noise from different sources in the Netherlands. These data were derived from surveys in 1998 and 2003 *(56)* in which 4000 and 2000 people, respectively, all of whom were randomly selected, were asked: "To what extent is your sleep disturbed by noise from (source mentioned) ...?" on a scale from 0 to 10 (pertains to noise perceived in the last 12 months). People recording the three highest points on the scale were considered "highly disturbed" according to an international convention that is close to the case definition used in the pooled analysis to define the exposure–response relationship *(46)*. About 12% of the general population reported being highly disturbed by road traffic noise during sleep in the Netherlands in 2003. The totals are calculated from the number of people reporting serious sleep disturbance from one or more sources. About 25% of the general population reported being highly disturbed by any source of noise during sleep in the previous 12 months. This approach allows

cases from multiple sources to be counted more directly. Since this study is based on a survey conducted in the Netherlands, it is not representative of other Member States in the EU.

Considering that the Netherlands had a population of 16 225 000 in 2003, approximately 1 947 000 and 4 056 250 people were highly disturbed during sleep by road traffic noise and any source of noise, respectively. The corresponding DALYs calculated with a DW of 0.07 are 136 290 years and 283 937 years for road traffic noise and any source of noise, respectively (Table 4.8). The uncertainty in the survey estimates was not factored in for this analysis.

Fig. 4.1. Percentages of the population claiming to be highly disturbed by noise during sleep from two surveys in the Netherlands



Source: van den Berg et al. *(36)*.

Table 4.8. The estimated DALYs lost due to sleep disturbance using prevalence data from the Netherlands

Noise source	Percentage of population highly sleep disturbed	Population of the Netherlands	Number of cases in the Netherlands	DALYs		
				DW = 0.04	DW = 0.07	DW = 0.10
Road traffic	12	16 225 000	1 947 000	77 880	136 290	194 700
One or more sources	25	16 225 000	4 056 250	162 104	283 937	405 625

Uncertainties, limitations and challenges

Comparing two approaches

The DALYs based on the second method are significantly greater than those based on the exposure-based estimates. One of the reasons for the difference may be that the exposure–response relationship is not given for values below 45 dB(A) and above 65 dB(A), where the uncertainties of the relationship are greater. By not counting the people in the exposure range below 45 dB(A), the prevalence of sleep disturbance is underestimated. In addition, the percentage of sleep disturbed above the level of 65 dB(A) may be underestimated, also resulting in an underestimation of the burden of sleep disturbance induced by road traffic noise. This could partly be solved by extrapolating the exposure–response relationship for the range between 40 and 70 dB(A), should exposure data be available in this range.

Uncertainty with respect to the exposure-response relationship

The amount of variance in sleep disturbance scores explained by the exposure–response relationships is intermediate (road traffic, railways) or at the low end within the range of usual values that are considered meaningful (aircraft), so that they are not suited to predicting individual reactions. However, in most cases the uncertainty regarding individual reactions is not what matters for noise policy. Most policy, including policy based on estimates of the burden of disease due to environmental noise, is made with a view to the overall reaction to exposures in a (reference) population. This means that it is not the uncertainty with respect to the prediction of an individual or group reaction that is important, but that regarding the exact relationship between exposure and response in the (reference) population. The accuracy of the estimation of this relationship is described by the confidence intervals around the curve. If properly established, the confidence interval takes into account the variation between individuals as well as the variation between studies *(57)*, which are much smaller than the wide prediction intervals for individuals. The functions can be useful for evaluating the nighttime noise exposure in a particular area by predicting what the response of the reference population would be in that area.

With regard to aircraft noise, it should be noted that the variance in the responses is large compared to the variance found for rail and road traffic, meaning that the uncertainty is higher. One of the reasons for higher uncertainty may be that the time pattern of noise exposures around different airports varies considerably due to specific nighttime regulations. Also, there are indications of a time trend, whereby the most recent studies show the highest self-reported sleep disturbance, leading to a possible underestimation of the response at a given aircraft noise exposure level by the current curve.

Applications and limitations of the exposure-response relationship

According to the EU position paper on dose–effect relationships for nighttime noise *(36)*, the exposure–response relationships above represent the current best estimates of the influences of nocturnal traffic noise exposure (conceptualized as L_{night}) on self-reported sleep disturbance for road traffic and for rail traffic, when no other factors are taken into account. As mentioned above, the uncertainty may be higher with respect to aircraft noise, and such responses should be considered as indicative only.

A limitation of the exposure–response relationship is that it does not take into account other (exposure) variables that determine, in addition to average nighttime noise levels outdoors at the most exposed facade, the exposure level in the bedroom. Most important may be the difference in exposure between the most exposed facade and the bedroom facade, as well as the difference between the outdoor exposure at the bedroom facade and the indoor exposure in the bedroom. Also, adding noise exposure descriptors other than the nighttime average, such as noise in the early or late parts of the night, descriptors of peak levels or number of events may improve the prediction of self-reported sleep disturbance.

Also, it must be stressed again that the sleeper is not aware of himself or his surroundings during most parts of the night, and hence subjective estimates of noise-induced sleep disturbance may differ substantially from objective measures. Indeed, recent laboratory studies indicate that the impact of traffic noise on sleep structure increases in the order air road rail, thus reversing the order observed for self-reported measures such annoyance and sleep disturbance *(19,48)*. Therefore, although the estimated DALYs may correctly reflect the burden of disease in terms of self-reported sleep disturbance, it is questionable whether the estimates correctly reflect aspects that would reflect consequences of chronically fragmented sleep in terms of impairment of daytime performance or long-term health effects that are not obtainable via self-reporting.

Conclusions

Although self-reported sleep disturbance may not reflect the total impact of nighttime noise on sleep, it is the only effect for which exposure–response relationships on the basis of L_{night} are available for the most important noise sources. Furthermore, while it is hard to weigh self-reported sleep disturbance, it may be even harder to assign a DW to physiological changes indicating a certain degree of sleep fragmentation.

An example using data from 2000 on exposure in the Netherlands indicates a conservative estimate of some 25 000 DALYs lost yearly due to sleep disturbance induced by road traffic noise.

With the increasing effort devoted to noise mapping, more and better data on the levels of exposure to nighttime noise will become available in the EU Member States, so that, by combining them with the relationships, the prevalence of self-reported sleep disturbance can be estimated. Our calculation using the noise maps data showed that DALYs assuming DW = 0.07 were 307 959 years for the EU population living in agglomerations with > 250 000 inhabitants. Cautious extrapolation indicated that DALYs assuming DW = 0.07 might be in the range 0.5–1.0 million years for whole EU population.

We adopted conservative assumptions whenever necessary except for extrapolation of exposure data from larger agglomerations to the population of the agglomerations with > 50 000 inhabitants in the EU Member States. Considering that we did not count cases of high sleep disturbance occurring below 45 dB(A) and milder sleep disturbance at all ranges, we are confident that the above DALY estimation is not an overestimation.

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5. ENVIRONMENTAL NOISE AND TINNITUS[7,8]

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Several authors consider tinnitus to be a symptom of the auditory system and not as a disease per se. On the other hand, tinnitus is an entry in the International Classification of Diseases (ICD-9 (388.3) and ICD-10 (H93.1)). Tinnitus is very often found to be present concomitantly with hearing loss. This is also true for noise-induced tinnitus and noise-induced hearing loss (NIHL) *(1,2)*. Nevertheless, tinnitus may be experienced by persons exposed to excessive noise without measurable hearing loss *(3)*. The natural history, the annoyance and disability, the clinical approaches for diagnosis and treatment and the consequences of tinnitus differ significantly from these elements in persons with NIHL. For instance, insomnia reported by tinnitus sufferers is not a consequence of NIHL. Therefore, the authors consider it justified that tinnitus be analysed per se as an independent outcome of environmental noise risk assessment and burden of disease.

Definition of outcome

Tinnitus is the general term for sound perception (for instance, roaring, hissing or ringing) that cannot be attributed to an external sound source. To put it in terms of auditory abilities, tinnitus is the inability to perceive silence *(4)*. Tinnitus defined in such broad terms is rather prevalent. It is widely believed that mild, occasional or acute temporary tinnitus is experienced by nearly everybody in their lifetime at some time or another, the majority resolving spontaneously *(5)*. There is considerable variation in tinnitus expression, its etiology and its effects on patient's lives *(6)*.

Tinnitus may be classified according to its different attributes: duration of a single episode (seconds, minutes; intermittent, continuous), temporal duration (days, months, years) or severity (degree of annoyance, interference with daily living). Dauman & Tyler *(7)* proposed a classification according to five parameters of tinnitus: pathology, severity, duration, site and etiology. Stephens & Hétu *(8)* proposed a clas-

7 This chapter is dedicated to the late Xavier Bonnefoy, who was an essential initiator, leader and motivator during its development. Part of this work was presented at the Internoise2006, 3-6 December 2006, Honolulu, Hawaii, USA.

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sification according to the patient's abilities and quality of life. In fact, there is no unique internationally recognized classification.

Tinnitus can cause in some patients one or several of the following consequences:

- sleep disturbance (difficulty in falling asleep or going back to sleep)
- cognitive effects (difficulty with attention and concentration)
- anxiety
- psychological distress
- depression (case reports of suicide)
- communication and listening problems (hearing problems)
- frustration
- irritability
- tension
- inability to work
- reduced efficiency
- restricted participation in social life.

