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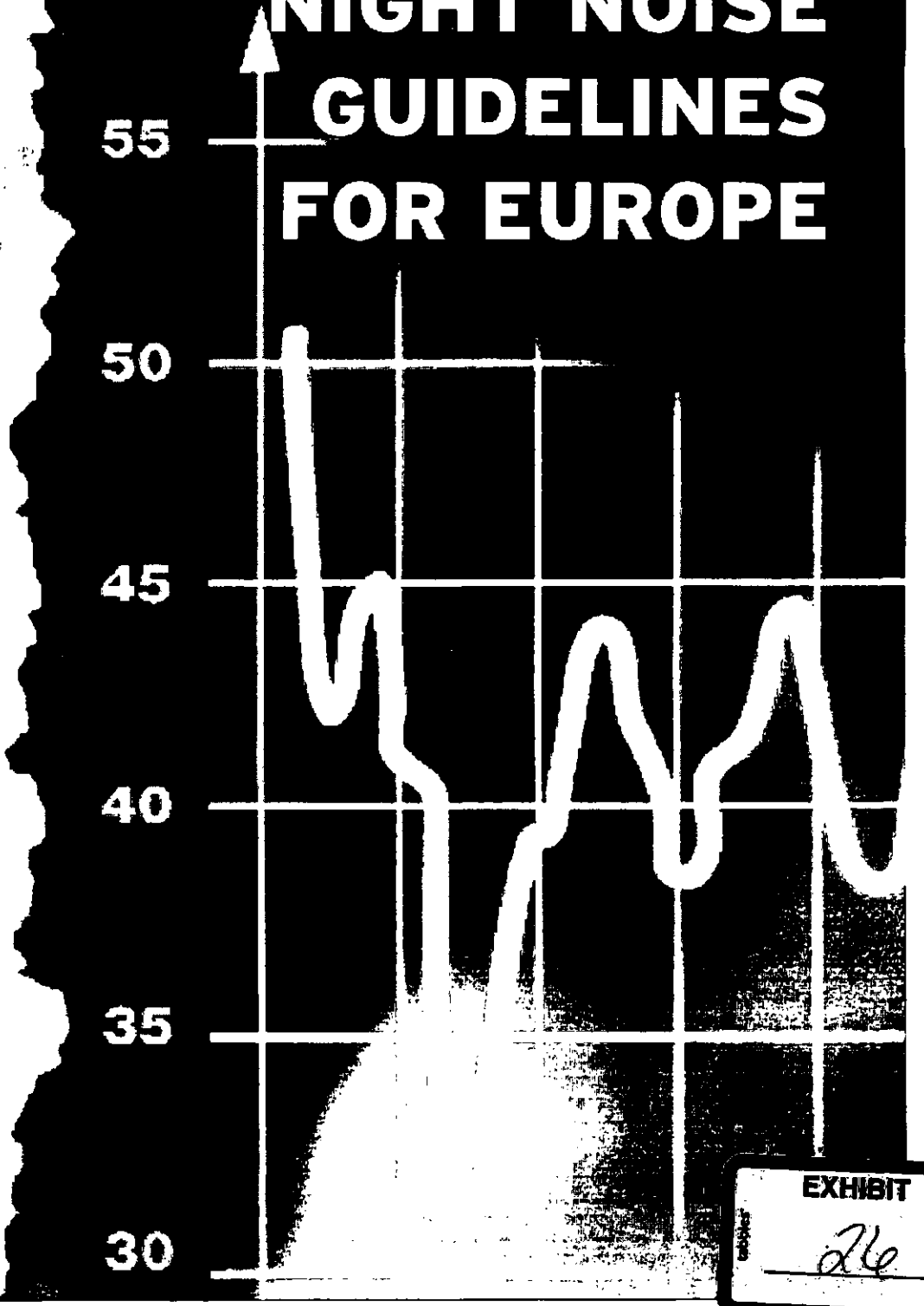
WORLD HEALTH ORGANIZATION

EUROPE

NIGHT NOISE GUIDELINES FOR EUROPE



EXHIBIT

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EUROPE

NIGHT NOISE GUIDELINES FOR EUROPE

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GUIDELINES

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ABSTRACT

The WHO Regional Office for Europe set up a working group of experts to provide scientific advice to the Member States for the development of future legislation and policy action in the area of assessment and control of night noise exposure. The working group reviewed available scientific evidence on the health effects of night noise, and derived health-based guideline values. In December 2006, the working group and stakeholders from industry, government and nongovernmental organizations reviewed and reached general agreement on the guideline values and key texts for the final document of the *Night noise guidelines for Europe*.

Considering the scientific evidence on the thresholds of night noise exposure indicated by $L_{night,outside}$ as defined in the Environmental Noise Directive (2002/49/EC), an $L_{night,outside}$ of 40 dB should be the target of the night noise guideline (NNG) to protect the public, including the most vulnerable groups such as children, the chronically ill and the elderly. $L_{night,outside}$ value of 55 dB is recommended as an interim target for the countries where the NNG cannot be achieved in the short term for various reasons, and where policy-makers choose to adopt a stepwise approach. These guidelines are applicable to the Member States of the European Region, and may be considered as an extension to, as well as an update of, the previous WHO *Guidelines for community noise* (1999).

FOREWORD

WHO defines health as a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity, and recognizes the enjoyment of the highest attainable standard of health as one of the fundamental rights of every human being. Environmental noise is a threat to public health, having negative impacts on human health and well-being. In order to support the efforts of the Member States in protecting the population's health from the harmful levels of noise, WHO issued *Guidelines for community noise* in 1999, which includes guideline values for community noise in various settings based on the scientific evidence available. The evidence on health impacts of night noise has been accumulated since then.

In the WHO European Region, environmental noise emerged as the leading environmental nuisance triggering one of the most common public complaints in many Member States. The European Union tackled the problem of environmental noise with an international law on the assessment and management of environmental noise. The WHO Regional Office for Europe developed the *Night noise guidelines for Europe* to provide expertise and scientific advice to the Member States in developing future legislations in the area of night noise exposure control and surveillance, with the support of the European Commission. This guidelines document reviews the health effects of night time noise exposure, examines exposure-effects relations, and presents guideline values of night noise exposure to prevent harmful effects of night noise in Europe. Although these guidelines are neither standards nor legally binding criteria, they are designed to offer guidance in reducing the health impacts of night noise based on expert evaluation of scientific evidence in Europe.

The review of scientific evidence and the derivation of guideline values were conducted by outstanding scientists. The contents of the document were peer reviewed and discussed for a consensus among the experts and the stakeholders from industry, government and nongovernmental organizations. We at WHO are thankful for those who contributed to the development and presentation of this guidelines and believe that this work will contribute to improving the health of the people in the Region.

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INTRODUCTION

The aim of this document is to present the conclusions of the WHO working group responsible for preparing guidelines for exposure to noise during sleep. This document can be seen as an extension of the WHO *Guidelines for community noise* (1999). The need for "health-based" guidelines originated in part from the European Union Directive 2002/49/EC relating to the assessment and management of environmental noise (commonly known as the Environmental Noise Directive and abbreviated as END) which compels European Union Member States to produce noise maps and data about night exposure from mid-2007. The work was made possible by a grant from the European Commission and contributions from the Swiss and German governments.

Although a number of countries do have legislation directed at controlling night noise exposure, there is little information on actual exposure and its subsequent effects on the population. Estimates made in some countries of the number of people highly disturbed by noise during sleep (see Fig. 1 for the Netherlands as an example) indicate that a substantial part of the population could be exposed to levels that might risk their health and well-being.

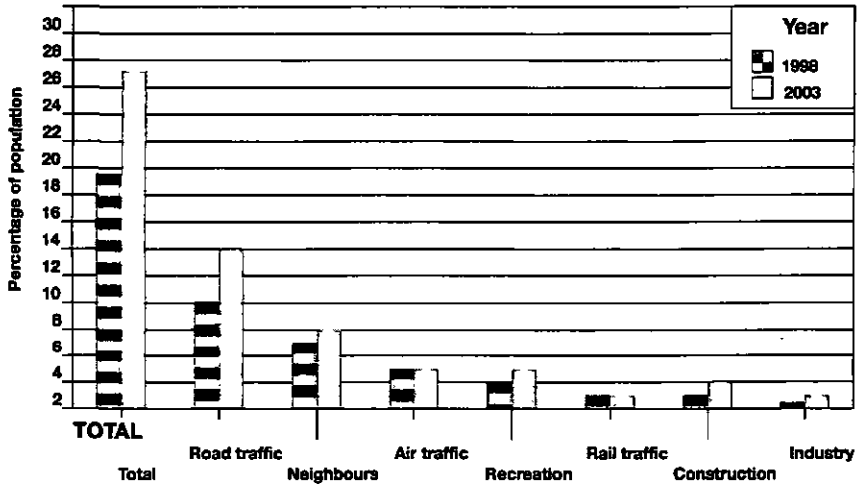


Fig. 1. Percentage of population highly disturbed by noise during sleep in the Netherlands: survey results for 1998 and 2003

As direct evidence concerning the effects of night noise on health is rarely available, these guidelines also use indirect evidence: the effects of noise on sleep and the relations between sleep and health. The advantage of this approach is that a lot of medical evidence is available on the relation between sleep and health, and detailed information also exists on sleep disturbance by noise.

PROCESS OF DEVELOPING GUIDELINES

In 2003, the WHO Regional Office for Europe set up a working group of experts to provide scientific advice to the European Commission and to its Member States for the development of future legislation and policy action in the area of control and surveillance of night noise exposure. The review of available scientific evidence on the health effects of night noise was carried out by an interdisciplinary team who set out to derive health-based guideline values. The contributions from the experts were reviewed by the team and integrated into draft reports following discussion at four technical meetings of the working group. In 2006, all the draft reports were compiled into a draft document on guidelines for exposure to noise at night, which was reviewed and commented on by a number of stakeholders and experts.

At the final conference in Bonn, Germany, on 14 December 2006, representatives from the working group and stakeholders from industry, government and non-governmental organizations reviewed the contents of the draft document chapter by chapter, discussed several fundamental issues and reached general agreement on the guideline values and related texts to be presented as conclusions of the final WHO *Night noise guidelines for Europe.*

NOISE INDICATORS

From the scientific point of view the best criterion for choosing a noise indicator is its ability to predict an effect. Therefore, for different health end points, different indicators could be chosen. Long-term effects such as cardiovascular disorders are more correlated with indicators summarizing the acoustic situation over a long time period, such as yearly average of night noise level outside at the facade ($L_{night,outside}$)[1], while instantaneous effects such as sleep disturbance are better with the maximum level per event (L_{Amax}), such as passage of a lorry, aeroplane or train.

From a practical point of view, indicators should be easy to explain to the public so that they can be understood intuitively. Indicators should be consistent with existing practices in the legislation to enable quick and easy application and enforcement. $L_{night,outside}$, adopted by the END, is an indicator of choice for both scientific and practical use. Among currently used indicators for regulatory purposes, L_{Aeq} (A-weighted equivalent sound pressure level) and L_{Amax} are useful to predict short-term or instantaneous health effects.

SLEEP TIME

Time use studies, such as that undertaken by the Centre for Time Use Research, 2006 (www.timeuse.org/access/), show that the average time adult people are in bed is around 7.5 hours, so the real average sleeping time is somewhat shorter. Due to personal factors like age and genetic make-up there is considerable variation in sleeping time and in beginning and end times. For these reasons, a fixed interval of 8 hours is a minimal choice for night protection.

Though results vary from one country to another, data show (see Fig. 2 as an example) that an 8-hour interval protects around 50% of the population and that it would take a period of 10 hours to protect 80%. On Sundays, sleeping time is consistently 1 hour longer, probably due to people recovering from sleep debt incurred during the week. It should also be borne in mind that (young) children have longer sleeping times.

[1] L_{night} is defined in the END as the outside level. In order to avoid any doubt, the suffix "outside" is added in this document.

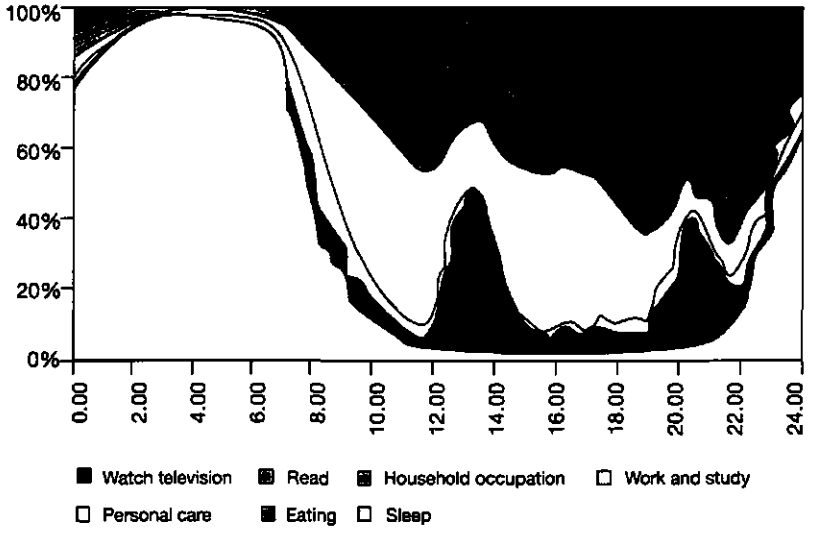


Fig. 2 Percentage of time that the Portuguese population spend asleep or in different activities

Source: http://www.ine.pt/prodserv/destaque/arquivo.asp, based on a study by the Instituto Nacional de Estatistica Portugal, 1999.

NOISE, SLEEP AND HEALTH

There is plenty of evidence that sleep is a biological necessity, and disturbed sleep is associated with a number of health problems. Studies of sleep disturbance in children and in shift workers clearly show the adverse effects.

Noise disturbs sleep by a number of direct and indirect pathways. Even at very low levels physiological reactions (increase in heart rate, body movements and arousals) can be reliably measured. Also, it was shown that awakening reactions are relatively rare, occurring at a much higher level than the physiological reactions.

DEFINITION OF "SUFFICIENT" AND "LIMITED" EVIDENCE

Sufficient evidence: A causal relation has been established between exposure to night noise and a health effect. In studies where coincidence, bias and distortion could reasonably be excluded, the relation could be observed. The biological plausibility of the noise leading to the health effect is also well established.

Limited evidence: A relation between the noise and the health effect has not been observed directly, but there is available evidence of good quality supporting the causal association. Indirect evidence is often abundant, linking noise exposure to an intermediate effect of physiological changes which lead to the adverse health effects.

The working group agreed that there is sufficient evidence that night noise is related to self-reported sleep disturbance, use of pharmaceuticals, self-reported health problems and insomnia-like symptoms. These effects can lead to a considerable burden of disease in the population. For other effects (hypertension, myocardial infarctions, depression and others), limited evidence was found: although the studies were few or not conclusive, a biologically plausible pathway could be constructed from the evidence.

An example of a health effect with limited evidence is myocardial infarction. Although evidence for increased risk of myocardial infarction related to L_{day} is sufficient according to an updated meta-analysis, the evidence in relation to $L_{night,outside}$ was considered limited. This is because $L_{night,outside}$ is a relatively new exposure indicator, and few field studies have focused on night noise when considering cardiovascular outcomes. Nevertheless, there is evidence from animal and human studies supporting a hypothesis that night noise exposure might be more strongly associated with cardiovascular effects than daytime exposure, highlighting the need for future epidemiological studies on this topic.

The review of available evidence leads to the following conclusions.

- Sleep is a biological necessity and disturbed sleep is associated with a number of adverse impacts on health.
- There is sufficient evidence for biological effects of noise during sleep: increase in heart rate, arousals, sleep stage changes and awakening.
- There is sufficient evidence that night noise exposure causes self-reported sleep disturbance, increase in medicine use, increase in body movements and (environmental) insomnia.
- While noise-induced sleep disturbance is viewed as a health problem in itself (environmental insomnia), it also leads to further consequences for health and well-being.
- There is limited evidence that disturbed sleep causes fatigue, accidents and reduced performance.
- There is limited evidence that noise at night causes hormone level changes and clinical conditions such as cardiovascular illness, depression and other mental illness. It should be stressed that a plausible biological model is available with sufficient evidence for the elements of the causal chain.