Tinnitus annoyance and experienced handicap can be measured in clinical or research settings on an individual basis by several valid questionnaires. The severity grading classification (grade I to grade IV) as measured by the Tinnitus Severity Questionnaire developed by Goebel et al. is probably one of the most frequently used tinnitus questionnaires in Germany *(9)*. Other countries use different questionnaires that have good psychometric properties (i.e. good internal consistency and test–retest reliability), such as the Tinnitus Reaction Questionnaire *(10)*, which measures emotional tinnitus-related distress, the Tinnitus Handicap Questionnaire *(11)*, which measures the self-reported severity of tinnitus as a handicap, and the Tinnitus Handicap Inventory *(12)*, which quantifies the impact of tinnitus on everyday life. Psychoacoustical measurements of tinnitus can also be made. Typically, however, these measurements do not predict the psychological distress reported by patients *(13)*.

In population-based survey studies, simple questions about duration and the degree of annoyance caused by tinnitus are usually used, rather than the tools described above to assess the individual status. According to Davis *(6)*, at least two elements should be included into any epidemiological study: tinnitus that lasts for five minutes or more (additionally whether it is present for some or all the time); and an assessment of the impact of tinnitus (for example, severity or annoyance). The general agreement of the authors and contributors to this chapter is to focus, for burden of disease purposes, on the degree of severity of disabling tinnitus rather than on its duration.

The proposed operational case definition of tinnitus is a sound perception (for instance roaring, hissing, ringing, noise in the ears or the like) at the time of the survey or during the past year that cannot be attributed to an external sound source,

and having disabling consequences in terms of constant disturbance of the emotional, cognitive, psychological or physical state of the patient. The term "constant" implies that the person has tinnitus that causes an impact on his or her functional life most of the time in at least one of these spheres.

Summary of evidence linking noise and tinnitus

A very small proportion of tinnitus cases signal the presence of an underlying treatable medical condition, such as a tumour or chronic partial opening of the Eustachian tube, but the majority of cases have no apparent or treatable cause. Tinnitus caused by excessive exposure to noise has long been described *(14–16)*. Fifty to 90% of patients with chronic noise trauma report tinnitus *(17)*.

Between 12% and 50% of persons with noise-induced hearing loss report having tinnitus *(18–21)*. Nevertheless, as stated before, tinnitus may be experienced by persons exposed to excessive noise who do not have measurable hearing loss *(3)*.

There is no single pathophysiological pathway to explain the occurrence of tinnitus. All structures of the auditory system have been suggested as possible sites of generation for tinnitus, from the periphery to the auditory cortex. Many explanatory models have been proposed, based on either anatomical, physiological, clinical or neuropsychological approaches. The underlying mechanisms responsible for transient and chronic tinnitus are most likely also different *(2)*. Despite those limitations in understanding the pathophysiology, however, there is no doubt that acute and chronic noise exposure can cause incapacitating tinnitus *(2,22)*. In noise-induced hearing loss and noise-induced tinnitus, it can be assumed that genesis is based on the same pathophysiological pathway *(23–27)*.

Hearing impairment is not expected to occur at $L_{Aeq,8h}$ levels of 75 dB(A) or below, even for prolonged occupational noise exposure. It is also expected that environmental noise exposure with a $L_{Aeq,24h}$ of 70 dB(A) or below will not cause hearing impairment in the large majority of people, even after a lifetime of exposure *(28)*. Although, to our knowledge, there are no empirical data to propose a no observed adverse effect level (NOAEL) for noise-induced tinnitus, it is reasonable and plausible to use the same protective NOAELs for tinnitus as those for noise-induced hearing loss. Therefore, for this burden of disease calculation, social/leisure noise is the most relevant source of exposure and concern for the EUR-A epidemiological subregion and North American countries, as these sources may typically exceed these thresholds. It is worth noting that traffic noise exceeds 85 dB(A) in some urban settings of developing countries *(29–31)*.

Exposure–response relationship

The exposure of interest in this context is leisure exposure, such as personal music players, gun shooting events, music concerts, sporting events and the use of firecrackers. To develop an exposure–response relationship, it would be necessary to find studies that linked these leisure noise exposures with the relative risk of occurrence of moderate to severe tinnitus. Although there are some studies based on this approach *(32–36)*, few could be identified and these did not cover all exposure settings. It was therefore not possible to develop an exposure–response relationship.

An alternative would be to estimate the relationship between noise and tinnitus derived from the risk curve relating noise exposure to hearing loss. This theoretical approach would be based on the existence of a valid quantitative relationship between noise-induced hearing loss levels and tinnitus risk. Should such a curve exist or be derived from existing data, the ISO 1999:1990 standard could be used to derive the risk of tinnitus per noise exposure level and duration. Although we know that the prevalence of tinnitus increases with the prevalence of noise-induced hearing loss, according to a recent literature review by Tyler *(37)* we are still not aware of any valid quantified relationship per hearing level between tinnitus prevalence and noise-induced hearing loss. Some authors do present data about this relationship, but we are not aware of any valid curves that could be used for burden of disease calculation.

Both these approaches also require population exposure data regarding the prevalence of exposure to leisure noise, which are not readily available at present.

Disability weight

There were no DWs readily available for tinnitus for burden of disease calculations. Three different approaches have been used to estimate DWs.

A first approach was for the authors to propose DWs by analogy with comparable diseases for which WHO already had DWs from the Global Burden of Disease Project. The best comparison proposed by the experts was with chronic pain, as this health problem shares several characteristics with tinnitus, such as: ongoing unwanted internal (centrally located) stimulus; causing or inducing co-morbidity (secondary symptoms) in terms of constant disturbance of the emotional, cognitive, psychological or physical state; not so well-understood pathophysiology; a lack of valid objective clinical findings or confirmatory laboratory tests; and possible response to cognitive therapy. Chronic pelvic pain has a DW of 0.122, whereas low back pain caused by chronic intervertebral disc protrusion has a DW of 0.121 (range 0.103–0.125). Other plausible comparisons are with cases of primary insomnia, which have a DW of 0.100 while a mild depressive episode has a DW of 0.140. As tinnitus may induce in some cases any of these two consequences, an interpolation in those ranges seemed reasonable. Thus, a DW of 0.120 was suggested *(38)*.

As this first approach was not considered to be very robust, a second approach was developed, based on the Canadian Population Health Impact of Disease Project, as an alternative to this first approach *(39)*. The preference scores (conceptually corresponding to one minus DW) were based on rating by health professionals and university experts using the Classification and Measurement System of Functional Health (CLAMES) *(40)* (see Appendix 1). This attempt did not give the expected results owing to unresolved methodological issues, and thus was not pursued.

Finally, an expert panel approach was undertaken. Based on all the available data, former proposals and an expert portrait of functional limitations caused by tinnitus (see Appendix 2), a third approach was proposed by the WHO expert on the Global Burden of Disease Project, Dr Colin D. Mathers, together with the WHO expert responsible for the Environmental Noise Burden of Disease Project, Dr Rokho Kim and the first author. This approach was based on the concept of "affecting ability to lead a normal life" (or affecting quality of life in terms of disabling consequences)

within the definition of disabling tinnitus. Two different DWs for different levels of severity of disabling tinnitus were proposed: 0.01 for mildly (slightly) disabling tinnitus and 0.11 for an aggregate moderate and severely disabling tinnitus. These two severity weights are for limitations in leading a normal life. These provisional proposals, pending a more formal valuation exercise, are based on approximate correspondence to the following conditions in a Dutch DW study that used the same methodology as the Global Burden of Disease Project *(41)*. This study estimated the following DWs for activities of daily living (ADL) limitations in the elderly:

- no to mild ADL limitations in the elderly, 0.01 (range 0.006–0.012)
- moderate to severe ADL limitations in the elderly, 0.11 (range 0.056–0.174).

For comparison, this study gave low back pain an average weight of 0.06, mild to moderate agoraphobia and epilepsy both a weight of 0.11, and mild stable angina (NYHA class 1–2) a weight of 0.08. Some comparable weights used in the GBD 2001 update of the Global Burden of Disease Study include:

- primary insomnia (causing problems with usual activities), 0.10
- dysthymia, 0.14
- moderate iron deficiency (80–109 g/l haemoglobin in women), 0.011.

It is worth mentioning that the DW of 0.11 for moderate to severely disabling tinnitus is very close to the proposed DW of 0.120 that emerged from the first approach. Therefore, DWs of 0.01 for slightly disabling tinnitus and of 0.11 for moderate to severely disabling tinnitus are used for the burden of disease calculations in this chapter.

EBD calculations

Outcome-based approach for leisure-noise-induced tinnitus in the EUR-A epidemiological subregion

The approach chosen for this chapter uses survey-based studies to estimate the prevalence of tinnitus on a population basis. With this approach, it is necessary to estimate the attributable portion of tinnitus caused by environmental noise exposure.

Prevalence of the outcome

A comprehensive review of the literature was made using published documents as identified by PubMed's internet resource through Laval University's Ariane search tool (http://ariane.ulaval.ca/web2/tramp2.exe/log_in?setting_key=french), references cited in selected articles, the authors and contributors of unpublished documents, and experts' opinions. When more than one published article was based on the same study population and design, the later or updated version was used.

The three research strategies retrieved more than 400 studies in English, French, Spanish or German. From that first extraction, 99 were selected as being potentially of interest. A global quality assessment of the studies was done independently by two reviewers, who classified each study as pass or fail based on criteria including external validity, internal validity and data analysis. Disagreements on the inclusion/exclusion of articles were resolved by consensus among the reviewers. Once

studies were selected, a data extraction form was used. This process led to the identification of 23 epidemiological studies of interest that met minimal specified quality criteria and these were presented in a background paper *(38)*.

To select the studies that are to be used for burden of disease calculations, the authors identified those that estimated point prevalence. Also, sampling had to be random and population-based. The authors analysed, when available, the wording of the questions. There is no internationally recognized standard definition of disabling tinnitus. None of the questions used in these studies answered specifically and in a standardized manner all the consequences of chronically disabling tinnitus. The selected studies estimated the prevalence of tinnitus through various concepts such as annoyance, difficulty falling asleep, and tinnitus moderately or very bothersome. Table 5.1 gives a summary of the six selected studies, with specification of the potential disability concept that could be used in each one. All six are cross-sectional descriptive prevalence studies estimating a point or yearly prevalence, based on random samples of the study population.

Table 5.1. Summary of studies selected for burden of disease calculations for tinnitus

Reference (age group in years, country) [sample size]	Question	Selected potential disability concept
Axelsson & Ringdahl *(42)* (20–80, Sweden) [3600]	Do you suffer from tinnitus?	Question 6. Severity of tinnitus (mark the most appropriate alternative) Tinnitus does not bother me particularly Tinnitus bothers me only in quiet surroundings Tinnitus disturbs my sleep […] Tinnitus plagues me all day
Davis *(43)* (17+, England) [48 313]	Nowadays do you get noises in your head or ears?	Tinnitus affecting quality of life
Hannaford et al. *(44)* 2005 (14+, Scotland) [15 788]	(missing exact question) ["Most questions related to current or recent (within the previous twelve months) symptoms … "]	Tinnitus problems "affected their ability to lead a normal life"
Nondahl et al. *(21)* 2002 (48–92, USA) [3737]	In the past year, have you had buzzing, ringing, or noise in your ears?	"Significant tinnitus" if at least moderate tinnitus or tinnitus causing difficulty in falling asleep
Paré & Levasseur *(45)* (15+, Canada) [20 773]	Do you hear ringing, buzzing or whistling noises in your ears or head that last 5 minutes or more at a time?	Do these noises [tinnitus] bother you? (moderately or a lot)
Sindhusake et al. *(18)* (55–99, Australia) [2015]	Have you experienced any prolonged ringing, buzzing or other sounds in your ears or head within the past year, that is, lasting for 5 minutes or longer?	Tinnitus "gets you down"

As the most common complaint from tinnitus sufferers is sleep disturbance, a first proposal by the experts was to use these data for burden of disease purposes. Although this was appealing, these results give only a partial picture of all the possible consequences of tinnitus. Of all the concepts used in the selected studies, those used by Davis *(43)* and by Hannaford *(44)*, as presented in Table 5.1, match more closely the global concept of disabling tinnitus and the similar concepts used for burden of disease calculations for other health problems. Therefore, the results of these two studies were used for burden of disease calculations of tinnitus induced by environmental noise. Despite the fact that the concepts used in these two studies do not correspond exactly to the wording of the operational case definition, the authors consider that these concepts match in an acceptable and reasonable way our definition of disabling tinnitus for calculating DALYs. Studies using similar concepts for disabling tinnitus could eventually be used for burden of disease calculations.

Based on the two selected studies, the authors calculated a weighted prevalence (with weights based on sample size) of tinnitus according to severity level (Table 5.2).

Table 5.2. Weighted population prevalence calculation for disabling tinnitus

Reference	Sample size (age group)	No. of cases of disabling tinnitus		
		Slight	Moderate	Severe
Davis *(43)*	19 023 (17+)	634 (3.3%)	228 (1.2%)	83 (0.4%)
Hannaford et al. *(44)*	15 788 (14+)	564 (3.6%)	189 (1.2%)	59 (0.4%)
Weighted mean prevalence	—	3.4	1.2	0.4

The general trend for the relationship between tinnitus prevalence and age generally shows that tinnitus prevalence increases with age and decreases after 60–70 years of age *(6)*. Hannaford et al. *(44)* do not present the results by age group for disabling tinnitus. Davis *(6)* reports an increasing prevalence with age for disabling tinnitus (see Table 5.3). For burden of disease calculations, the crude prevalence rate was used, as both studies cover almost the same age range (14 years and over or 17 years and over) and were done in two countries that have similar age distributions. For countries with different age distributions than European countries, the prevalence data by age group presented in chapter 9, Tables: section 1 page 901 under "Tinnitus affecting quality of life" of reference 43 can be used.

There are no clinically or statistically significant gender differences for noise-induced tinnitus *(6,38)*. Therefore, the authors suggest not taking gender into account for burden of disease calculations of tinnitus induced by environmental noise.

Prevalent cases in EUR-A countries were calculated based on population data extracted from the European health for all database *(46)* (Table 5.3). There is some evidence that noise-induced tinnitus is present in children *(47)*. To our knowledge, there are no population data on the prevalence of tinnitus in children. As the available prevalence data are based on two population studies of young people aged 14 years and over and 17 years and over, respectively, prevalent cases in EUR-A countries were calculated for age 15 years and over. The year 2001 was used for this example of calculation for comparison with *The world health report 2002 (48)*.

Table 5.3. Population and prevalent cases of disabling tinnitus per severity level for the WHO EUR-A epidemiological subregion, 15 years old and over, 2001

Total population	Population aged 15 years and over	Weighted prevalence per severity level	Prevalent cases of disabling tinnitus by severity level
413 967 744	344 131 386	Slight: 3.4%	11 845 523
		Moderate: 1.2%	4 122 166
		Severe: 0.4%	1 407 670
		Total	17 375 359

Attributable fraction of the outcome

As mentioned above, the prevalence approach involves proposing an attributable fraction of tinnitus specifically caused by environmental noise exposure in order to be able to calculate environmental noise burden of disease. Most studies reviewed, including the two selected ones, report the prevalence of tinnitus in the study population with no direct reference to cause. The few that do address cause do not specifically address environmental noise as a causal factor. There is no particular clinical presentation of tinnitus induced by environmental noise compared to tinnitus from other causes.

For burden of disease purposes, a case of environmental-noise-induced tinnitus is one that corresponds to the exclusive case definition. Cases due to mixed causes such as occupational and environmental noise exposures should be excluded from the attributable fraction. This choice will tend to give a conservative estimate of burden of disease due to tinnitus induced by environmental noise.

Only two data sources were readily available to estimate the population-attributable fraction for environmental noise. One is based on a large study in which 1535 patients attending the Tinnitus Clinic at the Oregon Health & Science University answered a standardized questionnaire. Among the 1406 patients with a valid noise exposure history, 16.2% (228/1406) reported having been exposed to recreational noise without any occupational or military exposures. Of these patients, 199 (14.2%) reported having usually or always at least one of 15 disability items. To the question "Were illness, accident or other special circumstances associated with the onset of your present tinnitus?", 26 (1.8%) reported that the onset of tinnitus was associated with exclusive recreational noise exposure. This last figure should be considered as an absolute minimum for this population, as people often do not relate the onset of their tinnitus with noise exposure unless it began suddenly following a brief, intense exposure (S.E. Griest & W.H. Martin, unpublished data, 2008).

The other available estimation is from Girard & Simard, who produced preliminary results based on a large medical surveillance database of over 88 320 workers' audiometric examinations carried out between 1983 and 1996 (S.A. Girard & M. Simard, unpublished data, 2005). After adjustment for occupational noise exposure level and duration, hearing level and age, the estimated attributable fraction of tinnitus caused exclusively by hobby or leisure noise exposure was 4.6% for this cohort *(38)*.
A third source of information was used. The authors asked 14 audiology experts (clinicians, rehabilitation centre professionals and university professors), one specialized psychologist and two ear, nose and throat medical specialists for their opinion on their estimation of the attributable portion of tinnitus caused exclusively by environmental

noise exposure. The experts first gave an individual estimate of the attributable fraction with figures ranging from 1% to 15%. After discussing this issue during a meeting with a subgroup of the same experts, based on the three available data sources, the consensus was for an estimated attributable fraction of 3% as a conservative but plausible and reasonable figure.

Calculation of DALYs

According to current knowledge and the data presented, the authors consider that there is no premature mortality caused by environmental-noise-induced tinnitus and therefore no YLL. Even though there are some reports of tinnitus sufferers committing suicide *(49)*, these are likely to be already accounted for in calculations of burden of disease attributed to suicide.

Table 5.4 presents the calculations of DALYs for disabling tinnitus, without reference to cause, for the WHO EUR-A epidemiological subregion in 2001.

Table 5.4. DALY calculation for disabling tinnitus per severity level for WHO EUR-A epidemiological subregion, 15 years of age and over, 2001

Severity	Prevalent cases	Disability weight	DALYs
Slight	11 845 523	0.01	118 455
Moderate	4 122 166	0.11	453 438
Severe	1 407 670	0.11	154 844
Total	17 375 359	—	726 737

As a comparison, the burden of non-cause-specific disabling tinnitus in EUR-A countries is higher than that of lower respiratory infections and several other well-recognized health problems (Table 5.5).

Table 5.5. Comparison of burden of disease for disabling tinnitus with some other common health problems, EUR-A epidemiological subregion, 2001

Health problem	DALYs
Unipolar depressive disorders	4 091 000
Hearing loss, adult onset	1 857 000
Diabetes mellitus	1 083 000
Disabling tinnitus	726 000
Lower respiratory infections	614 000
Oral diseases	353 000
Prostate cancer	335 000
Hypertensive heart disease	317 000
HIV/AIDS	208 000
Sexually transmitted diseases, excluding HIV	79 000

Source: World Health Organization *(48)* (except for disabling tinnitus).

DALYs for environmental-noise-induced disabling tinnitus for the WHO EUR-A epidemiological region in 2001 are presented in Table 5.6 by introducing the 3% population-attributable fraction into the calculations.