VULNERABLE GROUPS

Children have a higher awakening threshold than adults and therefore are often seen to be less sensitive to night noise. For other effects, however, children seem to be equally or more reactive than adults. As children also spend more time in bed they are exposed more to night noise levels. For these reasons children are considered a risk group.

Since with age the sleep structure becomes more fragmented, elderly people are more vulnerable to disturbance. This also happens in pregnant women and people with ill health, so they too are a group at risk.

Finally, shift workers are at risk because their sleep structure is under stress due to the adaptations of their circadian rhythm.

THRESHOLDS FOR OBSERVED EFFECTS

The no observed adverse effect level (NOAEL) is a concept from toxicology, and is defined as the greatest concentration which causes no detectable adverse alteration of morphology, functional capacity, growth, development or lifespan of the target organism. For the topic of night noise (where the adversity of effects is not always clear) this concept is less useful. Instead, the observed effect thresholds are provided: the level above which an effect starts to occur or shows itself to be dependent on the exposure level. It can also be a serious pathological effect, such as myocardial infarctions, or a changed physiological effect, such as increased body movement.

Threshold levels of noise exposure are important milestones in the process of evaluating the health consequences of environmental exposure. The threshold levels also delimit the study area, which may lead to a better insight into overall consequences. In Tables 1 and 2, all effects are summarized for which *sufficient and limited evidence* exists. For these effects, the threshold levels are usually well known, and for some the dose-effect relations over a range of exposures could also be established.

Effect		Indicator	Threshold, dB
Biological effects	Change in cardiovascular activity	*	*
	EEG awakening	$L_{Amax,inside}$	35
	Motility, onset of motility	$L_{Amax,inside}$	32
	Changes in duration of various stages of sleep, in sleep structure and fragmentation of sleep	$L_{Amax,inside}$	35
	Waking up in the night and/or too early in the morning	$L_{Amax,inside}$	42
Sleep quality	Prolongation of the sleep inception period, difficulty getting to sleep	*	*
	Sleep fragmentation, reduced sleeping time	*	*
	Increased average motility when sleeping	$L_{night,outside}$	42
Well-being	Self-reported sleep disturbance	$L_{night,outside}$	42
	Use of somnifacient drugs and sedatives	$L_{night,outside}$	40
Medical conditions	Environmental insomnia**	$L_{night,outside}$	42

Table 1 Summary of effects and threshold levels for effects where *sufficient* evidence is available

* *Although the effect has been shown to occur or a plausible biological pathway could be constructed, indicators or threshold levels could not be determined.*

***Note that "environmental insomnia" is the result of diagnosis by a medical professional whilst "self-reported sleep disturbance" is essentially the same, but reported in the context of a social survey. Number of questions and exact wording may differ.*

Table 2 Summary of effects and threshold levels for effects where *limited* evidence is available**

Effect		Indicator	Estimated threshold, dB
Biological effects	Changes in (stress) hormone levels	*	*
Well-being	Drowsiness/tiredness during the day and evening	*	*
	Increased daytime irritability	*	*
	Impaired social contacts	*	*
Medical conditions	Complaints	$L_{night,outside}$	35
	Impaired cognitive performance	*	*
	Insomnia	*	*
	Hypertension	$L_{night,outside}$	50
	Obesity	*	*
	Depression (in women)	*	*
	Myocardial infarction	$L_{night,outside}$	50
	Reduction in life expectancy (premature mortality)	*	*
	Psychic disorders	$L_{night,outside}$	60
	(Occupational) accidents	*	*

* *Although the effect has been shown to occur or a plausible biological pathway could be constructed, indicators or threshold levels could not be determined.*

** *Note that as the evidence for the effects in this table is limited, the threshold levels also have a limited weight. In general they are based on expert judgement of the evidence.*

RELATIONS WITH $L_{NIGHT,OUTSIDE}$

Over the next few years, the END will require that night 'noise' exposures are reported in $L_{night,outside}$. It is, therefore, interesting to look into the relation between $L_{night,outside}$ and adverse health effects. The relation between the effects and $L_{night,outside}$ is, however, not straightforward. Short-term effects are mainly related to maximum levels per event inside the bedroom: $L_{Amax,inside}$. In order to express the (expected) effects in relation to the single European Union indicator, some calculation needs to be done. The calculation for the total number of effects from reaction data on events (arousals, body movements and awakenings) needs a number of assumptions. The first that needs to be made is independence: although there is evidence that the order of events of different loudness strongly influences the reactions, the calculation is nearly impossible to carry out if this is taken into consideration. Secondly, the reactions per event are known in relation to levels at the ear of the sleeper, so an assumption for an average insulation value must be made. In the report a value of 21 dB has been selected. This value is, however, subject to national and cultural differences. One thing that stands out is the desire of a large part of the population to sleep with windows (slightly) open. The relatively low value of 21 dB takes this into account already. If noise levels increase, people do indeed close their windows, but obviously reluctantly, as complaints about bad air then increase and sleep disturbance remains high. This was already pointed out in the WHO *Guidelines for community noise* (1999).

From source to source the number of separate events varies considerably. Road traffic noise is characterized by relatively low levels per event and high numbers, while air and rail traffic are characterized by high levels per event and low numbers. For two typical situations estimates have been made and presented in graphical form. The first is an average urban road (600 motor vehicles per night, which corresponds roughly to a 24-hour use of 8000 motor vehicles, or 3 million per year, the lower boundary the END sets) and the second case is for an average situation of air traffic exposure (8 flights per night, nearly 3000 per year).

Fig. 3 shows how effects increase with an increase of $L_{night,outside}$ values for the typical road traffic situation (urban road). A large number of events lead to high levels of awakening once the threshold of $L_{Amax,inside}$ is exceeded. To illustrate this in practical terms: values over 60 dB $L_{night,outside}$ occur at less then 5 metres from the centre of the road.

In Fig. 4 the same graph is presented for the typical airport situation. Due to a lower number of events there are fewer awakenings than in the road traffic case (Fig. 3), but the same or more health effects. In these examples the worst case figures can be factors higher: the maximum number of awakenings for an $L_{night,outside}$ of 60–65 dB is around 300 per year.

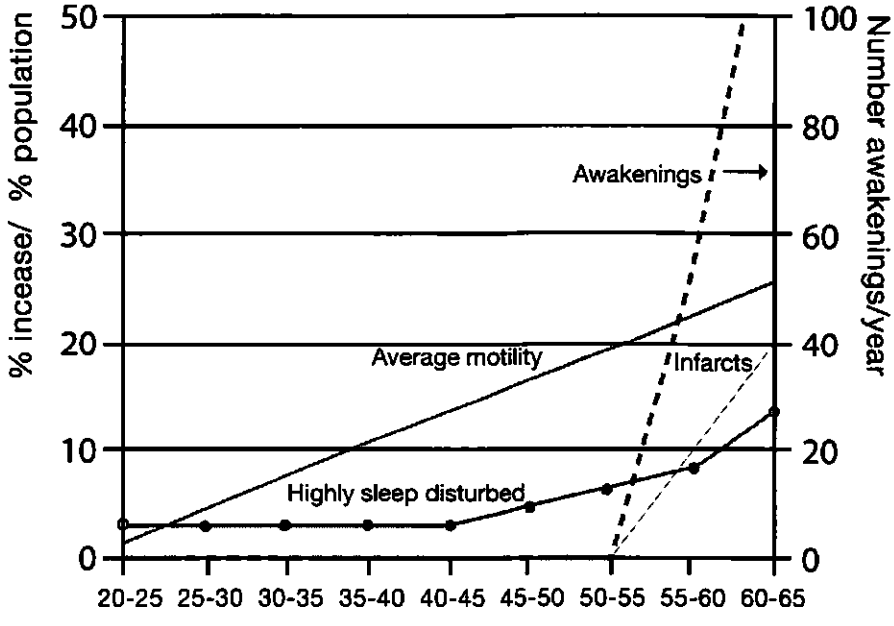


Fig. 3 Effects of road traffic noise at night*

**Average motility and infarcts are expressed in percent increase (compared to baseline number); the number of highly sleep disturbed people is expressed as a percent of the population; awakenings are expressed in number of additional awakenings per year.*

A recent study suggests that high background levels of noise (from motorways) with a low number of separate events can cause high levels of average motility.

Therefore, by using the $L_{night,outside}$ as a single indicator, a relation between effects and indicator can be established. For some effects, however, the relation can be

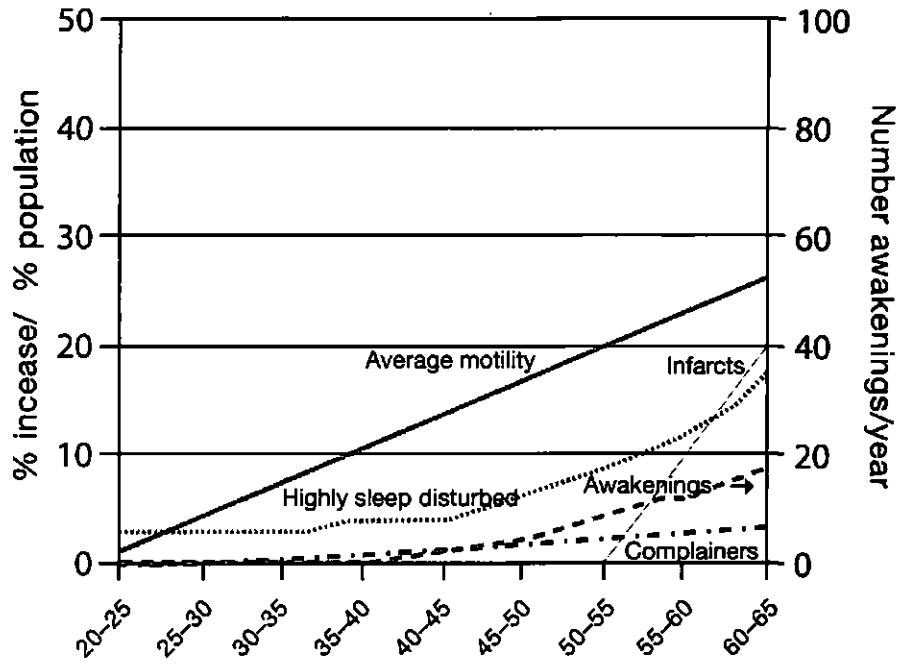


Fig. 4 Effects of aircraft noise at night*

**Average motility and infarcts are expressed in percent increase (compared to baseline number); the number of highly sleep disturbed people is expressed as a percent of the population; complainers are expressed as a % of the neighbourhood population; awakenings are expressed in number of additional awakenings per year.*

source dependent. Although L_{night} gives a good relation for most effects, there is a difference between sources for some. Train noise gives fewer awakenings, for instance. Once source is accounted for, the relations are reasonably accurate.

RECOMMENDATIONS FOR HEALTH PROTECTION

Based on the systematic review of evidence produced by epidemiological and experimental studies, the relationship between night noise exposure and health effects can be summarized as below. (Table 3)

Below the level of 30 dB $L_{night,outside}$, no effects on sleep are observed except for a slight increase in the frequency of body movements during sleep due to night noise. There is no sufficient evidence that the biological effects observed at the level below 40 dB $L_{night,outside}$ are harmful to health. However, adverse health effects are observed at the level above 40 dB $L_{night,outside}$, such as self-reported sleep disturbance, environmental insomnia, and increased use of somnifacient drugs and sedatives.

Therefore, 40 dB $L_{night,outside}$ is equivalent to the lowest observed adverse effect level (LOAEL) for night noise. Above 55 dB the cardiovascular effects become the major public health concern, which are likely to be less dependent on the nature of the noise. Closer examination of the precise impact will be necessary in the range between 30 dB and 55 dB as much will depend on the detailed circumstances of each case.

Average night noise level over a year $L_{night,outside}$	Health effects observed in the population
Up to 30 dB	Although individual sensitivities and circumstances may differ, it appears that up to this level no substantial biological effects are observed. $L_{night,outside}$ of 30 dB is equivalent to the no observed effect level (NOEL) for night noise.
30 to 40 dB	A number of effects on sleep are observed from this range: body movements, awakening, self-reported sleep disturbance, arousals. The intensity of the effect depends on the nature of the source and the number of events. Vulnerable groups (for example children, the chronically ill and the elderly) are more susceptible. However, even in the worst cases the effects seem modest. $L_{night,outside}$ of 40 dB is equivalent to the lowest observed adverse effect level (LOAEL) for night noise.
40 to 55 dB	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.
Above 55 dB	The situation is considered increasingly dangerous for public health. Adverse health effects occur frequently, a sizeable proportion of the population is highly annoyed and sleep-disturbed. There is evidence that the risk of cardiovascular disease increases.

Table 3
Effects of different levels of night noise on the population's health

A number of instantaneous effects are connected to threshold levels expressed in L_{Amax}. The health relevance of these effects cannot be easily established. It can be safely assumed, however, that an increase in the number of such events over the baseline may constitute a subclinical adverse health effect by itself leading to significant clinical health outcomes.

Based on the exposure-effects relationship summarized in Table 3, the night noise guideline values are recommended for the protection of public health from night noise as below.

Night noise guideline (NNG)	$L_{night,outside}$ = 40 dB
Interim target (IT)	$L_{night,outside}$ = 55 dB

Table 4
Recommended night noise guidelines for Europe

[1] $L_{night,outside}$ is the night-time noise indicator (L_{night}) of Directive 2002/49/EC of 25 June 2002: the A-weighted long-term average sound level as defined in ISO 1996-2: 1987, determined over all the night periods of a year; in which: the night is eight hours (usually 23.00 – 07.00 local time), a year is a relevant year as regards the emission of sound and an average year as regards the meteorological circumstances, the incident sound is considered, the assessment point is the same as for L_{den}. See *Official Journal of the European Communities*, 18.7.2002, for more details.

For the primary prevention of subclinical adverse health effects related to night noise in the population, it is recommended that the population should not be exposed to night noise levels greater than 40 dB of $L_{night,outside}$ during the part of the night when most people are in bed. The LOAEL of night noise, 40 dB $L_{night,outside}$, can be considered a health-based limit value of the night noise guidelines (NNG) necessary to protect the public, including most of the vulnerable groups such as children, the chronically ill and the elderly, from the adverse health effects of night noise.

An interim target (IT) of 55 dB $L_{night,outside}$ is recommended in the situations where the achievement of NNG is not feasible in the short run for various reasons. It should be emphasized that IT is not a health-based limit value by itself. Vulnerable groups cannot be protected at this level. Therefore, IT should be considered only as a feasibility-based intermediate target which can be temporarily considered by policy-makers for exceptional local situations.

RELATION WITH THE GUIDELINES FOR COMMUNITY NOISE (1999)

Impact of night-time exposure to noise and sleep disturbance is indeed covered in the 1999 guidelines, as below (WHO, 1999):

> "If negative effects on sleep are to be avoided the equivalent sound pressure level should not exceed 30 dBA indoors for continuous noise. If the noise is not continuous, sleep disturbance correlates best with L_{Amax} and effects have been observed at 45 dB or less. This is particularly true if the background level is low. Noise events exceeding 45 dBA should therefore be limited if possible. For sensitive people an even lower limit would be preferred. It should be noted that it should be possible to sleep with a bedroom window slightly open (a reduction from outside to inside of 15 dB). To prevent sleep disturbances, one should thus consider the equivalent sound pressure level and the number and level of sound events. Mitigation targeted to the first part of the night is believed to be effective for the ability to fall asleep."

The 1999 guidelines are based on studies carried out up to 1995 (and a few meta-analyses some years later). Important new studies (Passchier-Vermeer et al., 2002; Basner et al., 2004) have become available since then, together with new insights into normal and disturbed sleep. New information has made more precise assessment of exposure-effect relationship. The thresholds are now known to be lower than L_{Amax} of 45 dB for a number of effects. The last three sentences still stand: there are good reasons for people to sleep with their windows open, and to prevent sleep disturbances one should consider the equivalent sound pressure level and the number of sound events. The present guidelines allow responsible authorities and stakeholders to do this. Viewed in this way, the *night noise guidelines for Europe* are complementary to the 1999 guidelines. This means that the recommendations on government policy framework on noise management elaborated in the 1999 guidelines should be considered valid and relevant for the Member States to achieve the guideline values of this document.

CHAPTER 1

INTRODUCTION: METHODS AND CRITERIA

With regard to sleep and waking, we must consider what they are: whether they are peculiar to soul or to body, or common to both; and if common, to what part of soul or body they appertain: further, from what cause it arises that they are attributes of animals, and whether all animals share in them both, or some partake of the one only, others of the other only, or some partake of neither and some of both.