Table 5.6. Calculation of DALYs for environmental noise induced tinnitus by severity level for the WHO EUR-A epidemiological subregion, 15 years of age and over, 2001

Severity	Prevalent cases	Disability weight	Population-attributable fraction	DALYs
Slight	11 845 523	0.01	0.03	3 554
Moderate	4 122 166	0.11	0.03	13 603
Severe	1 407 670	0.11	0.03	4 645
Total	17 375 359	—	—	21 802

As a comparison, the burden of disease for environmental-noise-induced disabling tinnitus is higher than that for cataracts or hepatitis B in EUR-A countries (Table 5.7).

Table 5.7. Comparisons of burden of disease for environmental-noise-induced disabling tinnitus with some other common health problems, WHO EUR-A epidemiological subregion, 2001

Health problem (from all causes unless mentioned)	DALYs
Mild mental retardation caused by lead[a]	55 000
Hepatitis C[b]	30 000
Upper respiratory infections[b]	26 000
Environmental-noise-induced disabling tinnitus	22 000
Cataracts[b]	19 000
Hepatitis B[b]	18 000
Appendicitis[b]	16 000
Periodontal disease[b]	16 000
Gonorrhoea[b]	15 000

[a] *Source*: Fewtrell L et al. *(50)*.
[b] *Source*: World Health Organization *(48)*.

These calculations are likely to be valid for the WHO EUR-A epidemiological subregion. They are based on valid population prevalence data corresponding reasonably to the case definition and with DWs matching this case definition, using a rather conservative but plausible impact fraction. Although several aspects of the calculation method are based on expert opinion, all the best available data were integrated into a systematic logical reproducible analysis.

Uncertainties, limitations and challenges

Accuracy of estimates of tinnitus prevalence

The approach chosen for this chapter uses survey-based studies to estimate the prevalence of tinnitus on a population basis. Depending on the questions used for each individual survey, the results may represent anything from lifetime to point prevalence of tinnitus, with or without considerations of duration or severity. In a recent review of the literature *(38)*, prevalence of tinnitus varied from 3% to 36%.

Burden of disease calculations being based on an annual occurrence of the event of interest multiplied by duration, the prevalence data used must reflect a yearly prevalence. Therefore, only point prevalence data, or at the most the previous year's data on disabling tinnitus should be considered.

This approach has some limits for calculating global burden of disease: the prevalence of tinnitus may be different from one country to another; and the survey questions vary from one study to another as there is no standardization of questionnaires. Also, cross-sectional studies have some limitations as they cannot assess the evolution of the problem in terms of fluctuations in duration and severity.

Clinical studies reveal that some individual cases of tinnitus do fluctuate over time from more to less disabling and vice versa *(6)*. Nevertheless, it is assumed that, on average, the overall prevalence will remain stable all year round on a population level.

Lack of exposure data

To our knowledge, there are no valid population data available at present on the prevalence of exposure to leisure-time noise sufficient to induce tinnitus.

Calculating burden of disease in countries other than those in Europe

The authors were unable to identify population data on disabling tinnitus outside the Organisation for Economic Co-operation and Development (OECD) countries. As tinnitus is by essence a subjective experience, its natural history may differ in different cultural settings. The authors consider that it may be risky to infer similar prevalences for economically developing countries as those found in the selected studies. For instance, as stated above, traffic noise in some urban settings is above the levels that can produce tinnitus, thus likely adding to the number of noise sources that induce disabling tinnitus and therefore to the attributable fraction of environmental-noise-induced tinnitus. Should national burden of disease calculations for environmental-noise-induced tinnitus be estimated, calculations should adjust for the age distribution of the target population.

Some experts are convinced that the burden of tinnitus is influenced by the cultural situation. For instance, given that moderate tinnitus can impair cognitive functions such as auditory working memory and visual attention span *(51,52)*, the burden may be higher in cultures with frequent highly demanding professional work, where tinnitus may contribute to unacceptable mistakes.

Conclusions

To our knowledge, the global burden of disease for disabling tinnitus or environmental-noise-induced tinnitus has never been estimated before. The epidemiology of functional limitations caused by tinnitus is rather scarce and even more so for environmental-noise-induced tinnitus.

Although the proposed approach is in some aspects based on expert opinion, hopefully it will be useful as a starting place from which to better ascertain the burden of suffering caused by tinnitus. One of the fundamental goals in constructing summary measures of health is to identify the relative magnitude of different health problems, including diseases, injuries and risk factors *(53)*. The estimate of environmental-noise-induced tinnitus presented in this chapter is based on the best available sci-

ence and may err on the conservative side, according to the authors. Therefore, it is our hope that this work will help to better understand and value the importance of diseases such as tinnitus, which are often not very well known or understood outside specific expert circles, and therefore not a very high priority in the political agenda.

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Appendix 1. Classification and Measurement System of Functional Health (CLAMES)

Core attributes

Pain or discomfort	1. Generally free of pain and discomfort 2. Mild pain or discomfort 3. Moderate pain or discomfort 4. Severe pain or discomfort
Physical functioning	1. Generally no limitations in physical functioning 2. Mild limitations in physical functioning 3. Moderate limitations in physical functioning 4. Severe limitations in physical functioning
Emotional state	1. Happy and interested in life 2. Somewhat happy 3. Somewhat unhappy 4. Very unhappy 5. So unhappy that life is not worth while
Fatigue	1. Generally no feelings of tiredness, no lack of energy 2. Sometimes feel tired, and have little energy 3. Most of the time feel tired, and have little energy 4. Always feel tired, and have no energy
Memory and thinking	1. Able to remember most things, think clearly and solve day-to-day problems 2. Able to remember most things but have some difficulty when trying to think and solve day-to-day problems 3. Somewhat forgetful, but able to think clearly and solve day-to-day problems 4. Somewhat forgetful, and have some difficulty when trying to think or solve day-to-day problems 5. Very forgetful, and have great difficulty when trying to think or solve day-to-day problems
Social relationships	1. No limitations in capacity to sustain social relationships 2. Mild limitations in capacity to sustain social relationships 3. Moderate limitations in capacity to sustain social relationships 4. Severe limitations in capacity to sustain social relationships 5. No capacity or unable to relate to other people socially

Supplementary attributes

Anxiety	1. Generally not anxious 2. Mild levels of anxiety experienced occasionally 3. Moderate levels of anxiety experienced regularly 4. Severe levels of anxiety experienced most of the time
Speech	1. Able to be understood completely when speaking with strangers or friends 2. Able to be understood partially when speaking with strangers but able to be understood completely when speaking with people who know you well 3. Able to be understood partially when speaking with strangers and people who know you well 4. Unable to be understood when speaking to other people
Hearing	1. Able to hear what is said in a group conversation, without a hearing aid, with at least three other people 2. Able to hear what is said in a conversation with one other person in a quiet room, with or without a hearing aid, but require a hearing aid to hear what is said in a group conversation with at least three other people 3. Able to hear what is said in a conversation with one other person in a quiet room, with or without a hearing aid, but unable to hear what is said in a group conversation with at least three other people 4. Unable to hear what others say, even with a hearing aid
Vision	1. Able to see well enough, with or without glasses or contact lenses, to read ordinary newsprint and recognize a friend on the other side of the street 2. Unable to see well enough, even with glasses or contact lenses, to recognize a friend on the other side of the street but can see well enough to read ordinary newsprint 3. Unable to see well enough, even with glasses or contact lenses, to read ordinary newsprint but can see well enough to recognize a friend on the other side of the street 4. Unable to see well enough, even with glasses or contact lenses, to read ordinary newsprint or to recognize a friend on the other side of the street
Use of hands and fingers	1. No limitations in the use of hands and fingers 2. Limitations in the use of hands and fingers, but do not require special tools or the help of another person 3. Limitations in the use of hands and fingers, independent with special tools and do not require the help of another person 4. Limitations in the use of hands and fingers, and require the help of another person for some tasks 5. Limitations in the use of hands and fingers, and require the help of another person for most tasks

Source: Public Health Agency of Canada (http://www.phac-aspc.gc.ca/phi-isp/state_preference-eng.php#clames).

Appendix 2. CLAMES description of a typical (median or average) case of disabling tinnitus causing some consequences

CLAMES attribute	Experts' description of consequence of tinnitus	Corresponding CLAMES descriptor*	CLAMES score
Pain or discomfort	Moderate physical discomfort as the person hears the sound in a lot of day-to-day circumstances (discomfort refers to an unpleasant sensation that is not pain, such as nausea or itching)	Moderate pain or discomfort	3
Physical functioning	Generally no limitations in physical functioning	Generally no limitations in physical functioning	1
Emotional state	More unhappy or sad than happy during waking hours (more than 50% of the time unhappy), […]	Somewhat unhappy (you are not completely unhappy, but you are more unhappy than happy)	3
Fatigue	[…] with little energy and feeling tired most of the time	Most of the time feel tired, and have little energy (most of your waking hours are spent feeling tired or fatigued)	3
Memory and thinking	No problems with memory or thinking clearly, but will have some difficulty in solving day-to-day problems (tinnitus influence on cognition, on thinking capacity and on attention)	Able to remember most things but have some difficulty when trying to think and solve day-to-day problems	2
Social relation-ships	Induces mild limitations in the capacity to sustain social relationships (will limit the number of people and of groups of people they relate to)	Mild limitations in the capacity to sustain social relationships (you have an inhibited capacity for social relationships: you do not always have the ability to maintain the full range of usual social relationships)	2
Anxiety	Anxiety is a hallmark of tinnitus causing consequences (sequelae): there is a high level of anxiety experienced most of the time; there is a feeling of loss of control and helplessness	Severe levels of anxiety experienced most of the time (you experience excessive uneasiness, worry or fear most of the time)	4
Speech	No effect on speech	Able to be understood completely when speaking with strangers or friends	1

CLAMES attribute	Experts' description of consequence of tinnitus	Corresponding CLAMES descriptor*	CLAMES score
Hearing	The independent effect of tinnitus on communication is rather difficult to pinpoint, as a majority of tinnitus sufferers do have some hearing impairment (these are two concomitant health problems that may both affect communication capacities); hearing impairment affects particularly communication in a group conversation; Zenner states that the communication problems do not have the same origin for hearing loss and tinnitus; for tinnitus patients with hyperacusis without hearing loss, often hyperacusis is the source of difficulties communicating in groups of 3 or more people; better descriptor for tinnitus is that it causes more of a discomfort or intolerance in situations of group conversations, rather than an impossibility to hear a conversation; nevertheless, the experts consider that, on average, tinnitus does cause some communication problems in groups	Able to hear what is said in a conversation with 1 other person in a quiet room, with or without a hearing aid, but require a hearing aid to hear what is said in a group conversation with at least 3 other people Able to hear what is said in a conversation with 1 other person in a quiet room, with or without a hearing aid, but unable to hear what is said in a group conversation with at least 3 other people	3 (2)
Vision	No effect on vision	Able to see well enough, with or without glasses or contact lenses, to read ordinary newsprint and recognize a friend on the other side of the street	1
Use of hands and fingers		No limitations in the use of hands and fingers	1

6. ENVIRONMENTAL NOISE AND ANNOYANCE

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Noise annoyance is widely accepted as an end-point of environmental noise that can be taken as a basis for evaluating the impact of noise on the exposed population. As a consequence, EU Directive 2002/49/EC *(1)* recommends evaluating environmental noise exposures on the basis of estimated noise annoyance.

As discussed in Chapter 1, WHO defines health as "a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity" *(2)*. This implies that noise-induced annoyance may be considered an adverse effect on health. People annoyed by noise may experience a variety of negative responses, such as anger, disappointment, dissatisfaction, withdrawal, helplessness, depression, anxiety, distraction, agitation or exhaustion *(3–5)*. Furthermore, stress-related psychosocial symptoms such as tiredness, stomach discomfort and stress have been found to be associated with noise exposure as well as noise annoyance *(6,7)*. Some public health experts feel that severe forms of noise-related annoyance should be considered a legitimate environmental issue affecting the well-being and quality of life of the population exposed to environmental noise. The most important issue in the present context is to what extent health (according to the broad definition given above) is reduced by noise and whether a DW that expresses this reduction, when combined with the prevalence of annoyance, leads to a significant burden of "disease". The other possibility would be that noise annoyance does not significantly contribute to disability and, hence, should not be taken into account when considering the noise-induced burden of disease.

In this chapter, a method for estimating the burden of annoyance due to noise is proposed and illustrated, and related issues are discussed. The method was developed by the Netherlands National Institute for Public Health (RIVM) *(8)* and initially applied to the Netherlands. First, a closer look is taken at noise annoyance in the context of burden of disease calculations.

Definition of outcome

Noise annoyance is assessed at the level of populations by means of a questionnaire. Efforts have been made by the International Commission on Biological Effects of Noise and the International Organization for Standardization *(9)* towards the use of standardized questions asking for the degree of annoyance, and introducing an 11-point numerical scale and a 5-point semantic scale. Recoding scales into a 0–100 annoyance response scale, cut-off values of 50 and 72 have been used to determine the percentage of people annoyed and highly annoyed, respectively. For the 5-point scale, however, cut-off values of 40 and 60 are also in use, matching the three highest categories for annoyance and the two highest categories for high annoyance. The percentage highly annoyed, i.e. the percentage of persons with a response exceeding 72, is the most widely used indicator of the prevalence of annoyance in a population, although percentages using other cut-offs or the mean annoyance may also be used *(10)*. In the case study included in this chapter, high annoyance is used as the annoyance indicator. Using a lower cut-off value would give higher prevalence but

would be associated with a lower DW, resulting in either a higher or a lower estimate of the burden caused by noise annoyance. An important reason for using highly annoyed as the cut-off is the expectancy that only for rather severe annoyance may it be possible to gain consensus on a DW that can be meaningfully distinguished from zero.

Provided it contributes significantly, annoyance due to environmental noise can be included in estimates of the burden related to environmental noise when (*a*) the noise exposure of the population is known, (*b*) exposure–response relationships are available for estimating the annoyance on the basis of the exposures, and (*c*) a DW is attached to noise annoyance. In principle, it is also possible to replace steps (*a*) and (*b*) by direct estimates of annoyance prevalence through an annoyance survey in the population concerned (outcome-based approach).

Traffic noise exposure

Within the framework of Directive 2002/49/EC *(1)*, exposure data have been provided by agglomerations with more than 250 000 inhabitants, as reported by the Noise Observation and Information Service for Europe (NOISE) of the European Environment Agency (EEA) *(11)*. While not all Member States have reported yet, and some differences between Member States may be attributed to methodological differences rather than differences in exposure, these data provide an indication of the exposure distribution within large urban areas in the EU. The distribution of exposure to road traffic noise in Member States was used based on 110 million people, the total number of inhabitants in the agglomerations for which a report had been provided up to June 2010 *(11)*. It is assumed here that the observed exposure distribution may apply to the total urban population within the EU living in cities or agglomerations with more than 50 000 inhabitants, which is estimated to be around 285 million people (57% of the total EU population).

Exposure–response relationship

The EU Position Paper on dose–response relationships between transportation noise and annoyance *(12)* presented synthesis curves for noise annoyance from aircraft, road traffic and railway noise, with their 95% confidence intervals taking into account the variation between individuals and studies. These curves were based on all studies examined by Schultz *(13)* and Fidell et al. *(14)* for which L_{den} (and L_{dn}), and the percentage of "highly annoyed" persons (%HA) meeting certain minimal requirements could be derived, augmented by a number of additional studies *(10)*. The raw data from a total of 54 studies from Europe, North America and Australia investigating noise annoyance from road traffic, aircraft and railways were analysed. The percentage of "highly annoyed" persons (%HA) as a function of noise exposure indicated by L_{den} was found to be the following.

Aircraft:
$\%HA = -9.199 \cdot 10^{-5} (L_{den} - 42)^3 + 3.932 \cdot 10^{-2} (L_{den} - 42)^2 + 0.2939 (L_{den} - 42)$

Road traffic:
$\%HA = 9.868 \cdot 10^{-4} (L_{den} - 42)^3 - 1.436 \cdot 10^{-2} (L_{den} - 42)^2 + 0.5118 (L_{den} - 42)$

Railways:
$\%HA = 7.239 \cdot 10^{-4} (L_{den} - 42)^3 - 7.851 \cdot 10^{-3} (L_{den} - 42)^2 + 0.1695 (L_{den} - 42)$

Data below 45dB and above 75dB (L_{den}) were excluded because the risk of unreliable noise data is high at very low levels, whereas the risk of selection of "survivors" is high at very high levels. The confidence intervals found were narrow, indicating that, even though there is considerable variation between individuals and between studies, the uncertainty regarding the relationships between noise exposure and annoyance is rather limited.

In the same way, and based on the same data, Miedema & Oudshoorn *(10)* established the following relationships for L_{dn}.

Aircraft:
$\%HA = -1.395 \cdot 10^{-4} (L_{dn} - 42)^3 + 4.081 \cdot 10^{-2} (L_{dn} - 42)^2 + 0.342 (L_{dn} - 42)$

Road traffic:
$\%HA = 9.994 \cdot 10^{-4} (L_{dn} - 42)^3 - 1.523 \cdot 10^{-2} (L_{dn} - 42)^2 + 0.538 (L_{dn} - 42)$

Railways:
$\%HA = 7.158 \cdot 10^{-4} (L_{dn} - 42)^3 - 7.774 \cdot 10^{-3} (L_{dn} - 42)^2 + 0.163 (L_{dn} - 42)$

Disability weight

Given the limited number of studies on a DW for annoyance, and the sensitivity of the environmental burden attributed to noise annoyance for small changes in DW, a tentative DW of 0.02 is proposed with a relatively large uncertainty interval (0.01–0.12). The minimum value (0.01) is based on the value used by de Hollander et al. *(15)* and by Stassen et al. *(16)* in environmental burden of disease calculations. The maximum value (0.12) is based on the mean DW found for severe annoyance by Van Kempen (cited in Knol & Staatsen) *(17)*, who did a pilot study among 13 medical experts, working according to a protocol by Stouthard et al. *(18)*. De Hollander *(19)* expanded this study to 35 environmental physicians, epidemiologists and public health professionals and also assessed a mean DW of 0.12 (median: 0.07; standard deviation: 0.16; range 0–0.35) using the same protocol. The relatively high DW for annoyance in these studies may be explained by the presentation of the definition of annoyance with the description that annoyance could lead to various symptoms such as being not (95%) or mildly (5%) anxious or depressed, and having no (95%) to some (5%) cognitive impairment. In addition, Müller-Wenk *(20)* found a mean DW of 0.033 (median: 0.03; range: 0.01–0.12) for communication disturbance based on a survey of 42 Swiss physicians, which may apply to annoyance related to daytime noise exposure. Based on these data and taking a "conservative approach", here only severe cases of annoyance (highly annoyed) are given DW 0.02 for estimation of burden in terms of DALYs.