(Aristotle, On sleep and sleeplessness, 350 BC)

1.1 INTRODUCTION

1.1.1 EXISTING POLICY DOCUMENTS FOR NIGHT-TIME NOISE

The aim of this document is to present guidance for exposure to noise during sleep. What is already available?

There are three related documents at the international level:

- *Guidelines for community noise* (WHO, 1999)
- *Directive 2002/49/EC relating to the assessment and management of environmental noise* (European Commission, 2002b)
- *Position Paper on dose-effect relationships for night-time noise* (European Commission, 2004).

In Chapter 5 the relation with the *Guidelines for community noise* (1999) will be explained.

The European Union (EU) Directive relating to the assessment and management of environmental noise (or, as it is commonly known, the Environmental Noise Directive – END), establishes that Member States should create noise maps (2007) and action plans (2008) for parts of their territory. The noise maps should present noise levels expressed in the harmonized indicators L_{den} and L_{night}. Although in the first round only between 20% and 30% of the population will be covered, it is expected that through the use of harmonized methods and indicators a deeper insight will be gained into the exposure of the population to noise. The END does not, however, set any limit values: on the basis of the subsidiarity principle this is left to the Member States. The Directive does, however, require Member States to report on their limit values and express them in the standard indicators. On the CIRCA web site (Communication and Information Resource Centre Administrator, European Commission, 2006) an overview of the data reported to the Commission can be found. Out of the 25 Member States, 10 reported on the L_{night} limits. In Table 1.1 some of these data are summarized.

Due to differences in legal systems it is hard to predict what the actual effect of a certain limit value will be. It could be a relatively high value but rigidly enforced, or a very low value with no legal binding whatsoever.

The *Position Paper* on dose-effect relationships for night-time noise is foreseen in the END (Annex III) and aims to give the competent authorities a tool to evaluate the

impact on the population. However, it neither provides limit values nor guidelines. The same information that was used in the *Position Paper* also plays a role in these guidelines.

Table 1.1
Reported L_{night} limit values for road traffic noise in new residential areas

EU Member State	$L_{night,outside}$
France	62
Germany	49
Spain	45
Netherlands	40
Austria	50
Sweden	51 (converted from L_{Aeq} limit 30 dB(A) inside bedroom)
Finland	46
Hungary	55
Latvia	40
Estonia	45
Switzerland	50

Source: European Commission, 2006.

1.1.2 GENERAL MODEL

There is no doubt that a relation exists between sleep and health and well-being, as most of us know from personal experience. That does not mean, however, that this relation is simple. People who do not sleep well may not feel well the day after, but the reverse is also true: unfit people may have a disturbed sleep. Untangling the relations between health and disturbed sleep (night-time noise is only one of many causes) proved difficult, and Fig. 2.1 at the end of Chapter 2 shows why.

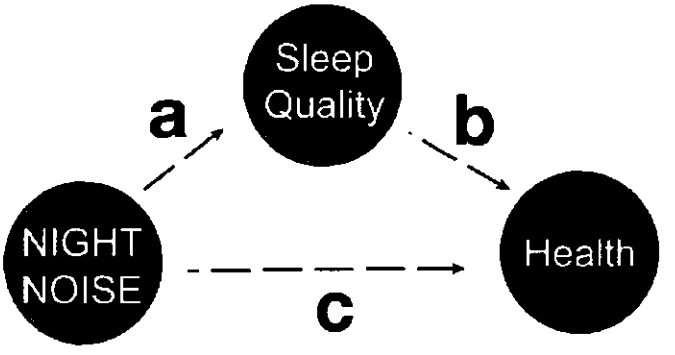


Fig. 1.1.
General structure of the report on the effects of night noise

The general structure of the report is given in Fig. 1.1: evidence for the effects of night-time noise on health (c) is supported by evidence on the indirect route via (a) and (b). In Chapter 2 the relations between sleep and health are examined (relation (b) in Fig. 1.1), and this involves clinical evidence from sleep laboratories, but also evidence from animal experiments. In Chapter 3 it is shown how noise disturbs sleep from the basic, autonomous level up to conscious awakenings: relation (a). Chapter 4 presents the evidence between night-time noise and health and well-being: relation (c) in Fig. 1.1. The last chapter, Chapter 5, then provides guidance on reducing health impacts caused by night-time noise exposure.

1.1.3 PROCESS OF DEVELOPING GUIDELINES

The WHO Regional Office for Europe started the night noise guidelines (NNGL) project with a grant from the European Commission's Directorate-General for Health and Consumer Affairs. In 2003, the WHO Regional Office for Europe set up a working group of experts to provide scientific advice for the development of guidelines for future legislation and policy action in the area of control and surveillance of night noise exposure. The review of available scientific evidence on the health effects of night noise was carried out by the working group to derive health-based guideline values. The contributions from the experts were reviewed by the team and integrated into draft reports following discussion at four technical meetings of the working group.

The first meeting of the working group was held in Bonn, June 2004. It was agreed that the experts would produce background papers on a number of topics identified and assigned at the meeting.

The second meeting in Geneva, December 2004, concentrated on such technical issues as exposure assessment, metrics, health effects and guideline set-up. The topic-specific experts presented the first drafts for the identified topics for detailed discussions at the meeting. The discussions concentrated on central issues such as exposure assessment and guideline derivation.

The third meeting in Lisbon, April 2005, reviewed the revised background papers, and discussed in detail the overall structure of the guidelines document, and the process of consensus building among the working group and stakeholders.

At the workshop of acoustics experts in The Hague, September 2005, a consensus was made on the use of L_{night} as the single indicator for guideline values as it effectively combines the information on the number of events and the maximum sound levels per event over a year.

In 2006, all the draft reports collected at previous meetings were compiled by Mr. Martin van den Berg into a coherent document on guidelines for exposure to noise at night. The latter was revised according to the comments collected though a peer-review by the working group experts.

At the concluding meeting in Bonn, December 2006, the working group and stakeholders from industry, government and nongovernmental organizations reviewed the contents of the draft document chapter by chapter, discussed several fundamental issues and reached general consensus on the guideline values. The final implementation report of NNGL project was submitted to the EU in early 2007.

The following countries and institutes contributed to the development of *Night noise guidelines for Europe* as project partners.

AUSTRIA:	Institute of Hygiene and Social Medicine, University of Innsbruck
CZECH REPUBLIC:	Charles University in Prague
DENMARK:	National Institute of Public Health
FRANCE:	INRETS/LTE–Laboratoire Transports et Environnement CNRS–Centre National de Recherche Scientifique
GERMANY:	Umweltbundesamt–Federal Environmental Agency Landesgesundheitsamt Baden-Württemberg

ITALY:	ARPAT–Environmental Protection Agency, Tuscany Region University of Rome "La Sapienza"– Center for Pediatric Sleep Disorders
NETHERLANDS:	TNO–Netherlands Organisation for Applied Scientific Research RIVM–National Institute of Public Health and the Environment
POLAND:	University of Warsaw, Clinic of Psychiatry of the Medical Academy
PORTUGAL:	IST–Instituto Superior Técnico
SLOVENIA:	Institute of Public Health of the Republic of Slovenia
SWEDEN:	University of Gävle, Centre for Built Environment
UNITED KINGDOM:	Queen Mary and Westfield College, University of London

In addition, WHO received advice and support from a number of national experts who participated in the working group. The affiliations of these additional expert advisers include:

CANADA:	Health Canada
GERMANY:	Forschungs- und Beratungsbüro Maschke
SWITZERLAND:	Bundesamt für Umwelt, Wald und Landschaft Universität St. Gallen, Institut für Wirtschaft und Ökologie
NETHERLANDS:	Ministry of Housing, Spatial Planning and Environment
UNITED KINGDOM:	Casella Stanger Environmental Consultants

Since the project report was published on the EU web site, various comments were received from experts who have not participated in the working group. The most critical points were regarding the achievability of guideline values in practice. Responding to these feedbacks, the WHO Regional Office for Europe, prepared a revision of guidelines and recommendations, and consulted with international experts and stakeholders including the EU. As of late 2008, it was agreed that the guideline should be based on the lowest observed adverse effects level (LOAEL) rather than the no observed effects level (NOEL). Interim target was also introduced as a feasibility-based level.

1.2 STRENGTH OF EVIDENCE

1.2.1 BASIC CONCEPTS

This document uses well-established practices from other disciplines and policy fields. Of main interest here are evidence-based medicine, the use of epidemiological evidence for environmental risk assessment and experiences with – principally– air quality guidelines.

The concept of "evidence" is further formalized, as variations in wording and scope are currently in use.

1.2.2 RISK ASSESSMENT AND RISK CONTROL

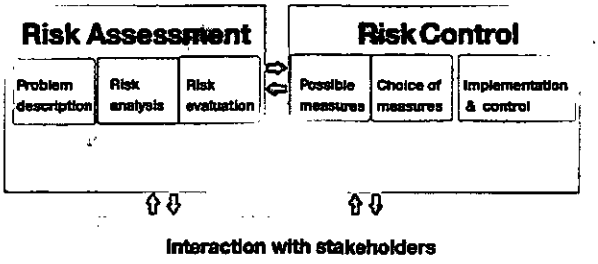


Fig. 1.2
Elements of risk control

Source:
Health Council of the Netherlands, 2004.

Fig. 1.2 outlines a general approach for risk assessment and control. This approach consists of the following steps:

1. problem description: assessing the impact on the population
2. risk analysis: evaluation of impact
3. risk evaluation: assessing impact considered undesirable
4. assessment of options to avoid or reduce impact
5. cost–benefit analysis of the options or of the mix of options
6. assessment of the preferred option
7. implementation and control.

It is important to observe that guideline values can be an input to, as well as an output of this process. At lower levels of decision (a particular infrastructure project, for instance) a preset guideline value reduces – intentionally – the degrees of freedom in the process. At the highest national or international level a guideline value is the outcome. As the scope of this document is to present the health consequences of night-time noise exposure (and not so much the economic outcomes of the choice of a certain value) it concentrates on the first three elements in the risk assessment block.

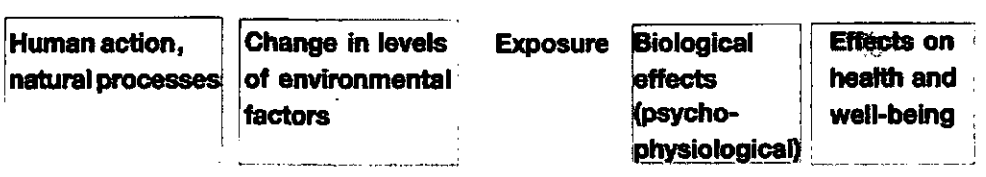
The following questions need to be addressed.

- What is the strength of the available evidence – what are the uncertainties?
- What is the health significance for the effects found?
- How serious is the impact on health?
- Does every instance of exposure lead to an effect and how are they related?
- How can the number of affected people be established?

1.2.3 CAUSE–EFFECT CHAIN

Underlying this approach is the notion of a cause–effect chain between environmental factors and health, symbolically simplified in Fig. 1.3.

Fig. 1.3
Cause-effect chain



Source: Health Council of the Netherlands, 2004.

There are important questions that need to be asked.

- Is there a causal relation between one link in the chain and the next?
- What are the intervening factors in that relation?
- How strong is the evidence for the relations?

The last question is the hardest to answer, as "strength of evidence" is not easy to express in simple numbers or labels. There are two forms of uncertainty: uncertainty because of variability of outcomes and uncertainty due to a lack of knowledge.

For the purpose of this document the following classification will be used, largely based on the IARC (International Agency for Research on Cancer) criteria accessible at http://monographs.iarc.fr/ENG/Preamble/currentb6evalrationale0706.php (see Table 1.2).

Table 1.2 Classification of evidence

Grade of evidence	Criteria
Sufficient evidence	A causal relation has been established between exposure to night-time noise and an effect. In studies where coincidence, bias and distortion could reasonably be excluded, the relation could be observed and it is plausible that the effect is (also) caused by the exposure.
Limited evidence	A relation was observed between exposure to night-time noise and an effect in studies where coincidence, bias and distortion could not reasonably be excluded. The relation is, however, plausible. A direct relation between cause and effect has not been observed, but there is indirect evidence of good quality and the relation is plausible. Indirect evidence is assumed if exposure leads to an intermediate effect and other studies prove that the intermediate effect leads to the effect.
Insufficient evidence	Available studies are of low quality and lack significance to allow conclusions about causality of the relation between exposure and effect. Plausibility of the relation is limited or absent.

1.2.4 PROCEDURE FOR DERIVING GUIDELINES

The following procedure was followed in order to derive an ordering of guideline values:

1. collection of relevant data
2. evaluation of data in terms of strength of evidence
3. evaluation of data in terms of biological effects, health and well-being
4. ranking of guideline values.

This procedure is essentially the same as in other guideline documents, although steps are more explicitly formalized. A major difference is that sound is a natural environmental quality, which makes defining a no-effect level a futile exercise. Therefore the choice was made for a series of levels with increasing severity of effects.

1.3 CONSIDERATIONS WITH REGARD TO NIGHT-TIME NOISE INDICATORS

Briefly, the fundamental choices of night-time noise indicators with respect to length of night, use of single event descriptors and long-term average are commented on to assist the reader in understanding the relations presented in later chapters.

1.3.1 LENGTH OF NIGHT

Time use studies (Centre for Time Use Research, 2006) show that the average time adult people are in bed is around 7.5 hours, so the real average sleeping time is somewhat shorter. Due to personal factors such as age and genetic factors there is considerable variation in sleeping time and in beginning and end times. For these reasons, a fixed interval of 8 hours is a minimal choice for night-time protection. From Fig. 1.4 it can be noted that around 50% of the population is protected with an interval of 8 hours and it would take a period of 10 hours to protect 80%. On Sundays, sleeping time is consistently one hour longer, probably due to people recovering from sleep debt incurred during the week. Data for other countries are readily available but this is the only study covering a long period in a consistent way. Fig. 1.5 (from a time use study in Portugal) shows that the stable pattern found in the Netherlands (Fig. 1.4) is not only typical for northern Europe, but also for the southern part. The pattern, however, seems to have shifted slightly. These figures stress that sleep times might be biologically fixed in humans, and culture has almost no influence.

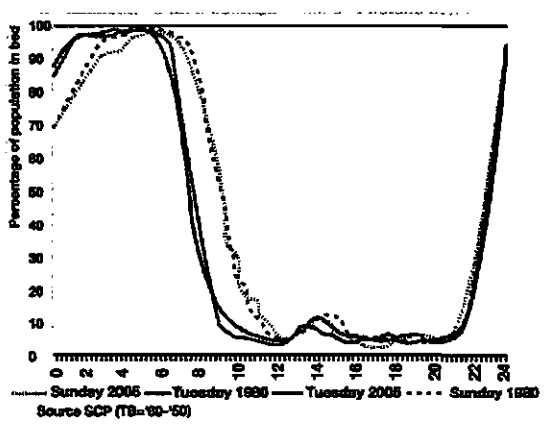


Fig. 1.4
Sleep pattern of Dutch population on weekdays and Sundays, 1980-2005

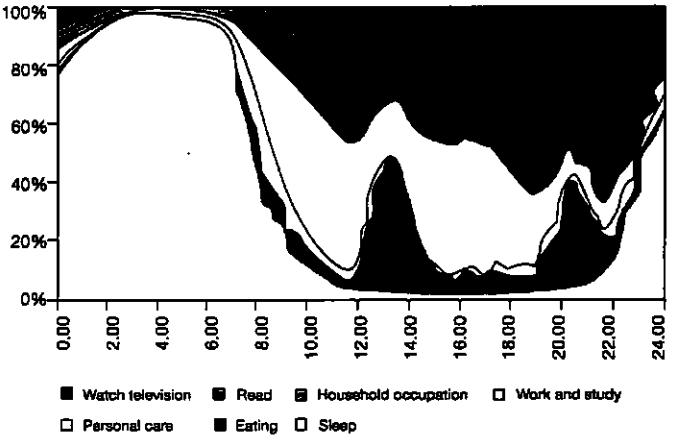


Fig. 1.5
Percentage of time that the Portuguese population spend asleep or in different activities

Source: http://www.ine.pt/prodserv/destaque/arquivo.asp, based on a study by the Instituto Nacional de Estatistica Portugal, 1999

1.3.2 EVENT OR LONG-TERM DESCRIPTOR

Much attention has been paid to the use of single event descriptors such as L_{Amax} (maximum outdoor sound pressure level) and SEL (sound exposure level). As the *Position Paper* on EU noise indicators (European Commission, 2000) points out, this is an important laboratory tool to describe instantaneous reactions to noise. But when it comes to long-term protection, the number of events is equally important. The possibility of predicting after-effects like sleepiness, reaction time, sleeping pill

use and health complaints, in particular, requires a combination of a number of events and their level instead of just the average L_{Amax} or average SEL. For events with a similar time pattern there is a relatively simple relation between L_{Amax} and SEL, and therefore between L_{Amax} and L_{night} (night-time noise indicator as defined

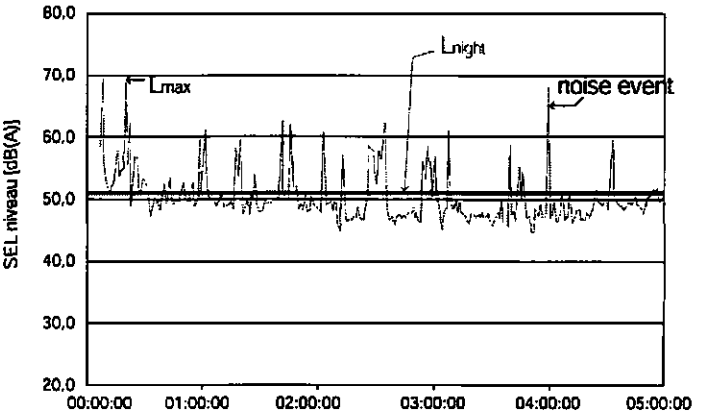


Fig. 1.6
Relation between L_{night}, L_{Amax} and SEL

Source: European Commission, 2000

by the END – see paragraph 1.3.4 below). Appendix 2 describes this in detail. For now let it suffice to say that a choice for an L_{night} level ties the L_{Amax} related effects to a maximum and therefore allows for a protective/conservative approach.