EBD calculations

Here we provide a method for estimating the environmental burden of disease for noise, estimating the prevalence of noise annoyance by combining exposure data with the exposure–response relationships for noise annoyance. One year is proposed as the duration for exposure causing severe annoyance, as annoyance is an effect that disappears when the noise stops. Age was not considered, assuming that children are annoyed in the same way as adults. While this assumption seems justified, since children showed similar patterns of annoyance to those of their parents *(21)*, it may lead to a slight overestimation since annoyance does not appear to be a relevant concept for infants.

We calculated the DALYs for noise annoyance using the exposure distribution in L_{den} presented by EEA *(11)* for large agglomerations (> 250 000 inhabitants), the exposure–response relationships for annoyance (with expected percentage of highly annoyed people at the midpoint of the category, as a function of L_{den} in the range 42–80 dB(A)) and a range of DWs. This calculation suggests that there are about 654 000 DALYs lost due to noise-induced annoyance within the EU population living in urban areas. Taking 0.01 and 0.12 as the extremes of the range for DWs, the credible range for the DALYs is 0.32–3.92 million (Tables 6.1–6.4). It should be noted that the burden in rural areas or small town with less than 50 000 inhabitants is not included here, and that we took a very conservative assumption about the exposure distribution below 50 dB(A).

Table 6.1. DALYs for highly annoyed people due to road traffic noise in the EU

Exposure category L_{den} (dB(A))	Percentage of population exposed [a]	Percentage of people highly annoyed [b]	Number of cases per million [b]	DALYs lost in the urban population [c]		
				DW = 0.01	DW = 0.02	DW = 0.12
< 55	50	2.77	13 835	39 430	78 859	473 155
55–59	17	8.16	13 868	39 524	79 047	474 285
60–64	19	12.96	24 621	70 170	140 341	842 044
65–69	9	20.08	18 068	51 494	102 989	617 933
70–74	4	30.25	12 100	34 485	68 969	413 815
> 75	1	30.25 [d]	3 025	8 621	17 242	103 454
Total	100		85 517	243 724	487 448	2 924 686

[a] The source of exposure data is the Noise Observation and Information Service for Europe (NOISE) as of June 2010.

[b] The percentage and number of cases were calculated using the mid-level value of each exposure category. For the category of < 55 dB(A), the mid-level value was conservatively set to 48 dB(A).

[c] DALYs were calculated for the 285 million persons living in agglomerations with > 50 000 inhabitants.

[d] As the exposure–response function does not apply to the range over 75 dB(A), the percentage of people highly annoyed in this exposure category was assumed to be the same as in the 70–74 dB(A) category.

Table 6.2. DALYs for highly annoyed people due to rail traffic noise in the EU

Exposure category L_{den} (dB(A))	Percentage of population exposed [a]	Percentage of people highly annoyed [b]	Number of cases per million [b]	DALYs lost in the urban population [c]		
				DW = 0.01	DW = 0.02	DW = 0.12
< 55	95	0.89	8 462	24 116	48 233	289 397
55–59	3	3.44	1 031	2 938	5 877	35 261
60–64	1	6.41	641	1 827	3 655	21 929
65–69	1	11.22	1 122	3 198	6 396	38 374
70–74	0	18.41	0	0	0	0
> 75	0	18.41 [d]	0	0	0	0
Total	100		11 256	32 080	64 160	384 960

[a] The source of exposure data is the Noise Observation and Information Service for Europe (NOISE) as of June 2010.

[b] The percentage and number of cases were calculated using the mid-level value of each exposure category. For the category of < 55 dB(A), the mid-level value was conservatively set to 48 dB(A).

[c] DALYs were calculated for the 285 million persons living in agglomerations with > 50 000 inhabitants.

[d] As the exposure–response function does not apply to the range over 75 dB(A), the percentage of people highly annoyed in this exposure category was assumed to be the same as in the 70–74 dB(A) category.

Table 6.3. DALYs for highly annoyed people due to air traffic noise in the EU

Exposure category L_{den} (dB(A))	Percentage of population exposed [a]	Percentage of people highly annoyed [b]	Number of cases per million [b]	DALYs lost in the urban population [c]		
				DW = 0.01	DW = 0.02	DW = 0.12
< 55	96	3.16	30 327	33 360	66 719	400 315
55–59	3	13.66	4 098	11 679	23 358	140 147
60–64	1	21.76	2 176	6 201	12 401	74 408
65–69	0	31.54	0	0	0	0
70–74	0	42.93	0	0	0	0
> 75	0	42.93 [d]	0	0	0	0
Total	100		36 601	51 239	102 478	614 869

[a] The source of exposure data is the Noise Observation and Information Service for Europe (NOISE) as of June 2010.

[b] The percentage and number of cases were calculated using the mid-level value of each exposure category. For the category of < 55 dB(A), the mid-level value was conservatively set to 48 dB(A).

[c] DALYs were calculated for the 285 million persons living in agglomerations with > 50 000 inhabitants.

[d] As the exposure–response function does not apply to the range over 75 dB(A), the percentage of people highly annoyed in this exposure category was assumed to be the same as in the 70–74 dB(A) category.

Table 6.4. DALYs for highly annoyed people due to all traffic noise in the EU[a]

Source of traffic noise	DALYs		
	DW = 0.01	DW = 0.02	DW = 0.12
Road	243 724	487 448	2 924 686
Rail	32 080	64 160	384 960
Air	51 239	102 478	614 869
Total	327 043	654 086	3 924 515

[a] For the 285 million population living in agglomerations with > 50 000 inhabitants.

Uncertainties, limitations and challenges

Alternative approaches

The burden in terms of DALYs may also be directly estimated on the basis of noise annoyance survey data in the population concerned, if available. However, we expect that the approach starting with the noise exposure levels will be most feasible in the future with the increase of the noise exposure mapping effort. Moreover, it is less sensitive to the idiosyncrasies of the different surveys conducted in different populations and the differences in the processing of the data obtained with the surveys, and it is less sensitive to temporary factors affecting the response of a population surveyed. Therefore, provided that the noise exposure assessment is sufficiently harmonized, the approach that estimates the prevalence of noise annoyance by combining exposure data with the exposure–response relationships for noise annoyance appears to be most promising.

Choice of the exposure–response relationship for annoyance

Various authors have synthesized existing data from community annoyance surveys to develop an exposure–response relationship for use in environmental impact analyses and related community planning efforts, such as Schultz *(13)*, Fidell et al. *(14)* and Miedema & Oudshoorn *(10)*. Schultz recognized the preliminary nature of his original synthesis curve, and did not expect it to remain the final word for long *(19)*. The most comprehensive of these meta-analyses is clearly that published in 2001 by Miedema & Oudshoorn *(10)*. There are, however, two types of qualification that have to be made, which are not elaborated on here:

- the relationships can be refined by taking into account non-acoustical factors and, probably more relevant, acoustical factors that can be affected by policy other than the exposure at the most exposed side, such as sound insulation of the dwelling or the presence or absence of a quiet side *(7)*; and

- there are strong indications that the exposure–response relationships for aircraft noise have changed, so that the curves presented here probably underestimate the annoyance at a given aircraft noise exposure level *(20)*.

Uncertainty with respect to the exposure-response relationship

One cause of doubt regarding the predictability of noise annoyance is that the studies show a large variation in individual annoyance reactions to the same noise exposure level. The other cause of doubt is that attempts to integrate the results from different studies show that there is a large variation in the relationships found in different studies. The large individual variation and the large study variation suggest that it is difficult to predict annoyance with sufficient accuracy. Indeed, the annoyance response of a particular individual or group of individuals can be predicted on the basis of the exposure only with a large amount of uncertainty. This uncertainty can be described by the prediction interval for individuals or groups around the exposure–response curves.

Nevertheless, in most cases, the uncertainty regarding individual or group reactions is not what matters for noise policy. Most policy, including that based on estimates of the burden of disease due to environmental noise, is made with a view to the overall reaction to exposures in a (reference) population. This means that it is not the uncertainty with respect to the prediction of an individual or group reaction that is important, but the uncertainty regarding the exact relationship between exposure and response in the (reference) population. The accuracy of the estimation of this relationship is described by the confidence interval around the curve. If properly established, the confidence interval takes into account the variation between individuals as well as the variation between studies. As found by Miedema & Oudshoorn *(10)*, this results in relatively narrow confidence intervals (as opposed to the wide prediction intervals for individuals or groups).

Applications and limitations of the exposure-response relationship

According to the EU Position Paper, which also recommends the exposure–response relationships presented here, they are only to be used for aircraft, road traffic and railway noise and for assessing long-term, stable situations *(12)*. They can be utilized for strategic assessments, in order to estimate the effects of noise on populations in terms of annoyance. They are not applicable to local, complaint-type situations or to the assessment of the short-term effects of a change of noise climate. The curves have been derived for *adults*. The curves are not recommended for specific sources such as helicopters, low-flying military aircraft, train shunting, shipping, or aircraft on the ground (taxiing) *(12)*.

Conclusions

Compared to other effects of environmental noise and also compared to effects of environmental factors in general, there are relatively many data directly obtained from exposed humans in the field from which exposure–response relationships for noise annoyance could be derived. It appears that, with the increasing effort on noise mapping, more and better noise exposure data will become available so that, by combining them with the relationships, the prevalence of annoyance can be estimated. The third ingredient for estimating the burden due to environmental noise appears the most difficult. It is hard to weigh "annoyance" and it is difficult to relate it to existing weighted outcomes. We used the limited data on the weights available, giving the indication that about 0.62 million DALYs are lost yearly among the urban population in EU countries owing to the occurrence of noise annoyance.

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7. CONCLUSIONS

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Environmental noise: a public health problem

Environmental noise, also known as noise pollution, is among the most frequent sources of complaint regarding environmental issues in Europe, especially in densely populated urban areas and residential areas near highways, railways and airports. In comparison to other pollutants, the control of environmental noise has been hampered by insufficient knowledge of its effects on humans and of exposure–response relationships, as well as a lack of defined criteria. In 1999, WHO published its *Guidelines for community noise (1).*

The European Parliament and Council adopted Directive 2002/49/EC of 25 June 2002 *(2)* with the main aim of providing a common basis for tackling noise problems across the EU. This Directive defines environmental noise as unwanted or harmful outdoor sound created by human activities, including noise from road traffic, railway traffic airports and industrial sites, and focuses on three action areas: the determination of exposure to environmental noise through noise mapping, based on common assessment methods; the adoption of action plans by the Member States based on noise-mapping results; and public access to information on environmental noise and its effects.

Among the various effects of environmental noise, health effects are a growing concern of both the general public and policy-makers in the Member Status in Europe. Most of the assessments performed so far to evaluate the impact of environmental noise have been based on the annoyance it causes. Its consideration as a public health problem with measurable health outcomes has been limited *(3).*

In 2009, WHO published the *Night noise guidelines for Europe (4).* This publication presented new evidence of the health damage of nighttime noise exposure and recommend threshold values that, if breached at night, would threaten health. An annual average night exposure not exceeding 40 dB outdoors is recommended in the guidelines.

Considering the scientific evidence on the threshold of night noise exposure indicated by L_{night} as defined in Directive 2002/49/EC, a L_{night} value of 40 dB should be the target of the night noise guidelines to protect the public, including the most vulnerable groups such as children, the chronically ill and the elderly. A L_{night} value of 55 dB is recommended as an interim target for countries that cannot follow night noise guidelines in the short term for various reasons and where policy-makers choose to adopt a stepwise approach. These guidelines can be considered an extension to the previous WHO *Guidelines for community noise (1)*.

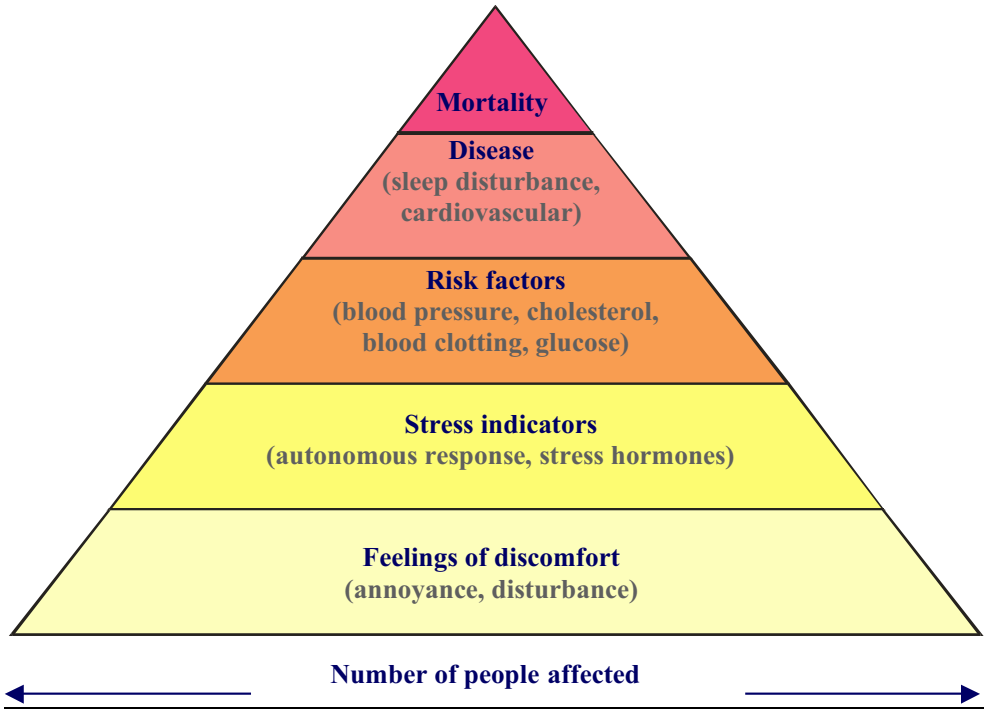
Over the past few years, the working group of experts convened by the European Centre for Environment and Health, Bonn Office and supported by the Joint Research Centre of the European Commission, has collaborated to estimate the burden

of disease from environmental noise, using available evidence and data to inform policy-makers and the public about the health impacts of noise exposure in Europe. The chapters in this publication contain the summary of synthesized reviews of evidence on the relationship between environmental noise and specific health effects. Following the EBD methodology of WHO, the health impacts of environmental noise were estimated using exposure–response relationships, exposure distribution, background prevalence of disease and DWs. For each chapter on specific health outcome, a case study is provided. Policy-makers and their advisers can use these chapters as good practice guidance for the process of quantifying specific health risks of environmental noise.

Effects of environmental noise on selected health outcomes

The severity of health effects due to noise versus the number of people affected is schematically presented by Fig. 7.1. Annoyance, sleep disturbance, cardiovascular disease, cognitive impairment, hearing impairment and tinnitus were initially selected by the working group as health outcomes related to environmental noise.

Fig. 7.1. Severity of health effects of noise and number of people affected



Source: Babisch *(3)*.

Sufficient evidence was available to perform calculations of burdens of such outcomes as annoyance, sleep disturbance and cardiovascular disease. The epidemiological evidence was not as sufficient but was still enough for assuming the relationship of environmental noise to cognitive impairment and tinnitus. The epidemiological studies linking hearing impairment to environmental noise exposure are so sparse that any generalization can be considered exploratory and speculative. Therefore, following the recommendations of the peer-reviewers, the chapter on hearing impairment was not included in this publication.

Cardiovascular disorders

The noise indicators used for noise mapping in the EU can – in principle – be used for a quantitative risk assessment regarding cardiovascular risk if exposure–response relationships are known. Only two end-points – hypertension and ischaemic heart disease – should be considered at this stage. If necessary, different exposure–response curves could be used for different exposures. The noise indicator L_{den} may be useful for assessing and predicting annoyance in the population. However, non-weighted day and night noise indicators may be more appropriate for health-effect-related research and risk quantification.

Cognitive impairment

Scientific evidence indicates the adverse effects of chronic noise exposure on children's cognition. There is no generally accepted criterion for quantification of the degree of cognitive impairment into a DW. However, it is possible to make a conservative estimate of loss in DALYs using the methods presented in this chapter. It is important to consider the assumptions, uncertainties and limitations of the methods when interpreting the estimated values of EBD.

Sleep disturbance

Although self-reported sleep disturbance may not reflect the total impact of nighttime noise on sleep, it is the effect for which exposure–response relationships on the basis of L_{night} are available for the most important noise sources. Furthermore, while it is hard to weigh self-reported sleep disturbance, it may be even harder to assign a DW to physiological changes indicating a certain degree of sleep fragmentation. Now that exposure data from noise mapping will become available as well as the exposure–response relationships, the prevalence of self-reported sleep disturbance can be estimated.

Tinnitus

There is a method to estimate burden of tinnitus from environmental noise based on expert opinion, which will be useful as a starting point using conservative assumptions and approaches.

Annoyance

There are relatively many data directly obtained from exposed humans in the field from which exposure–response relationships for noise annoyance could be derived. It is hard to weigh "annoyance" and it is difficult to relate it to existing DW values. However, if the national and local authorities are willing to take into account the most common complaints of environmental noise, they could assign an acceptable DW value to annoyance, and estimate EBD accordingly.

Estimated DALYs for western European countries

It is estimated that DALYs lost from environmental noise in the EU countries are 60 000 years for ischaemic heart disease, 45 000 years for cognitive impairment of children, 903 000 years for sleep disturbance, 21 000 years for tinnitus and 654 000 years for annoyance. Sleep disturbance and annoyance mostly related to road traffic noise comprise the main burdens of environmental noise in western Europe. If all

of these impacts are considered together, the interval estimate would be 1.0–1.6 million DALYs.[9] The total burden of health effects from environmental noise would be greater than one million years in western Europe, even with the most conservative assumptions that avoid any possible duplication.

Uncertainties, limitations and challenges

The process of risk assessment involves the gathering, synthesizing and interpretation of available evidence. The EBD process, as applied by WHO, is one way of synthesizing this evidence in a standardized manner. EBD methods depend on the availability of data, information, and specific assumptions. To obtain valid and reliable estimates of EBD, good data are needed on the distribution of exposure, on outcomes and on the exposure–response relationship. In the European region, more and better data are available on the distribution of environmental noise, and it is expected that the process of ongoing implementation of EU Directive 2002/49/EC will provide higher quality data in standardized formats comparable between the countries. Regarding outcomes, high-quality data are available for some (e.g. cardiovascular disease) but not for others (e.g. tinnitus). Established exposure–response relationships exist for annoyance, sleep disturbance (subjective), cognitive impairment (children) and cardiovascular disease.

Selection of health effects

Unfortunately, the quality and the quantity of the evidence and data are not the same across the different health outcomes. Other than for cardiovascular disease, obtaining prevalence estimations for the conditions discussed in this publication posed some difficulties. Most of the subclinical conditions are not recorded in routine mortality and morbidity statistics. For tinnitus, the proportion caused by leisure noise rather than occupational noise was difficult to estimate. And conditions such as cognitive impairment in children, sleep disturbance and annoyance are difficult to characterize, let alone estimate the proportion caused by environmental noise. Nevertheless, this publication brings together the best literature and available data and provides transparent justifications of the estimates using conservative assumptions.