Fig. 1.6 is based on a sound recording in a bedroom for one night. The top of the peaks are the L_{Amax} levels, the total energy is the L_{night} (thick horizontal line). The sound energy in one event is the SEL (not represented). In reality the L_{night} is the average over all nights in one year. This reasoning applies also to the issue of long-term average. A value for an arbitrary single night will, except in extreme cases, bear no relationship to an individual's long-term health status, whereas a sustained sufficiently high level over a long period may.

1.3.3 NUMBER OF EVENTS

There is no generally accepted way to count the number of (relevant) noise events. Proposals range from the number of measured L_{Amax}, the number of units (vehicles, aeroplanes, trains) passing by, to the number exceeding a certain L_{Amax} level (commonly indicated by NAxx; NA70 is the number of events higher than 70 dB).

1.3.4 CONVERSION BETWEEN INDICATORS

1.3.4.1 Introduction

L_{night} is defined as the 1 year L_{Aeq} (exposure to noise) over 8 hours outside at the most exposed facade. For the purpose of strategic noise mapping and reporting the height is fixed at 4 metres. As L_{night} is a relatively new definition and because the studies rarely cover such a long period, the research data are rarely expressed in L_{night}. The most frequently used noise descriptor in sleep research is the L_{Amax} or SEL near the sleeper. This means that a considerable amount of conversion work needs to be done if relations are to be expressed in L_{night}. There are four issues:

- conversion between SEL and L_{Amax}
- conversion from instantaneous to long-term
- conversion from inside to outside
- conversion from (outside) bedroom level to most exposed façade.

Further background information on these issues is provided in section 1.3.5. This section details the conversions that are actually carried out.

1.3.4.2 SEL to L_{Amax}

SEL is only used for aircraft noise in this report and, according to Ollerhead et al. (1992) from ground-based measurements, the following relation was found:

$$SEL = 23.9+0.81*L_{Amax} \quad [1].$$

A more general approach can be used to estimate SEL for transportation noise.

If the shape of the time pattern of the sound level can be approximated by a block form, then $SEL \approx L_{Amax} + 10\lg t$, where t (in seconds) is the duration of the noise event. This rule can be used, inter alia, for a long freight train that passes at a short distance. When t is in the range from 3 to 30 seconds, then SEL is 5–15 dB higher than L_{Amax}. For most passages of aircraft, road vehicles or trains, the shape of the time pattern of the sound level can be better approximated with a triangle. If the sound level increases with rate a (in dB per second), and thereafter is at its maximum for a short duration before it decreases with rate -a, then $SEL \approx L_{Amax} - 10\lg(a) + 9.4$. Depending on the distance to the source, for most dwellings near transportation sources the rate of increase is in the order of a few dB per second up to 5 dB per second. When (a) is in the range from 9 dB to 1 dB per second, then SEL is 0–9 dB higher than L_{Amax}.

1.3.4.3 Events to long-term

When the SEL values are known (if necessary after converting from L_{Amax}) they can be converted to L_{night}. In general terms, the relation between L_{night} and SEL is:
$L_{night} = 10*\lg \sum_i 10^{SEL_i/10} - 10*\lg(T)$.

If all (N) events have approximately the same SEL level, this may be reduced to:

$$L_{night} = SEL + 10*\lg(N) - 70.2 \quad [2],$$

in which:
N = the number of events occurring in period T;
T = time during which the events occur in seconds. For a (night) year 10lg(T) is 70.2.

The notation adheres to the END where the L_{night} is defined as a year average at the most exposed facade. Any reference to an inside level is noted as such, that is, as $L_{night,inside}$. In order to avoid any doubt the notation $L_{night,outside}$ may be used, for instance in tables where both occur.

1.3.4.4 Inside to outside

As the L_{night} is a year value, the insulation value is also to be expressed as such. This means that if the insulation value is 30 dB with windows closed and 15 dB with windows open, the resulting value is 18 dB if the window is open 50% of the time. If these windows are closed only 10% of the time, the result is little more than 15 dB. The issue is complicated by the fact that closing behaviour is, to a certain extent, dependent on noise level. When results about effects are expressed with indoor (that is, inside bedrooms) exposure levels, they need to be converted to L_{night}, in accordance with the END definition. The most important assumption is the correction for inside levels to outside levels. An average level difference of 21 dB has been chosen, as this takes into account that even in well-insulated houses windows may be open a large part of the year. In general:

$$L_{night} = L_{night,inside} + Y \text{ dB} \quad [3].$$

Y is the year average insulation value of the (bedroom) facade. In this report a default value of 21 dB is used (see also section 1.3.5). It should be stressed that this conversion is thought to be highly dependent on local building habits, climate and window opening behaviour.

1.3.4.5 Most exposed facade

If an inside level is converted to an outside level with [3], it is assumed that this is equivalent to an L_{night} value on the most exposed facade. No information is available on bedroom position and use, so no explicit conversion factor can be given in this report.

This means that the effect estimated on the basis of L_{night} corresponds to an upper limit, because part of the bedrooms will be on a less exposed facade. If an estimate of the exposed population is based on a relation derived with [3], the actual prevalence will be less. From a practical point of view the most exposed facade safeguards protection in cases where there is a possibility that rooms can be swapped.

It should be pointed out that the above does not apply if a relation is based on L_{night} values which are directly measured or computed. These relations will show a large variation because of a misclassification effect, but they give a "correct" estimate of the prevalence of effects in the population. In other words, in some cases a low effect may be attributed to a high L_{night} because the bedroom is on the quiet side.

1.3.5 INSIDE/OUTSIDE DIFFERENCES

Night-time environmental noise affects residents mainly inside their homes. In order to protect residents inside their homes from noise from outside sources, attention should be focused on windows since they are generally the weakest points in the sound propagation path. Roofs must also be considered with regard to aircraft noise.

There are many types of window in the EU, varying from single thin panes within frames without additional insulation, to four-pane windows within insulated frames. The simplest types of facade have a sound reduction (from outside to inside) of usually less than 24 dB, and the most elaborate facades (built to cope with cold climates, for example), have sound reductions of more than 45 dB. In central Europe, most windows are double-glazed, mounted in a rigid and well-insulated frame. Their range of sound reduction is between 30 dB and 35 dB when closed.

When night-time environmental noise reaches high levels, residents tend to close their bedroom windows (cf. Langdon and Buller, 1977; Scharnberg et al., 1982; Schreckenberg et al., 1999; Diaz et al., 2001). The studies by Scharnberg et al. and Schreckenberg et al. found that more than 50% of bedroom windows are closed when outside road traffic noise levels exceed 55 dB (L_{Aeq}). These findings have been replicated in Sweden, according to recent results from the Swedish soundscape research programme on road traffic noise (Fig. 1.7). Nevertheless, while residents with closed windows reported a reduction of sleep disturbances due to noise, they also reported an increase in sleep disturbances due to poor ventilation. Schreckenberg et al. (1999) report a much steeper increase in the incidence of closed windows when road traffic noise reaches high levels than is the case with increased levels of railway noise. Even when night-time noise levels reach 55 B, only 35% of the residents exposed to railway noise reported that they closed their windows at night.

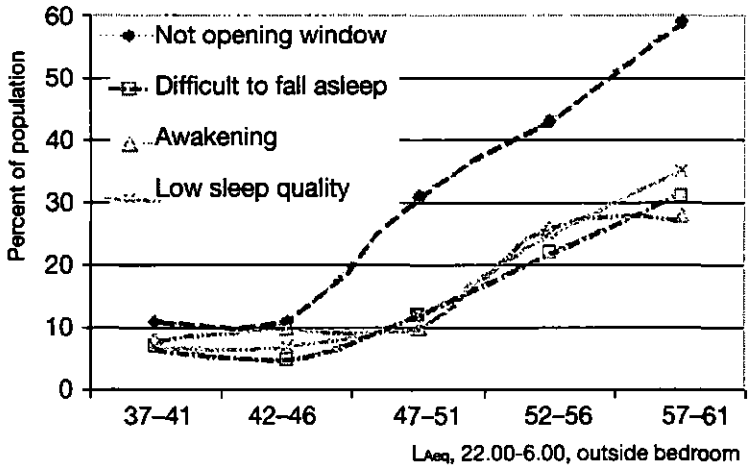


Fig. 1.7
Results from Swedish soundscape research programme on road traffic noise

Source: Öhrström, in European Commission, 2002a.

When windows are slightly open, outside sound levels are usually reduced by 10–15 dB. It should be kept in mind that most European residents want to keep their bedroom windows slightly open at night in order to provide proper ventilation (Scharnberg et al., 1982; Lambert and Plouhinec, 1985; Lambert and Vallet, 1994), and the WHO paper on community noise (WHO, 1999) also recommends that people should be able to sleep with their bedroom windows open.

Passchier-Vermeer et al. (2002) carried out detailed noise measurements inside and outside the bedroom and at the same time measured window position with sensors. The results (Table 1.3) showed that windows are fully closed only in 25% of the nights.

Window position	% nights
Closed	25
Slightly open	43
Hand width	23
Half open	5
Fully open	4

Table 1.3
Window positions during research period (April–November)

This results in average inside/outside differences of around 21 dB, with there being only a slight difference between single- and double-glazed windows (Table 1.4). The survey did not include dwellings which had been specifically insulated against noise. Nevertheless, there was a large variation in insulation values.

	Single-glazed window	Double-glazed window
Average difference at night	21.3	22.2

Table 1.4
Average inside/outside differences in dB

It should be stressed that this figure only applies to facades that have not been fitted with special appliances to reduce noise impact. To give an extreme example of where this general finding does not apply, rooms may be equipped with air conditioning so that windows can stay closed or could even be sealed. Less drastic provisions are sound-attenuated ventilation openings. Little is known, however, about the inhabitants' experiences (long-term use, appreciation) of these and other solutions. For example, sound-attenuated ventilation openings are sometimes blocked in order to cut out draughts.

1.3.6 BACKGROUND LEVEL

A simple definition of background level or "ambient noise" level is the noise that is not targeted for measurement or calculation. Background noise can interfere with the target noise in a number of ways. It can:

- mask the signal
- interact physically
- interact psychologically.

As this report is often dealing with low-level target noise, masking is an important issue. The other two interactions are more important in the domain of annoyance. Masking, however, is a complex process. The human auditory system is uncannily good at separating signals from "background". Microphones (and the software behind them) have been slow to catch up, as the unsatisfactory results show when it comes to automatically recognizing aircraft in long-term unmanned measuring stations.

The rule of thumb that a noise can be considered masked if the signal is 10 dB below the background is only valid if the noises have the same frequency composition and if they actually occur at the same time. This is particularly important to stress where L_{Aeq} levels are compared: even a relatively continuous motorway of 50 dB cannot mask aircraft noise of 30 dB, because this may be composed of five aircraft arriving at an L_{Amax} of 57 dB. Neither can birdsong, because the frequency domains do not overlap.

Another factor relevant for this report is that background levels are lower at night-time than they are in the daytime. This is true for most man-made noises, but also for the natural background levels as wind speeds at night slow down.

Most levels mentioned in this report do not take background levels into account – explicitly. Where long-term L_{Aeq} levels are related to effects like hypertension and self-reported sleep disturbance, background levels are ignored, but they could obscure the effect at the lower end of the scale. This then influences the lowest level where an effect starts to occur.

In sleep laboratory studies the background level is kept as low as possible, around 30 dB. The background of the instrumentation is 20 dB.

In semi-field experiments it has been found that background noise levels inside bedrooms are very low, partly because people tend to choose their bedrooms on the quiet side of the building. This may have the side-effect of exposing children to higher levels.

1.3.7 CHOICE OF INDICATORS FOR REGULATORY PURPOSES

From the scientific point of view the correct choice for a noise indicator is its performance in predicting the effect. There are, however, a number of additional criteria which may influence the choice. Firstly, for different health end points different indicators could be suitable. Further considerations are of a more political nature, as mentioned in the *Position Paper* on EU noise indicators (European Commission, 2000). Indicators should also be easy to explain to the public – intuitively understandable, avoiding unnecessary breaks with current practice and enforceable. This is probably why in many countries L_{Amax} is a popular indicator: it has undeniable qualities in these areas.

This is also the case for L_{Aeq} indicators for short periods of, for example, one or a few hours in the middle of the night. Other fashionable indicators are those looking at numbers above a threshold.

For these indicators the relation between health end points and their values is either not well established, or the correlation between them and current indicators is high, or the correlation between the indicator and an effect is low.

1.4 EXPOSURE IN THE POPULATION

1.4.1 NOISE LEVELS

Surprisingly little information is available on the exposure of houses to night-time noise. It is possible that, in a few years time, the END will lead to the creation of a substantial database on these levels, but up till now only two countries have detailed data available (Table 1.5).

	L_{night} in dB				
Country	40–45	46–50	51–55	56–60	61–65+
Switzerland (Müller-Wenk, 2002)	–	24%	14%	7%	2%
Netherlands (Nijland and Jabben, 2004)	25%	31%	19%	6%	1%

Table 1.5 Percentage of dwellings per noise class of L_{night} in dB

Notwithstanding the obvious differences between these two countries, the data show a remarkable similarity.

A first result of the END (see Table 1.6) comes from a study into night regulations for (large) airports (Wubben and Busink, 2004).

Airport	Number of inhabitants	Number of night operations per year	Night operations as percentage of daytime operations
Amsterdam	21 863	23 462	5.8%
Frankfurt	134 651	46 662	10.1%
London	477 289	26 465	5.7%
Paris	180 184	51 683	10.3%

Table 1.6 Number of inhabitants within 45 L_{night} contour

1.4.2 REPORTED NIGHT-TIME NOISE DISTURBANCE

Complaints about night-time exposure to noise are widespread and not exactly new: Roman writers used to complain about the racket in the streets at night (Juvenal, 160). Surprisingly, little detailed information is available today.

Nevertheless, data collected from a few Member States can help to give an impression of the order of magnitude of effects.

Fig. 1.8 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 (van Dongen et al., 2004) in which 4000 and 2000 people, randomly selected, were asked: "To what extent is your sleep disturbed by noise from [source mentioned]..." on a scale from 1 to 10. People recording the three highest points in the scale were considered "highly disturbed", according to an international convention. The totals are calculated from the number of people reporting serious sleep disturbance from one or more sources.

Unfortunately, comparable research data from other countries or regions is not available, and there is reason to believe that there may be considerable differences in the figures. Since this study is based on a survey conducted in the Netherlands, it is not representative for other Member States in the EU. General (not specific for night-time) annoyance data from Germany and the United Kingdom give an indication that similar numbers of people are affected.