Some other outcomes have been suggested as being associated with environmental noise, including hearing impairment, psychiatric conditions such as depression and anxiety, next-day effects of sleep disturbance such as motor accidents. As more evidence accumulates on whether these conditions are indeed associated with environmental noise, further refinements of the estimates in this volume can be made.

Noise exposure indicators

The EU adopted harmonized noise metrics across its Member States: L_{den} to assess annoyance and L_{night} to assess sleep disturbance *(1).* These metrics are used for strategic mapping of exposure in the EU Member States and are common across all transport sources and other sources of environmental noise. The quality of the exposure data produced through the first round of strategic noise maps in EU may not be optimal in terms of validity and reliability. This will have an unavoidable impact

9 The extent to which years lost from different effects are additive across different outcomes is unclear. The different health outcomes might have synergistic rather than antagonistic when the combined effects occur in a person. Therefore, it would be a conservative approach to add the DALYs of different outcomes not considering synergistic effects.

on the accuracy and precision of any risk assessment using these exposure data. With the full implementation of Directive 2002/49/EC, L_{den} and L_{night} are widely accepted as standard indicators of noise exposure in Europe *(6)*. Many previous studies used other metrics that can be converted to L_{den} and L_{night} with some assumptions. However, this conversion from old to new indicators will contribute to the uncertainties of the estimate.

Exposure–response relationships

Although the exposure–response relationships presented in this publication are based on the available evidence at the time of the working group meetings, there are uncertainties especially when they are derived from limited numbers of studies. It should be noted that the exposure–response relationships will need to be updated using the results of future studies.

Confounding factors and effect modifiers

Most epidemiological studies are prone to bias if confounding factors are not properly controlled by design or statistical methods. Confounding factors include age, gender, smoking, obesity, alcohol use, socioeconomic status, occupation, education, family status, military service, hereditary disease, medication, medical status, race and ethnicity, physical activity, noisy leisure activities, stress-reducing activities, diet and nutrition, housing conditions (crowding) and residential status. Future epidemiological research will have to consider effect modifiers (vulnerable groups, sensitive hours of the day, coping mechanisms, different noise sources, etc.) as well as potential confounding factors.

Combined exposure to noise, air pollution and chemicals

The health impacts of the combined exposure to noise, air pollutants and chemicals are rarely considered in epidemiological studies. Combined exposures occur, for example, when people are exposed to road traffic where noise and air pollution co-exist. The stressors that might be considered in the context of combined exposure with noise include: indoor air pollutants (environmental tobacco smoke, volatile organic compounds), outdoor air pollutants (particulate matter, carbon monoxide, sulphur dioxide, nitrogen dioxide), asphyxiants (carbon monoxide, hydrogen cyanide), solvents (xylene, styrene, toluene, benzene, etc.), heavy metals (lead, mercury), pesticides (organophosphates), variables related to housing (biological agents), and vibration.

An international workshop organized by the Joint Research Centre of the European Commission in cooperation with EEA and WHO in 2007 *(7)* concluded that the best knowledge on the health effects due to combined exposure to noise and solvents or heavy metals exists in occupational environments. However, there are few studies showing combined effects of noise and air pollutants in urban environments. Some data exist only on respiratory disorders caused by combined effects of noise and outdoor air pollutants, balance disorders caused by occupational exposure to noise and solvents, and effects on human growth caused by combined effects of noise and heavy metals. The workshop concluded that a substantial amount of research is needed to determine the health effects of combined exposure to environmental noise and other environmental pollutants.

Total burden from environmental noise

In general, care should be taken to avoid "double counting" when DALYs from different outcomes are totalled to estimate an overall burden of disease from an environmental risk factor. In the case of environmental noise, this should not be a big problem. For example, the burdens of annoyance during the daytime and sleep disturbances at night can be safely added up. Nevertheless, because of the different qualities of the evidence underlying the different EBD calculations, special care should be taken when making direct comparisons between DALYs for different outcomes.

If DALYs caused by environmental noise are compared with those from other pollutants, it is important to take into account the approximations and assumptions made in the calculation process. More information on these issues has been summarized in documents on the methodology of EBD *(8)*.

Health inequality and vulnerable groups

Some noise exposures may be worse for some subgroups than for others. Issues such as the lower housing prices near noisy roads mean that the effect of noise is not uniformly distributed throughout the population. Except for a chapter on cognitive impairment in children, this publication did not explore the additional burdens in potentially vulnerable subgroups such as older people and lower socioeconomic groups.

Uses of this publication

The evidence and methods for quantifying the health impacts of environmental noise presented and illustrated in this volume can be used by policy-makers, planners and engineers to measure the magnitude of health problems related to noise pollution in society today. Because many European countries have already produced strategic noise maps and action plans on noise control according to Directive 2002/49/EC *(2)*, the good practices of risk assessment presented in this volume can be readily applied to the national and local situations in many countries. In countries where all the required data for a complete calculation of burden of disease may not be available, this publication demonstrates a range of options that can be used to make estimations according to which components of the risk assessment are accessible.

Although this publication has been prepared with a European focus in terms of policy, available data and legislation, the processes of risk assessment illustrated here can also be used outside Europe as long as the assumptions, limitations and uncertainties described in the various chapters are carefully taken into account.

The effects of neighbourhood noise were not addressed in this publication as they need to be better characterized and measured in future studies. In addition, the effects of leisure noise were not considered because there is very little information available on the prevalence of voluntary exposure to leisure noise through amplified music at concerts and other public events and through personal music players.

Noise and the Parma Declaration on Environment and Health

There is overwhelming evidence that exposure to environmental noise has adverse effects on the health of the population. Recognizing the special need to protect children from the harmful effects of noise, the Parma Declaration adopted at the Fifth Ministerial Conference on Environment and Health *(9)* called on all stakeholders to work together to reduce the exposure of children to noise, including that from personal electronic devices, from recreation and traffic (especially in residential areas), at child care centres, kindergartens and schools and in public recreational settings. This publication provides an evidence base for the future development of suitable guidelines on noise by WHO, as was urged by the Member States in the Parma Declaration. The evidence on burden of disease presented here will inform the new European health policy, Health 2020, which will be presented for endorsement at the WHO Regional Committee for Europe in 2012.

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Burden of Disease from Environmental Noise

The health impacts of environmental noise are a growing concern among both the general public and policy-makers in Europe. This publication provides technical support to policy-makers and their advisers in the quantitative risk assessment of environmental noise, using evidence and data available in Europe. It contains the summary of synthesized reviews of evidence on the relationship between environmental noise and specific health effects, including cardiovascular disease, cognitive impairment, sleep disturbance, tinnitus, and annoyance. For each outcome, the environmental burden of disease methodology, based on exposure–response relationship, exposure distribution, background prevalence of disease and disability weights of the outcome, is applied to calculate the burden of disease in terms of disability-adjusted life-years. The results indicate that at least one million healthy life years are lost every year from traffic-related noise in the western part of Europe. Owing to a lack of exposure data in south-east Europe and the newly independent states, it was not possible to estimate the disease burden in the whole of the WHO European Region. The procedure of estimating burdens presented in this publication can be used by international, national and local authorities in prioritizing and planning environmental and public health policies.

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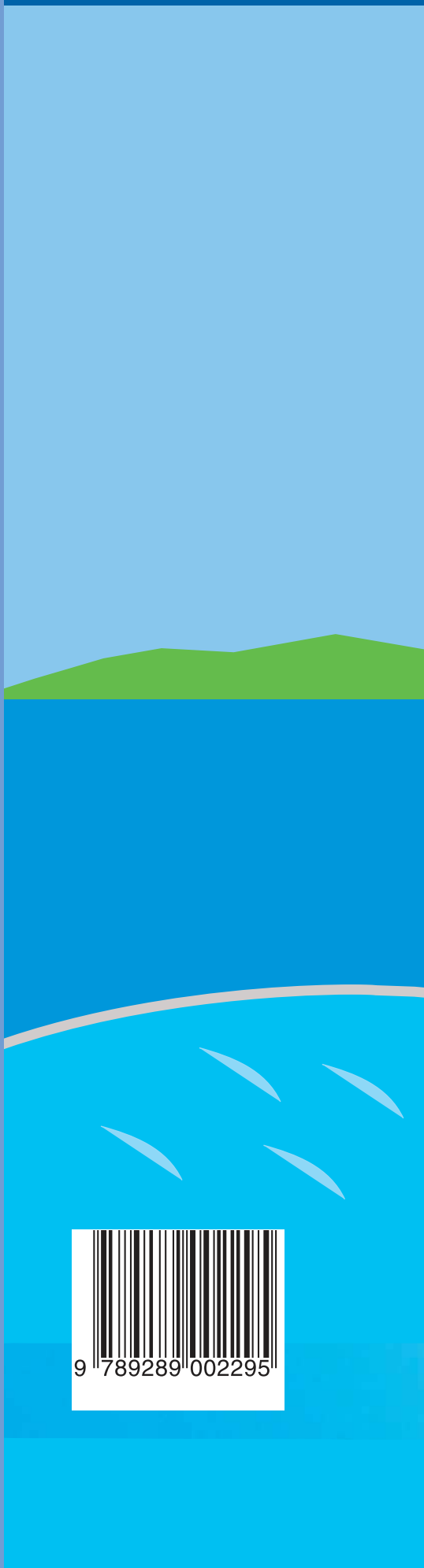

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