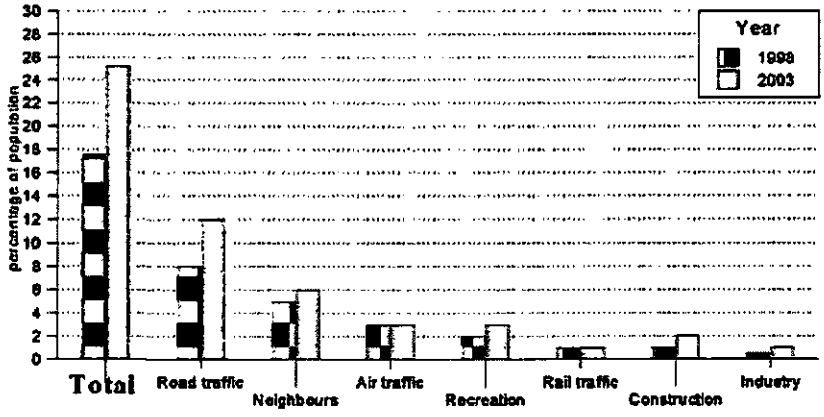


Fig. 1.8 Percentage of population highly disturbed by noise during sleep in the Netherlands

However, the fact that other noise nuisances may contribute significantly to overall sleep disturbance should not be overlooked. Further research on this topic is needed in order to gain an insight into the contribution of various noise sources to sleep disturbance.

1.5 CONCLUSIONS

The methods and criteria for deriving guidelines rest on well-established procedures from epidemiology. To relate the effects to the dose, standard metrics will be used wherever available. If possible, the values found in literature will be converted to avoid confusion. Most of the conversions are relatively straightforward and depend on physical laws; others, in particular the conversion between outside and inside levels, depend on local factors and should be used only if no other information is available.

Information about night-time noise exposure is relatively scarce, despite 10 EU Member States having limit values for night-time noise. The END could substantially increase this information (large-scale noise mapping is foreseen in 2007), increasing the demand for guidance.

CHAPTER 2

THE RELATION BETWEEN SLEEP AND HEALTH

A night of quiet and repose in the profound silence of Dingley Dell, and an hour's breathing of its fresh and fragrant air on the ensuing morning, completely recovered Mr Pickwick from the effects of his late fatigue of body and anxiety of mind.

(Charles Dickens, The Pickwick Papers, 1836)

2.1 SLEEP, NORMAL SLEEP, DEFINITIONS OF SLEEP DISTURBANCE, CHARACTERISTICS MECHANISMS, THE INSOMNIA MODEL

2.1.1 NORMAL SLEEP (OBJECTIVE MEASUREMENTS)

Sleep is part of living and, along with being awake, forms an inherent biological rhythm (Cooper, 1994). Normal sleep can be defined in an objective or subjective manner. The objective criteria are defined using a polysomnographic recording (PSG) of sleep, the method that measures different physiological functions during sleep. Minimal polygraphic requirements to measure sleep adequately include two channels of electroencephalography (EEG), one channel for the electrooculogram (EOG), and one channel for the submental electromyography (EMG). In routine PSG, additional channels are used to assess respiration, leg movements, oxygenation and cardiac rhythm (Ebersole and Pedley, 2003).

Scoring of sleep stages is usually done on an epoch-by-epoch basis, with a 30-second length used as a standard. Epochs are scored according to the guidelines of Rechtschaffen and Kales (1968). Each epoch is scored as the stage that occupies more than 50% of that epoch. Sleep can be divided into the following stages.

- **Arousal** is not a uniform concept and has been defined differently by different researchers. Commonly, the occurrence of alpha rhythms is required for EEG arousal. Depending on the additional requirements and on the length of time that the slower cortical rhythms are interrupted, arousals have been called, for instance, micro-arousal, minor arousal, EEG awakening or transient activation phases. The American Sleep Disorders Association (1992, 1997) devised a scoring system, taking sequences of 3–15 seconds into account for transient arousals which are not transferred to macroscopic behavioural awakening. Eleven further criteria must be met (see also Chapter 3, section 3.1.2).

- **Vegetative arousals** are activations of the sympathic nervous system.

- **Stage W** corresponds to the waking stage and is characterized by alpha activity or low-voltage, mixed-frequency EEG activity. Rapid eye movements (REMs), eye blinks, and tonic EMG activity are usually present.

- **Stage 1** is scored when more than 50% of an epoch is low-voltage, 2–7 Hertz (Hz) activity. Vertex waves may occur in late stage 1. Slow rolling eye movements lasting several seconds are routinely seen early in stage 1, but K complexes and sleep spindles are absent by definition. Tonic EMG activity is usually less than that of relaxed wakefulness.

- Stage 2 requires the presence of sleep spindles or K complexes, and less than 20% of the epoch contains delta activity. Bursts of sleep spindles must last at least 0.5 seconds before they can be scored. K complexes are defined as biphasic vertex sharp waves with a total duration of greater than 0.5 seconds.

- Stage 3 is scored when 20–50% of an epoch consists of delta activity that is 2 Hz or slower and is greater than 75 μV in amplitude. Sleep spindles may or may not be present.

- Stage 4 is scored when more than 50% of an epoch consists of delta activity that is 2 Hz or slower and is more than 75 μV in amplitude. Reliable differentiation of stage 3 and stage 4 sleep is difficult by visual inspection, and most laboratories combine stages 3 and 4 into a single determination of slow-wave sleep (SWS).

- Stage REM is characterized by relatively low-voltage, mixed-frequency EEG activity with episodic REMs and absent or markedly reduced axial EMG activity. Phasic EMG activity may occur, but tonic activity must be at a level that is as low as, or lower than, that during any other time in the study. Sleep spindles and K complexes are absent. Series of 2- to 5- Hz vertex-negative "saw-tooth waves" occur, particularly just before phasic REM activity. The requirements to score sleep as REM sleep are: REMs, low or absent axial EMG, and typical mixed-frequency EEG recording that does not preclude the scoring of REM.

Movement time is scored when more than 50% of an epoch is obscured by movement artefact. Movement time must be preceded or followed by sleep and is thus distinguished from movement occurring during wakefulness.

Additional sleep values are determined from each sleep study and contribute to the clinical interpretation of the study. These additional variables include the following.

- Recording time is the time elapsed between "lights out" and "lights on" at the end of the study.

- Total sleep time (TST) is the total time occupied by stage 1, stage 2, SWS and REM sleep.

- Sleep efficiency (SE) is defined as total sleep time divided by recording time and is expressed as a percentage.

- Sleep latency (SL) is the time from "lights off" to the first epoch scored as sleep. Some authors prefer to use the first epoch of stage 2 in order to be more confident about identifying the onset of sustained sleep. However, when sleep is very disrupted, there may be an extended interval from recognition of stage 1 until an epoch that can be scored as stage 2.

- REM latency is the time from sleep onset (as described earlier) to the first time period scored as REM, minus any intervening epochs as wakefulness.

- Sleep stage percentages (% in stage 1, stage 2, SWS and REM sleep) are determined by dividing time recorded in each sleep stage by total sleep time.

- Wake after sleep onset (WASO) is time spent awake after sleep onset.

The objective criteria defining normal sleep are based on: sleep latency, total sleep time, sleep efficiency and the number of awakenings, including cortical arousals. However, all these parameters are age-related, sometimes also gender-related, and may vary from one individual to another.

Normal sleep has a clearly defined architecture that is relatively stable. Predictable changes in sleep architecture occur with age. Beginning in middle age, SWS becomes less prominent, the number of awakenings increase, and sleep efficiency decreases. Published information on normal sleep can serve as an outline for normal values in PSG (Williams, Karacan and Hursch, 1974; see also Table 2.1), but each laboratory must study control subjects to identify any significant effects on sleep that result from differences in technique or environment (Ebersole and Pedley, 2003).

Table 2.1
Average normal values for adults of different ages

Sleep parameter (normal values)	20–29 years	40–49 years	60–69 years
TST (min)	419	389	407
Sleep efficiency (TST/TIB[a])	95%	91%	90%
WASO	1%	6%	8%
Stage 1 (% of TST)	4%	8%	10%
Stage 2 (% of TST)	46%	55%	57%
SWS (% of TST)	21%	8%	2%
REM (% of TST)	28%	23%	23%
Sleep latency (min)	15	10	8

[a] *Time in bed*
Source: Williams, Karacan and Hursch, 1974.

Passchier-Vermeer (2003a) reports that subjects not exposed to loud night noise typically report waking up one and a half to two times during an average sleep period, while the number of EEG awakenings including cortical arousals averages 10–12 per night (Table 2.2).

Table 2.2
Parameters of normal sleep

Subjects not exposed to loud night noise	Subjective report of number of awakenings	Number of EEG awakenings
Normal adult subjects	1.5–2	10–12

Source: Passchier-Vermeer, 2003a.

Night arousals result in fragmented sleep, which in turn leads to excessive daytime sleepiness (EDS). The gold standard for the assessment of EDS is the multiple sleep latency test (MSLT) (see Table 2.3), which provides an objective quantification of "sleepiness". The preceding night's sleep requires the PSG to ensure adequate sleep and to exclude sleep disruption. During the day, four or five nap times are scheduled every two hours. For each scheduled nap time the patient lies down and assumes a comfortable sleep position with the technician's instructions to "close your eyes and attempt to sleep". Each nap is terminated 20 minutes after the nap time started if no sleep occurred; or after 15 minutes of continuous sleep as long as sleep onset (SO) criteria are met before the end of 20 minutes; or after 20 minutes if the patient awakens, even if the patient has been asleep less than 15 minutes. The patient is instructed to stay awake between the nap periods.

Table 2.3
Mean sleep latency

Group	MSLT (min)	No REM SO (% of group)	1 REM SO (% of group)	2 or more REM SO (% of group)
Narcoleptics	2.9 ± 2.7	2	2	96
EDS (non-narcoleptic, non-sleep apnoeic)	8.7 ± 4.9	92	8	0
Controls	13.4 ± 4.3	100	0	0

Source: Ebersole and Pedley, 2003.

2.1.2 DEFINITIONS OF DISTURBED SLEEP

Sleep disorders are described and classified in the International Classification of Sleep Disorders (ICSD) (American Academy of Sleep Medicine, 2005).

When sleep is permanently disturbed and becomes a sleep disorder, it is classified in the ICSD 2005 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. Secondary deficits may result, including deficits in concentration, attention and cognitive performance, reduced vigilance, daytime fatigue, malaise, depressed mood and irritability. The exact prevalence is not known. Fewer than 5% of patients seen at sleep disorder centres receive this diagnosis. The sex ratio is not known. The disorder may occur at any age, although the elderly are at more risk for developing this condition (American Academy of Sleep Medicine, 2005).

2.1.2.1 Insomnia

In the ICSD 2005 the section on insomnia includes a group of sleep disorders all of which have in common the complaint of insomnia (adjustment insomnia, psychophysiological insomnia, paradoxical insomnia, idiopathic insomnia, etc.), defined as repeated difficulty with sleep initiation, duration, consolidation or quality that occurs despite adequate time and opportunity for sleep and results in some form of daytime impairment. Insomnia is a symptom that often arises from primary medical illness, mental disorders and other sleep disorders, but may also arise from abuse or exposure. However, the general criteria for insomnia are the same for all subgroups of insomnias.

2.1.2.2 General criteria for insomnia

A. A complaint is made concerning difficulty initiating sleep, difficulty maintaining sleep, waking up too early or sleep that is chronically non-restorative or poor in quality. In children, the sleep difficulty is often reported by the carer and may consist of observed bedtime resistance or inability to sleep independently.

B. The above sleep difficulty occurs despite adequate opportunity and circumstances for sleep.

C. At least one of the following forms of daytime impairment related to the nighttime sleep difficulty is reported by the patient:

- fatigue or malaise
- attention, concentration, or memory impairment
- social or vocational dysfunction or poor school performance
- mood disturbance or irritability

- daytime sleepiness
- motivation, energy, or initiative reduction
- proneness to errors or accidents at work or while driving
- tension, headaches, or gastrointestinal symptoms in response to sleep loss
- concerns or worries about sleep.

Defining the cause of a sleep/wake disturbance in an insomnia patient is a complex task since it is often multifactorial. In fact, a confluence of factors that support multiple insomnia diagnoses may be judged important in many patients with insomnia. Although selection of a single diagnosis is preferable and this selection may be appropriate, such a selection should not necessarily imply the absence of a subset of factors relevant to an alternate diagnosis. When criteria for multiple insomnia diagnosis are met, all relevant diagnosis should be assigned.

2.1.2.3 Environmental sleep disorder

In the ICSD 2005, environmental sleep disorder is listed in the category of "other sleep disorders". Noise-induced sleep disturbance is one of the disturbing environmental factors that cause a complaint of either insomnia or daytime fatigue and somnolence.

The diagnostic criteria for environmental sleep disorder are the following.

A. The patient complains of insomnia, daytime fatigue or a parasomnia. In cases where daytime fatigue is present, the daytime fatigue may occur as a result of the accompanying insomnia or as a result of poor quality of nocturnal sleep.

B. The complaint is temporally associated with the introduction of a physically measurable stimulus or environmental circumstance that disturbs sleep.

C. It is the physical properties, rather than the psychological meaning of the environmental factor, that accounts for the complaint.

D. The sleep disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use or substance use disorder.

The prevalence of environmental sleep disorder is not known. Fewer than 5% of patients seen at sleep disorder centres receive this diagnosis.

International standardization and quantification for measurement of the depth of sleep is based on Rechtschaffen and Kales criteria from 1968. Sleep is divided into 30-second epochs, and a phase is only assessed if the specific features are evident for more than 50% of the epoch length. For example, wakefulness is scored when at least 15 seconds of continuous awakening is present. Arousal reactions not leading to macroscopic awakening were not included in the definition by Rechtschaffen and Kales. With the arousals as described by the American Sleep Disorders Association (1992) it is possible to display subvigilant sleep fragmentation, caused by intrinsic sensory and autonomic alarm reactions. An arousal index providing the arousal density (events per hour of sleep) was taken as a measure of the degree of severity. In one hour, 10–20 arousals are considered as normal in healthy adults. However, the use of EEG arousals with the American Sleep Disorders Association definition provides no sufficient explanation of daytime sleepiness (Ali, Pitson and Stradling 1996; Ayas et al., 2001) unless they are accompanied by vegetative arousals.

Regarding noise, different vigilance level assessments in various functional systems are important. Dumont, Montplaisir and Infante-Rivard (1988) proposed investigations of

vegetative, motor and sensory functions independently of each other. One of the possible factors indicating disturbed sleep is a vegetative arousal index. A vegetative arousal index of more than 30 per hour is certainly considered as serious, more than 20 per hour as intermediate and more than 10 as a light form of sleep disorder.

With respect to insomnia (section 2.1.2), there is the possibility of misclassification if the general practitioner (GP) overlooks excessive noise as the possible cause of the complaint. There is also the possibility that the insomnia is aggravated by noise.

2.1.3 CONCLUSIONS

Published information on normal sleep can serve as an outline for normal values in PSG. However, these values are only informative, because each sleep laboratory must study control subjects to identify any significant effects on sleep that result from differences in technique or environment. Excessive daytime sleepiness is a consequence of disturbed night sleep and can be objectively assessed by MSLT, which provides an objective quantification of "sleepiness".

2.2 LONG-TERM HEALTH RISK MEDIATED BY SLEEP DISTURBANCES

2.2.1 STRESSORS, NEUROBEHAVIOURAL DATA AND FUNCTIONAL NEUROIMAGING

It is generally accepted that insufficient sleep and particularly sleep loss has a great influence on metabolic and endocrine functions (Spiegel, Leproult and van Cauter, 1999), as well as on inflammatory markers, and contributes to cardiovascular risk. C-reactive protein (CRP) as a major marker of the acute phase response to inflammatory reaction promotes secretion of inflammatory mediators by vascular endothelium and may be therefore directly involved in the development of atherosclerotic lesions. CRP as a risk predictor of strokes and heart attacks linearly increases with total and/or partial sleep loss (Meier-Ewert et al., 2004).

An additional factor, closely linked to cardiovascular health, glucose regulation and weight control, is leptin. Leptin is one of the major regulators of energy homeostasis and its circadian profile interacts closely with sleep.

Secretion of leptin increases at night and decreases during the day. A decreased leptin level, that is connected with sleep loss, increases appetite and predisposes to weight gain, impaired glucose tolerance and impaired host response.

Other studies have focused on how sleep loss affects neurobehavioural functions, especially neurocognitive performance. Functional brain imaging and EEG brain mapping studies show that the patterns of functional connectivity between brain regions, evident in the performance of specific cognitive tasks, are altered by sleep loss (NCSDR, 2003). According to this finding, the maintenance of sustained performance during sleep loss may depend upon regional functional plasticity.

Cumulative waking, neurocognitive deficits and instability of state that develop from chronic sleep loss have a basis in a neurobiological process that can integrate homoeostatic pressure for sleep across days. Increased efforts have helped to determine the roles of REM and non-REM sleep in memory.
Functional brain imaging techniques, such as positron emission tomography (PET), functional magnetic resonance imaging (fMRI), magnetic resonance spectroscopy (MRS), single photon emission computed tomography (SPECT) and magneto-electroencephalography (MEG), have recently been analysed in a study of sleep and waking (NCSDR, 2003). These techniques allow the measurement of metabolic and neurochemical activity throughout the brain, and can reveal dynamic patterns of regional cerebral activity during various brain states, including stages of sleep and levels of alertness during wakefulness or during functional challenge. These techniques can also help identify both normal and abnormal sleep/wake processes.

In the last five years, functional neuroimaging techniques (particularly PET) have revealed that non-REM sleep is associated with the deactivation of central encephalic regions (brainstem, thalamus, basal ganglia) and multimodal association cortices (for instance, prefrontal and superior temporal/inferior parietal regions). REM sleep is characterized by reactivation of all central encephalic regions deactivated during non-REM sleep except the multimodal association areas. PET studies during sleep-deprived wakefulness have revealed regional cerebral deactivations that are especially prominent in prefrontal and inferior parietal/superior temporal cortices, and in the thalamus. This pattern is consistent and helpful in explaining the nature of cognitive performance deficits that occur during sleep loss. As revealed by means of fMRI techniques during cognitive task performance, the maintenance of performance following sleep loss may be a function of the extent to which other cortical brain regions can be recruited for task performance in the sleep-deprived state.

PET, SPECT and fMRI studies have revealed, in depressed patients, initially elevated activation in anterior cingulate and medial orbital cortices (NCSDR, 2003). In these patients, sleep deprivation reduces this regional hyperactivation, and improvements in mood are a function of the extent to which this activity is reduced. These studies point to possible mechanisms by which antidepressant drugs may exert their effects. Further research should be oriented towards neuroimaging and measurements of changes in the brain's metabolic activity at the neurotransmitter level.

2.2.2 SIGNALS MEDIATED BY A SUBCORTICAL AREA (THE AMYGDALA), THE ROLE OF STRESS HORMONES IN SLEEP DISTURBANCE AND THEIR HEALTH CONSEQUENCES

Experimental as well as clinical studies (Waye et al., 2003; Ising and Kruppa, 2004) showed that the first and fastest signal of stressors introduced by noise is detected and mediated by a subcortical area represented by the amygdala while the stress response to noise is mediated primarily by the hypothalamus-pituitary-adrenal (HPA) axis. A major intrinsic marker of the circadian rhythm is in the level of circulating corticosteroids derived from activity within the HPA axis. A protracted stress response with activation of the HPA axis is a major physiological response to environmental stressors. The cortisol response to awakening is an index of adrenocortical activity, and long-term nocturnal noise exposures may lead, in persons liable to be stressed by noise, to permanently increased cortisol concentration above the nor-

mal range. The hypothesis that an increased risk of cardiovascular diseases is connected with stress concepts is generally accepted (Ekstedt, Åkerstedt and Soderstrom, 2004; Ising and Kruppa, 2004). Stress reactions may lead to derangement of normal neurovegetative and hormonal processes and influence vital body functions. Cardiovascular parameters such as BP, cardiac function, serum cholesterol, triglycerides, free fatty acids and haemostatic factors (fibrinogen) impede the blood flow through increased viscosity and presumably blood sugar concentration as well. Insulin resistance and diabetes mellitus, stress ulcers and immune system deficiency are also frequent consequences of stress reaction. Disturbed sleep may lead to immunosuppression and diminished protein synthesis (Horne, 1988).

As well as nonspecific effects of the stress response on the functioning of the immune system, there is considerable evidence for a relation between sleep, especially SWS, and the immune system (Brown, 1992). This evidence includes surges of certain immune parameters and growth hormones at onset of SWS, correlation of non-REM sleep, total sleep time and sleep efficiency with natural killer cell activity, and correlation of SWS with recovery from infections. These data, taken together with information on the effect of intermittent transportation noise on SWS during the first sleep cycles and overnight, suggest that the immune response could also be impacted directly by environmental noise during sleep (Carter, 1996).

2.2.3 SLEEP RESTRICTION, ENVIRONMENTAL STRESSORS (NOISE) AND BEHAVIOURAL, MEDICAL AND SOCIAL HEALTH CONSEQUENCES OF INSUFFICIENT SLEEP: RISK OF MORBIDITY AND MORTALITY

Sleep restriction due to environmental stressors leads to primary sleep disorders, but health is also influenced by the consequence of stress response to noise mediated by the HPA axis and/or by restriction of specific sleep stages (see above).

Sleep restriction leads, in approximately 40% of affected subjects, to daytime sleepiness that interferes with work and social functioning. Excessive daytime sleepiness is thus a major public health problem, as it interferes with daily activities, with consequences including cognitive problems, motor vehicle accidents (especially at night), poor job performance and reduced productivity (Lavie, Pillar and Malhotra, 2002). In the last decade, experimentally based data have been collected on chronic restriction of sleep (by 1–4 hours a night), accumulating daytime sleepiness and cognitive impairment. Most individuals develop cognitive deficits from chronic sleep debt after only a few nights of reduced sleep quality or quantity. New evidence suggests additional important health-related consequences of sleep debt related to common viral illnesses, diabetes, obesity, heart disease, depression and other age-related chronic disorders.

The effects and consequences of sleep deprivation are summarized in Table 2.4 (Lavie, Pillar and Malhotra, 2002).

The relationship between sleep quantity and quality and estimates of morbidity and mortality remains controversial. Epidemiological data (NCSDR, 2003) suggest that habitually short sleep (less than 6 hours sleep per night) is associated with increased mortality. Epidemiological studies in recent years elucidated, however, that too much sleep is a problem as well. Kripke et al. (2002) evaluated a questionnaire study of 1.1 million men and women aged 30–102 years and found the lowest mortality risk between respondents sleeping 7 hours per night.

Type	Short-term	Long-term
Behavioural	Sleepiness Mood changes Irritability and nervousness	Depression/mania Violence
Cognitive	Impairment of function	Difficulty in learning new skills Short-term memory problems Difficulty with complex tasks Slow reaction time
Neurological	Mild and quickly reversible effects	Cerebellar ataxia, nystagmus, tremor, ptosis, slurred speech, increased reflexes, increased sensitivity to pain
Biochemical	Increased metabolic rate Increased thyroid activity Insulin resistance	Decreased weight despite increased caloric intake (in animals) Diabetes, obesity (in humans)
Others	Hypothermia Immune function impairment	Susceptibility to viral illness

Table 2.4 Consequences of sleep deprivation

Mortality risk significantly increased when sleep duration was less than 6 or higher than 8 hours per night. Other authors have also published similar results (Patel et al., 2004; Tamakoshi and Ohno, 2004). Patel et al. (2004) in a prospective study of sleep duration and mortality risk in 5409 women confirmed previous findings that mortality risk is lowest among those sleeping 6–7 hours per night. The mortality risk for death from other causes significantly increased in women sleeping less than 5 and more than 9 hours per night. It is not clear how the length of sleep can increase this risk, although animal evidence points to a direct link between sleep time and lifespan (see section 2.5 in this chapter). Up to now, no epidemiological prospective study has been published that examines the relationship between sleep and health outcomes (morbidity and mortality) with subjective and objective estimates. Recent studies, however, show that sleep duration of least 8 hours is necessary for optimal performance and for prevention of daytime sleepiness and accumulation of sleep debt.

Environmental stressors, including noise, mostly cause insomnia. Insomnia also involves daytime consequences, such as tiredness, lack of energy, difficulty concentrating and irritability. A reasonable prevalence estimate for chronic insomnia in the general population is about 10%; for insomnia of any duration or severity this rises to between 30% and 50%, and incidence increases with ageing. In the course of perimenopausal time, women are particularly vulnerable to developing this complaint. The major consequences and co-morbidity of chronic insomnia (see Table 2.5) consist of behavioural, psychiatric and medical problems. Several studies also report a higher mortality risk (Zorick and Walsh, 2000).

Table 2.5 Consequences of chronic insomnia

Type	Consequence
Behavioural	Poor performance at work, fatigue, memory difficulties, concentration problems, motor vehicle accidents
Psychiatric	Depression, anxiety conditions, alcohol and other substance abuse
Medical	Cardiovascular, respiratory, renal, gastrointestinal, musculoskeletal disorders Obesity Impaired immune system function
Mortality	Increased risk is reported

2.2.3.1 *Behavioural consequences*

Transient (short-term) insomnia is usually accompanied by spells of daytime sleepiness and performance impairment the next day. Persistent (long-term) insomnia tends to be associated with poor performance at work, fatigue, memory difficulties, concentration problems and twice as many fatigue-related motor vehicle accidents as in good sleepers.

2.2.3.2 *Psychiatric conditions*

Epidemiological research indicates that the prevalence of any psychiatric disorder is two or three times higher in insomniacs. The risk of depression as a co-morbid state appears to be particularly strong, being approximately four times more likely in insomnia patients. Furthermore, insomnia may be an early marker for psychiatric disorders such as depression, anxiety conditions and alcohol abuse. Anxiety has been quite commonly found in insomniacs compared with the general population. About 25–40% of insomnia patients are estimated to have significant anxiety, and the abuse of alcohol and other substances is increased in insomniacs relative to good sleepers (Ford and Kamerow, 1989). Samples of unselected psychiatric patients have about a threefold increase in the frequency of insomnia compared with healthy control subjects, and the severity of the condition correlates with the intensity of the psychiatric symptoms. Among samples of outpatients who consulted their GPs for insomnia, about 50% presented with psychiatric conditions, and about half of these patients were probably depressed (Zorick and Walsh, 2000).

2.2.3.3 *Medical consequences*

Insomnia has been statistically associated with various medical conditions, including disorders of the cardiovascular, respiratory, gastrointestinal, renal and musculoskeletal systems. A large series of insomniac patients showed that poor sleepers are more than twice as much at risk of ischaemic heart disease (IHD) as good sleepers (Hyyppa and Kronholm, 1989). Insomnia patients were also shown (Irwin, Fortner and Clark, 1995) to have impaired immune system function. Keith et al. (2006) hypothesize a connection between sleep deficit as one of the possible factors to explain the rise in obesity. Hormone changes and animal experiments apparently support this.

2.2.3.4 *Mortality risk*

Only a few epidemiological studies deal with mortality in insomniacs. According to Kripke et al. (1979), reduced sleep time is a greater mortality risk than smoking, hypertension and cardiac disease. Higher death rates are also reported among short sleepers. In this respect, however, further systematic investigation of the link between insomnia, short sleep and death is desirable.

2.3 RISK GROUPS

Risk groups are people who may be either sensitive (showing more reaction to a stimulus than the average), are more exposed (also called vulnerable) or both.

2.3.1 HEALTH EFFECTS OF DISTURBED SLEEP IN CHILDREN

Although children appear to tolerate a single night of restricted sleep with no detrimental effect on performance of brief tasks, perhaps more prolonged restriction and prolonged tasks similar to those required in school would show negative effects. In addition, as children seem to require more time to recuperate fully from nocturnal sleep restriction than adults (Carskadon, Harvey and Dement, 1981a), with additional nights of partial sleep deprivation, cumulative sleepiness might become a significant problem.

Empirical data that directly address the effects of repeated sleep loss on children's mood or cognitive function are sparse. A range of clinical and observational data support a general picture that inadequate sleep results in tiredness, difficulties in focusing attention, low thresholds for negative reactions (irritability and easy frustration), as well as difficulty in controlling impulses and emotions. In some cases, these symptoms resemble attention-deficit hyperactivity disorder (ADHD).

Environmental noise experienced at home during night-time is a sometimes unpredictable and most often discontinuous event (for example traffic noise, aircraft or train noise, a noisy environment for other reasons, for instance proximity with a discotheque, etc.), that might lead to sleep disruption without leading to behavioural awakenings through the alteration of sleep microstructure, in a similar manner as other sleep disturbing events such as respiratory disturbances.

Therefore, in respect of clinical settings, we can assume that, in children, an experimental model for the consequences of noise can be represented by respiratory disturbances during sleep, such as snoring, upper airway resistance syndrome (UARS) or obstructive sleep apnoea syndrome (OSAS), either for the noise produced by snoring or for the effects on the arousal system and sleep microstructure.

For this reason, this section describes the well-studied effects of sleep breathing disorders on children's health and then evaluates the indicators of sleep disruption from the point of view of sleep microstructure.

In the literature few data on the medium- and long-term effects of disturbed sleep in children are available from the longitudinal point of view. Most reports focused on respiratory disturbances during sleep as a theoretical model to evaluate the long-term effects of disturbed sleep in children. This review reports on the medium- and long-term negative consequences of disturbed sleep on cognitive functioning, behaviour, mental health, growth and the cardiovascular system.

2.3.1.1 Sleep deprivation in children

The effects of sleep deprivation were evaluated in children. The findings only indirectly pertain to this general report, although repeated noise-induced sleep disruption favours sleep deprivation.

In another study, 15 healthy infants aged 78+/-7 days were studied during two nights: one night was preceded by sleep deprivation (kept awake for as long as pos-

sible beyond their habitual bedtime: median onset 150 min; range 0–210 min) (Thomas et al., 1996). Of the 15 children, 13 slept supine, 12 were breastfed and 4 were from smoking parents. Following sleep deprivation, infants maintained a greater proportion of quiet sleep (44% vs. 39%; p=.002). There was no measurable change in arousal propensity by either graded photic (stroboscope) or auditory stimuli (1 kHz pure tone, delivered in the midline of the cot, from 73 dB and increased in 3 dB steps to 100 dB) during quiet sleep.

Forty-nine Finnish children (26 boys/23 girls) aged 7–12 years were interviewed together with their parents and school teachers, and recorded for 72 hours with a belt-worn activity monitor during weekdays.

The objectively measured true sleep time was associated with psychiatric symptoms reported by a teacher. The decreased amount of sleep was associated more with externalizing than internalizing types of symptoms (aggressive and delinquent behaviour, attention, social, and somatic problems) (Aronen et al., 2000).

In a survey, it was also shown that out of 100 Belgian school children, aged between 9 and12 years, those with poor sleep (insomnia) were also showing more frequent poor school performance (failure to comply with expected grades) than good sleepers. The relation between poor sleep and a noisy environment was, however, not evaluated (Kahn et al., 1989).

2.3.1.2 Neurocognitive manifestations

Several studies in adults have shown that sleep fragmentation and hypoxaemia can result in daytime tiredness and loss of concentration, retrograde amnesia, disorientation, morning confusion, aggression, irritability, anxiety attacks and depression. One could hypothesize that sleep fragmentation and hypoxaemia would affect the neuropsychological and cognitive performance also in children, where the impact of abnormal sleep may be even greater than in adults. In fact, neurocognitive and behavioural deficits and school problems have been reported recently in children with sleep-related obstructive breathing disorders (SROBD).

2.3.1.3 Attention capacity

This represents the ability to remain focused on a task and appropriately attend to stimuli in the environment. Taken together the studies to date indicate that children with SROBD are less reflective, more impulsive, and show poorer sustained and selective attention. Blunden et al. (2000) reported that, compared to 16 controls, 16 children with mild SROBD showed reduced selective and sustained attention. Owens-Stively et al. (1997) suggested a dose–response in attention–impulsivity with moderate to severe obstructive sleep apnoea syndrome (OSAS) children showing greater deficits than mild OSAS children. Importantly, early treatment showed that attention deficits in children with OSAS are reversible (Guilleminault et al., 1982b). In another study, 12 children with moderate to severe OSAS showed a significant reduction in inattention and an improvement in aggressive and hyperactive behaviours and vigilance after surgical treatment (Ali, Pitson and Stradling, 1996).

2.3.1.4 Memory

Rhodes et al. (1995) found inverse correlations between memory and learning performance and the apnoea hypopnea index in 14 morbidly obese children. Smaller deficits were observed by Blunden et al. (2000), who found in their sample of children with mild SROBD that mean global memory performance was in the lower end of the normal range compared to controls.

A recent study using actigraphy in normal school-age children showed that lower sleep efficiency and longer sleep latency were associated with a higher percentage of incorrect responses in working memory tasks; shorter sleep duration was associated with performing tasks at the highest load level only. Also, controlling for age, gender, and socioeconomic status, sleep efficiency and latency were significantly associated with the mean incorrect response rate in auditory working memory tasks. This study showed that sleep quality (evaluated as sleep efficiency = 100* [sleep + light sleep]/duration) is more strongly associated with performance in working memory tasks than sleep duration, suggesting that in assessing sleep, attention should be directed not only at the amount of sleep but also at sleep quality.

2.3.1.5 Intelligence

Inspection of the mean IQ scores reported in the study by Rhodes et al. (1995) suggested that their sample of five obese children with moderate to severe OSAS performed in the borderline range whereas controls performed in the normal range. Blunden et al. (2000) showed smaller deficits in children with mild SROBD whose mean verbal and global IQ were in the lower end of the normal range.

It remains unclear as to whether the putative negative effects of SROBD on intelligence are global in nature or confined to specific areas such as verbal rather than performance or visuospatial intelligence and whether these impairments can be reversed.

2.3.1.6 Learning and school performance

It has been widely reported (Stradling et al., 1990; Guilleminault et al., 1996; Richards and Ferdman, 2000) that children with SROBD show reduced academic performance and learning.

Weissbluth et al. (1983) found that poor academic achievers had a higher prevalence of night-time snoring (38% vs. 21%) and breathing difficulties (13% vs. 6%). Out of 297 children with SROBD (22% snorers and 18% sleep-associated gas exchange abnormalities), 40% were in the lowest 10th percentile of academic performance (Gozal, 1998) and SROBD in early childhood may continue to adversely affect learning in later years (Gozal and Pope, 2001). Gozal (1998) found in his sample of poor academic achievers that school grades improved post-adenotonsillectomy in treated but not untreated children.

As well as those with SROBD, healthy normal children with fragmented sleep (measured by actigraphy) also showed lower performance on neurobehavioural functioning (NBF) measures, particularly those associated with more complex tasks, and also had higher rates of behavioural problems (Sadeh, Gruber and Raviv, 2002). Furthermore, in normal children without sleep disorders, modest sleep restriction can also affect children's NBF. Sadeh, Gruber and Raviv (2003) monitored 77 children for 5 nights with activity monitors. On the third evening, the children were asked to extend or restrict their sleep by an hour on the following three nights. Their NBF was reassessed on the sixth day following the experimental sleep manipulation and showed that sleep restriction led to improved sleep quality and to reduced reported alertness.

These studies suggest that fragmented sleep or insufficient sleep is highly relevant during childhood and that children are sensitive to modest alterations in their natural sleep duration.

Early reports documented that untreated OSAS can have long-term negative effects, such as failure to thrive, cor pulmonale and mental retardation. These severe consequences are less common now due to early diagnosis and treatment, but recent reports have focused on other long-term effects mainly related to neurocognitive deficits, such as poor learning, behavioural problems and ADHD (Marcus, 2001).

Gozal and Pope (2001) tried to determine the potential long-term impact of early childhood snoring. Analysing questionnaires of 797 children in a low academic performance group (LP) and 791 in a high academic performance (HP) group, they found that frequent and loud snoring during early childhood was reported in 103 LP children (12.9%) compared with 40 HP children (5.1%). Therefore, children with lower academic performance in middle school are more likely to have snored during early childhood and to require surgery for snoring compared with better performing schoolmates. These findings suggest that children who experienced sleep-disordered breathing during a period traditionally associated with major brain growth and substantial acquisition of cognitive and intellectual capabilities may suffer from a partially irreversible compromise of their a priori potential for academic achievement. Three major components that result from the intermittent upper airway obstruction that occurs during sleep in children could theoretically contribute to such neurocognitive deficits, namely episodic hypoxia, repeated arousal leading to sleep fragmentation and sleep deprivation, and periodic or continuous alveolar hypoventilation.

Schooling problems may underlie more extensive behavioural disturbances such as restlessness, aggressive behaviour, EDS and poor neurocognitive test performances. Nearly 20–30% of children affected by OSAS or loud and frequent snoring show important signs of behavioural problems such as inattention and hyperactivity. Problems similar to symptoms of ADHD are linked to the presence of repeated sleep arousals, and intermittent hypoxic events, inducing a lack of behavioural inhibition with negative implications for working memory, motor control and self-regulation of motivation and affect.

In contrast with these data, Engle-Friedman et al. (2003) recently found a significant improvement of functions, at least in mild to moderate OSAS, when measured several months following an adenotonsillectomy, but they confirmed that their results could not rule out the possibility, even after treatment, of partial irreversible damage to academic function that may be detected only later in life. In addition, they stated that adults who also had deficits of neurocognitive executive functions related to the prefrontal area failed to improve significantly after treatment.

The negative long-term effects may be mediated by the irreversible alteration of the prefrontal cortex (PFC) and be related to structural changes of the brain as a consequence of both hypoxaemia and sleep fragmentation induced by OSAS or other pathologies affecting sleep.

In a recent report concerning OSAS adults, Macey et al. (2002) demonstrated grey matter loss in cerebral sites involved in motor regulation of the upper airway as well as in areas contributing to cognitive function (frontal and parietal cortex, temporal lobe, anterior cingulate, hippocampus and cerebellum). It can be argued that, in critical stages of brain development (that is, in childhood), these effects can lead to even more severe consequences, which could explain the negative long-term effects.

It is speculative to think that the remodelling of the brain could also be mediated by sleep and, therefore, sleep fragmentation could affect the process of brain plasticity (that is, the capacity of the brain to modify its structure and function over time). Recent studies show-

ing experience-dependent gene-expression of gene *zif-268* during paradoxical sleep in rats exposed to a rich sensorimotor environment, and the role of sleep in enhancing the remodelling of ocular dominance in the developing visual cortex are also in line with the hypothesis that sleep affects neuronal plasticity and memory processes (Peigneux et al., 2001).

2.3.1.7 Neurobehavioural manifestations

Behavioural disturbances are common in children with SRODB, with higher prevalence rates of both internalized (for instance being withdrawn, shy, anxious and psychosomatic) and externalized (for instance impulsivity, hyperactivity, aggression and delinquency) problematic behaviours (Blunden, Lushington and Kennedy, 2001). The most frequently documented problematic behaviour in children with SROBD is attention deficit hyperactivity with a prevalence rate of 20–40% (Weissbluth et al., 1983; Ali, Pitson and Stradling, 1993). Conversely, children with ADHD showed a high prevalence rate of snoring (Chervin et al., 1997) and a co-diagnosis of ADHD has been reported in 8–12% of children with OSAS (O'Brien and Gozal, 2002).

A few studies have documented that children with sleep disorders tend to have behavioural problems similar to those observed in children with ADHD. A survey of 782 children documented daytime sleepiness, hyperactivity, and aggressive behaviour in children who snored, with 27% and 38% of children at high risk for a sleep or breathing disorder displaying clinically significant levels of inattention and hyperactive behaviour, respectively (Ali, Pitson and Stradling, 1994).

At 3 years of age children with persistent sleep problems (n = 308) were more likely to have behaviour problems, especially tantrums and behaviour management problems (Zuckerman, Stevenson and Bailey, 1987).

In a study of 16 children with a mean age of 12+/-4 years suffering from chronic pain due to juvenile rheumatoid arthritis and secondary poor sleep, polysomnographic recordings showed poorer night-time sleep, longer afternoon naptime and more daytime sleepiness than normal values from the literature (Zamir et al., 1998). In a school survey of children aged 9–12 years (n = 1000), those with poor sleep (insomnia for more than 6 months) had poorer school performance, defined as failure to comply with expected grades, than good sleepers. Their learning problems were tentatively attributed to the long-term effect of poor sleep (Kahn et al., 1989).

A questionnaire administered to children aged 4–12 years (n = 472) showed a relation between sleep problems and tiredness during the day (Stein et al., 2001).

In children aged 9–12 years (n = 77), shortening sleep by one hour was associated with reduced alertness and significant lowering of neurobehavioural functioning (Sadeh, Gruber and Raviv, 2003). In school-age children (n = 140) recorded at home with an actigraph, a significant relation was shown between the presences of fragmented sleep, daytime sleepiness and lower performance in neurobehavioural functioning evaluated by various performance tests (Sadeh, Gruber and Raviv, 2000). These children also had higher rates of behavioural problems, as reported by their parents (Sadeh, Gruber and Raviv, 2002).

In Finland, children aged 7–12 years (n = 49) were interviewed together with their parents and schoolteachers and recorded for 72 hours with a belt-worn activity monitor during weekdays. The decreased amount of sleep was associated with symptoms such as aggressive and delinquent behaviour, attention, social and somatic problems. The findings of this research were better associated with the teachers' than the par-

ents' reports, suggesting that parents may be unaware of their child's sleep deficiencies as the behavioural problems may be more evident at school than at home (Aronen et al., 2000).

A prospective long-term study conducted in Sweden on 2518 children revealed that within a subgroup of 27 children with severe and chronic sleep problems, 7 children developed symptoms that met the criteria for ADHD by the age of 5.5 years (Thunström, 2002). Compared to the other children with sleep problems, these subjects had more frequent psychosocial problems in the family, bedtime struggles and long sleep latency at bedtime.

A population-based, cross-sectional questionnaire survey was conducted in Massachusetts on 30 195 children aged 5 years (Gottlieb et al., 2003). Children described by their parents as having sleep-disordered breathing (snoring, noisy breathing, apnoea) were significantly more likely to have daytime sleepiness and problem behaviours, including hyperactivity, inattention and aggressiveness (all with an odds ratio >2.0). These problem behaviours were suggestive of ADHD.

Similar findings were found in a group of children aged 5–7 years with periodic limb movement disorder who were studied polygraphically and their recording compared with those of age-matched children with ADHD. Their repeated sleep fragmentation resulting from the periodic limb movement disorder favoured the development of symptoms similar to those seen in ADHD (Crabtree et al., 2003).

The parents of a group of children with an average age of 8.6 years (range 2–17 years) reported that their children had difficult behaviours on the day that followed a 4-hour night-time sleep restriction (Wassmer et al., 1999). In one study, a 2-hour sleep reduction induced by delayed bedtime has been shown to increase daytime sleepiness, mainly during morning hours (Ishihara and Miyke, 1998; Ishihara, 1999).

Following one night of 4 hours of sleep deprivation imposed on children (aged 11–13 years), a decrease in performance tests has been observed (Carskadon, Harvey and Dement, 1981a).

Following one night's sleep loss, adolescents showed increased sleepiness, fatigue and reaction time. They selected less difficult academic tasks during a set of tests, but the percentages of correct responses were comparable to those seen following a normal night's sleep (Engle-Friedman et al., 2003).

Another study has been conducted on 82 children, aged 8–15 years. They were assigned an optimized, 10-hour night of sleep, or a restricted 4-hour night of sleep. Sleep restriction was associated with shorter daytime sleep latency, increased subjective sleepiness, and increased sleepy and inattentive behaviours, but was not associated with increased hyperactive-impulsive behaviour or impaired performance in tests of response inhibition and sustained attention (Fallone et al., 2001).

2.3.1.8 Mental health

A recent longitudinal study on the outcomes of early life sleep problems and their relation to behaviour problems in early childhood stressed the importance of studying the natural history of sleep problems and their consequences in order to identify whether persistent or recurrent sleep problems at age 3–4 years are associated with co-morbidities such as child behaviour problems, maternal depression and poor family functioning (Peiyoong, Hiscock and Wake, 2003).

The authors found that night waking at 3–4 years of age continued to be common. Seventy eight percent of mothers reported that their child awoke during the night at least once during the week, and of these waking children, 43% were reported to have awakenings 4 or more nights per week. Children with early sleep problems had significantly higher mean scores on internalizing and externalizing behaviour and the aggressive behaviour and somatic problems subscales of the Child Behavior Checklist (CBCL).

It has been noted that within groups of children and adolescents with psychiatric, behavioural or emotional problems, rates of sleep disorders are elevated (Sadeh et al., 1995). On the other hand, children and adolescents with disturbed sleep report more depression, anxiety, irritability, fearfulness, anger, tenseness, emotional instability, inattention and conduct problems, drug use and alcohol use.

Only a few longitudinal studies in adolescents have evaluated the impact of insomnia on future functioning. In a large sample of 11–17-year-old adolescents, followed for one year, using symptoms of DSM-IV criteria for insomnia, Roberts, Roberts and Chen (2002) found that nearly 18% of the youths 11–17 years of age reported non-restorative sleep almost every day in the past month, over 6% reported difficulty in initiating sleep, over 5% waking up frequently during the night, another 3% had early-morning awakening almost every day, over 7% reported daytime fatigue and 5% daytime sleepiness. Combining "often" and "almost every day" response categories dramatically increases prevalence, ranging from 60% for non-restorative sleep to 23% for daytime fatigue and 12% for waking up at night with difficulty going back to sleep. The re-evaluation of the sample at follow-up showed that insomnia predicted two indicators of psychological functioning: self-esteem and symptoms of depression (Roberts, Roberts and Chen, 2002).

2.3.1.9 Growth impairment

Failure to thrive is a well-known complication of disturbed sleep and childhood OSAS. The cause of poor growth is not known, although many different reasons have been implicated: (a) poor caloric intake associated with adenotonsillar hypertrophy; (b) excessive caloric expenditure secondary to increased work of breathing; (c) abnormal growth hormone (GH) release secondary to loss of deep non-REM sleep. The relative roles of these factors are unclear (Marcus et al., 1994; ATS, 1999). Circulating concentrations of insulin-like growth factor-I (IGF-I) and IGF-binding protein 3 (IGFBP-3) reflect mean daily GH levels, and seem to correlate well with physiological changes in GH secretion. In the operated children with initial OSAS a highly significant reduction in the apnoea-hypopnea index (AHI) was found and both the IGF-I and the IGFBP-3 concentrations increased significantly. GH is released in a pulsatile fashion; the initial secretion is synchronized with the onset of SWS and strongly correlated with slow-wave activity, within 90 to 120 minutes from the onset of sleep (Nieminen et al., 2002). In OSAS children, the sleep architecture is relatively well-preserved, but the microstructural alteration of SWS due to micro-arousals induced by respiratory disturbance could play a role in the abnormal profile of GH secretion.

2.3.1.10 Cardiovascular complications

Children with OSAS had a significantly higher diastolic blood pressure (BP) than those with primary snoring. Multiple linear regression showed that BP could be predicted by apnoea index, body mass index and age. The aetiology of OSAS-related hypertension is thought to be due to a number of factors, particularly sympathetic nervous system activation secondary to arousal and, to a lesser degree, hypoxaemia.

Although cortical arousals at the termination of obstructive apnoeas are less common in children than in adults, children may manifest signs of subcortical arousal, including autonomic changes such as tachycardia. It is therefore possible that these subcortical arousals are associated with elevations of BP. A correlation between the frequency of obstructive apnoea and BP, but no correlation between SaO2 (arterial oxygen saturation) and BP was found, suggesting that respiratory-related subcortical arousals rather than hypoxaemia may be a major determinant of BP elevation in children (Marcus, Greene and Carroll, 1998). Similarly to BP variations induced by OSAS, other studies suggest that chronic exposure to environmental noise during sleep could contribute to a permanent increases in BP in otherwise healthy individuals and that no habituation to noise was apparent over three consecutive sleep sessions (Carter et al., 2002). This is further elaborated in Chapter 4, section 4.5.

2.3.1.11 Risk of accidents

Only one study was found that evaluated the association between sleep and duration of wakefulness and childhood unintentional injury (Valent, Brusaferro and Barbone, 2001).

Two hundred and ninety-two injured children who attended the Children's Emergency Centre in Udine, Italy, or their parents were interviewed following a structured questionnaire. The sleeping time and wakefulness of the child was assessed retrospectively for each of the 48 hours before injury. For each child, the authors compared the 24 hours immediately before the injury (hours 1–24; case period) with hours 25–48 (control period).

Overall, more children had longer hours of sleep during the control period than during the case period. A direct association between injury risk and sleeping less than 10 hours was found among boys (RR: 2.33; 95% CI: 1.07–5.09) but not among girls (RR: 1.00; 95% CI: .29–3.45). The study also found a direct association between injury occurring between 16.00 and midnight, and being awake for at least 8 hours before injury occurred (both sexes, RR: 4.00; 95% CI: 1.13–14.17). Sleeping less than 10 hours a day was associated with an 86% increase in injury risk. A significantly increased risk did not emerge in all subgroups of patients but it was evident among children aged 3–5 years, boys in particular. A fourfold increase in injury risk was also associated with being awake for at least 8 hours among males only. These findings demonstrated that inadequate sleep duration and lack of daytime naps are transient exposures that may increase the risk of injury among children. Results of a study on sleep disturbance and injury risk in young children show inadequate sleep duration and lack of daytime naps. A lack of daytime naps means transient exposures that may increase the risk of injury among children. Among children (boys in particular) aged 3–5 years, sleeping less than 10 hours a day was associated with an 86% increase in injury risk. A fourfold increase in injury risk was also associated with being awake for at least 8 hours.

Daytime sleepiness in children is often manifested by externalizing behaviours noted by parents or teachers, such as increased activity levels, aggression, impulsivity, as well as by poor concentration, instantiation irritability and moodiness (Fallone, Owens and Deane, 2002).

Analysing attendance at school, data show that accidents took place at school (25.6%) and at home (22.0%), and statistics show that there is a highly significant greater total accident rate among boys than among girls. The most frequent injuries happening at school are fractures and dislocation of joints, head injuries being more common among school injuries compared with spare-time injuries. Most injuries

occurred when children were in sports areas and it is noteworthy that 25% of all injuries were caused through intentional violence by other pupils.

2.3.1.12 Use of sleeping pills

Several studies demonstrated that the use of sleeping pills is common among children and that paediatricians are prone to prescribe these medications. Twenty-five percent of firstborn infants had been given "sedatives" by 18 months (Ounsted and Hendrick, 1977). A research study into parental reports of 11 000 preschool children showed that 12% took psychoactive drugs, most commonly for sleep: 39% daily and 60% intermittently for 1–2 years (Kopferschmitt et al., 1992). Another study (Trott et al., 1995) revealed that 35% of prescriptions for children less than a year old were for sleep disturbances and that sleep disturbances were also the most common reason for prescribing medications to preschool children (23%). Two French surveys on adolescents showed that 10–12% of the respondents reported use of prescription or over-the-counter drugs for sleep disturbances (Patois, Valatz and Alperovitch, 1993; Ledoux, Choquet and Manfredi, 1994). Recently it has been reported that of 671 community-based United States paediatricians, 75% had recommended over-the-counter and 50% prescription medicines for insomnia during the past 6 months (Owens, Rosen and Mindell, 2003). In addition, an Italian survey showed that pharmacological treatment for sleep problems was prescribed during the past 6 months by 58.54% of paediatricians and by 61.21% of child neuropsychiatrists (Bruni et al., 2004).

2.3.2 BASIC INDIVIDUAL FACTORS: GENDER AND AGE

Gender shows itself to be an important predictor of disturbed sleep in virtually all epidemiological studies (Karacan et al., 1976; Bixler, Kales and Soldatos, 1979; Ancoli-Israel and Roth, 1999; Leger et al., 2000; Sateia et al., 2000). On the other hand, there does not seem to be much of a difference in polysomnographical parameters between males and females, except for the former losing SWS with increasing age and having slightly reduced sleep efficiency also with increasing age (Williams, Karacan and Hursch, 1974; Hume, Van and Watson, 1998). Ehlers and Kupfer (1997) timed the start of differences between genders to between 20 and 40 years. Spectral analysis also indicates slightly larger amounts of low frequency activity in females (Dijk, Beersma and Bloem, 1989; Dijk, Beersma and Van den Hofdakker, 1989). In addition, men seem to run a higher risk of morbidity and mortality related to sleep problems than women (Nilsson et al. 2001). The inconsistency between polysomnography and subjective measures has not been resolved but it may be important that most polysomnographical studies have controlled for anxiety and depression. Thus, it is conceivable that the higher level of subjective complaints in women reflects a higher prevalence of anxiety. The latter is a speculation, however. A confounding factor in gender comparisons is that phases in female biological cycles are also usually controlled for in polysomnographical studies, meaning that potential effects of, for example, menstruation, may not receive their proper weight. A recent review has gone through the literature in this area (Moline et al., 2003). It found that the luteal phase of the menstrual cycle is associated with subjective sleep problems, but polysomnographical studies have not supported this. Pregnancy affects sleep negatively as early as in the first trimester and the effects mainly involve awakenings and difficulties getting back to sleep. Napping is a frequent coping method. The post-partum period is often associated with severe sleep disruption, mainly due to feeding and comforting the infant. There seems to be some relation between sleep disruption and post-partum mood, but nothing is known about the

causal relations. Menopause seems to involve disrupted sleep in relation to hot flushes, depression/anxiety and sleep-disordered breathing. Oestrogen is associated with improved sleep quality but it is not clear whether the effects are due to a reduction of hot flushes. Oestrogen also improves sleep-disordered breathing.

With respect to background factors, age is an established predictor of disturbed sleep (Karacan et al., 1976; Bixler, Kales and Soldatos, 1979; Ancoli-Israel and Roth, 1999; Ribet and Derriennic, 1999; Leger et al., 2000; Sateia et al., 2000). Interestingly, however, older age may be related to a lower risk of impaired awakening (Åkerstedt et al., 2002c), that is, in this study it was easier to wake up and one felt better rested with increasing age, while at the same time sleep quality was lower. The increased risk of disturbed sleep is consistent with the increasingly strong interference of the circadian morning upswing of the metabolism with increasing age (Dijk and Duffy, 1999). Thus sleep maintenance is impaired and when sleep is interrupted "spontaneously", the awakening is, by definition, easily accomplished and will be lacking in inertia. This ease of awakening may be interpreted as "being well-rested", and obviously the need for sleep is not great enough to prevent an effortless transition into wakefulness.

In addition, sleep homeostasis seems to be weakened with age in the sense that sleep becomes more fragmented and SWS or power density in the delta bands decrease (Williams, Karacan and Hursch, 1974; Bliwise, 1993; Dijk et al., 1999). As mentioned above, the effects are more pronounced in males, a fact that may be linked to reduced levels of growth hormone and testosterone.

2.3.3 PERSONS EXPOSED TO STRESSORS AS A RISK GROUP

A number of epidemiological studies point to a strong link between stress and sleep (Åkerstedt, 1987; Urponen et al., 1988; Ancoli-Israel and Roth, 1999). In fact, stress is considered the primary cause of persistent psychophysiological insomnia (Morin, Rodrigue and Ivers, 2003). That stress can affect proper sleep seems obvious, but Vgontzas et al. (2001) at Pennsylvania State University College of Medicine have found another reason why middle-aged men may be losing sleep. It is not just because of what they worry about; rather, it is due to "increased vulnerability of sleep to stress hormones".

As men age, it appears they become more sensitive to the stimulating effects of corticotropin-releasing hormones (CRH). When both young and middle-aged men were administered CRH, the older men remained awake longer and slept less deeply. (People who don't get enough of this "slow-wave" sleep may be more prone to depression.)

The increased prevalence of insomnia in middle age may, in fact, be the result of deteriorating sleep mechanisms associated with increased sensitivity to arousal-producing stress hormones, such as CRH and cortisol. In another study, the researchers compared patients with insomnia to those without sleep disturbances. They found that "insomniacs with the highest degree of sleep disturbance secreted the highest amount of cortisol, particularly in the evening and night-time hours", suggesting that chronic insomnia is a disorder of sustained hyperarousal of the body's stress response system. Also, recent epidemiological studies have shown a connection between disturbed sleep and later occurrence of stress-related disorders such as cardiovascular diseases (Parish and Shepard, 1990; Nilsson et al., 2001;

Leineweber et al., 2003) and diabetes type II (Nilsson et al., 2002). The mechanism has not been identified but both lipid and glucose metabolisms are impaired in relation to experimentally reduced sleep (Åkerstedt and Nilsson, 2003). Burnout is another result of long-term stress and a growing health problem in many industrialized countries (Weber and Jaekel-Reinhard, 2000). In Sweden, burnout is thought to account for most of the doubling of long-term sickness absence since the mid-1990s (RFV, 2003). The characteristic clinical symptoms of the condition are excessive and persistent fatigue, emotional distress and cognitive dysfunction (Kushnir and Melamed, 1992; Melamed, Kushnir and Sharom, 1992). Self-reports of disturbed sleep are pronounced in subjects scoring high on burnout (Melamed et al., 1999; Grossi et al., 2003). Since shortened and fragmented sleep is related to daytime sleepiness and impaired cognitive performance (Bonnet, 1985, 1986a, 1986b; Dinges et al., 1997; Gillberg and Åkerstedt, 1998; Åkerstedt, 1990), disturbed sleep might provide an important link between the state of chronic stress and the complaints of fatigue and cognitive dysfunction seen in burnout.

Partinen, Eskelinen and Tuomi (1984) investigated several occupational groups and found disturbed sleep to be most common among manual workers and much less so among physicians or managing directors. Geroldi et al. (1996) found in a retrospective study of older individuals (above the age of 75) that former white-collar workers reported better sleep than blue-collar workers. Kupperman et al. (1995) reported fewer sleep problems in subjects satisfied with work.

In what seems to be the most detailed study so far, Ribet and Derriennic (1999) studied more than 21 000 subjects in France, using a sleep disturbance index and logistic regression analysis. They found that shift work, a long working week, exposure to vibrations, and "having to hurry" appeared to be the main risk factors, controlling for age and gender. Disturbed sleep was more frequent in women (Karacan et al., 1976; Bixler, Kales and Soldatos, 1979; Ancoli-Israel and Roth, 1999) and in higher age groups.

The particular stressor linked to disturbed sleep may be linked to pressure of work (Urponen et al., 1988; Ancoli-Israel and Roth, 1999; Ribet and Derriennic, 1999; Åkerstedt et al., 2002b). The demands of work are a classical work stress factor and, when combined with low decision latitude, a relation has been shown to cardiovascular diseases (Theorell et al., 1998) and absenteeism (North et al., 1996). Interestingly, when "persistent thoughts about work" was added to the regression in the study by Åkerstedt et al. (2002b) this variable took over part of the role of work demands as a predictor. This suggests that it may not be work demands per se that are important, but rather their effect on unwinding after work. In two studies it has been demonstrated that even moderate worries about being woken during the night or having a negative feeling about the next day will affect sleep negatively, mainly reducing SWS (Torsvall and Åkerstedt, 1988; Kecklund and Åkerstedt, 1997). On the other hand, there is very little data to connect real life stress with polysomnographical indicators of disturbed sleep. Most studies have used rather innocuous and artificial stressors in a laboratory environment. Field studies of stress are virtually lacking, with some exceptions (Hall et al., 2000).

A lack of social support at work is a risk indicator for disturbed sleep (Åkerstedt et al., 2002b). Few previous data of this type have been found, but poor (general) social support has been associated with sleep complaints in Vietnam veterans (Fabsitz, Sholinsky and Goldberg, 1997). On the other hand, there are several studies indicating a close connection with poor social support for, for example,

cardiovascular diseases (Arnetz et al., 1986) or muscle pain (Ahlberg-Hultén, Theorell and Sigala, 1995).

Interestingly, the metabolic changes seen after sleep curtailment in normal sleepers or in insomniacs and sleep apnoeics are similar to those seen in connection with stress. That is, lipid and glucose metabolisms are increased, as are cortisol levels (Spiegel, Leproult and van Cauter, 1999; Vgontzas et al., 2000, 2001). Together with the prospective links to stress-related diseases such as diabetes type II, to cardiovascular diseases as discussed above and with mortality (Kripke et al., 1979, 2002; Åkerstedt et al., 2002a; Dew et al., 2003), the findings could suggest that disturbed sleep may be an important mediator in the development of stress-related diseases.

2.3.4 SHIFT WORK AS A RISK FACTOR FOR SLEEP DISTURBANCE AND HEALTH EFFECTS

The dominating health problem reported by shift workers is disturbed sleep and wakefulness. At least three quarters of the shift working population is affected (Åkerstedt, 1988). When comparing individuals with a very negative attitude to shift work with those with a very positive one, the strongest discriminator seems to be the ability to obtain sufficient quality of sleep during the daytime (Axelsson et al., 2004). EEG studies of rotating shift workers and similar groups have shown that day sleep is 1–4 hours shorter than night sleep (Foret and Lantin, 1972; Foret and Benoit, 1974; Matsumoto, 1978; Tilley, Wilkinson and Drud, 1981; Torsvall et al., 1989; Mitler et al., 1997). The shorter time is due to the fact that sleep is terminated after only 4–6 hours without the individual being able to return to sleep. The sleep loss is primarily taken out of stage 2 sleep and stage REM sleep (dream sleep). Stages 3 and 4 ("deep" sleep) do not seem to be affected. Furthermore, the time taken to fall asleep (sleep latency) is usually shorter. Night sleep before a morning shift is also reduced but the termination is through artificial means and the awakening usually difficult and unpleasant (Dahlgren, 1981a; Tilley et al., 1982; Åkerstedt, Kecklund and Knutsson, 1991; Kecklund, 1996).

Interestingly, day sleep does not seem to improve much across a series of night shifts (Foret and Benoit, 1978; Dahlgren, 1981b). It appears, however, that night workers sleep slightly better (longer) than rotating workers on the night shift (Kripke, Cook and Lewis, 1971; Bryden and Holdstock, 1973; Tepas et al., 1981). The long-term effects of shift work on sleep are rather poorly understood. However, Dumont, Montplaisir and Infante-Rivard (1988) found that the amount of sleep/wake and related disturbances in present day workers were positively related to their previous experience of night work. Guilleminault et al. (1982a) found an over-representation of former shift workers with different clinical sleep/wake disturbances appearing at a sleep clinic. Recently, we have shown that in pairs of twins with different night work exposure, the exposed twin reports somewhat deteriorated sleep quality and health after retirement (Ingre and Åkerstedt, 2004).

The main reason for short daytime sleep is the influence exerted by the circadian rhythm. The more sleep is postponed from the evening towards noon next day, the more truncated it becomes and when noon is reached the trend reverts (Foret and Lantin, 1972; Åkerstedt and Gillberg, 1981). Thus, sleep during the morning hours is strongly interfered with, despite the sizeable sleep loss that, logically, should enhance the ability to maintain sleep (Czeisler et al., 1980). Also, homeostatic influences control sleep. For example, the expected 4–5 hours of daytime sleep, after a

night spent awake, will be reduced to 2 hours if a normal night's sleep precedes it and to 3.5 hours if a 2-hour nap is allowed (Åkerstedt and Gillberg, 1986). Thus, the time of sleep termination depends on the balance between the circadian and homeostatic influences. The circadian homeostatic regulation of sleep has also been demonstrated in great detail in studies of forced or spontaneous desynchronization under conditions of temporal isolation and ad lib sleeping hours (Czeisler et al., 1980; Dijk and Czeisler, 1995).

2.3.4.1 Alertness, performance and safety

Night-oriented shift workers complain as much of fatigue and sleepiness as they do about disturbed sleep (Åkerstedt, 1988). The sleepiness is particularly severe on the night shift, hardly appears at all on the afternoon shift and is intermediate on the morning shift. The maximum is reached towards the early morning (05.00–07.00). Frequently, incidents of falling asleep occur during the night shift (Prokop and Prokop, 1955; Kogi and Ohta, 1975; Coleman and Dement, 1986). At least two thirds of the respondents report that they have experienced involuntary sleep during night work.

Ambulatory EEG recordings verify that incidents of actual sleep occur during night work in, for example, process operators (Torsvall et al., 1989). Other groups, such as train drivers or truck drivers show clear signs of incidents of falling asleep while driving at night (Caille and Bassano, 1977; Torsvall and Åkerstedt, 1987; Kecklund and Åkerstedt, 1993). This occurs towards the second half of the night and appears as repeated bursts of alpha and theta EEG activity, together with closed eyes and slow undulating eye movements. As a rule the bursts are short (1–15 seconds) but frequent, and seem to reflect lapses in the effort to fend off sleep. Approximately a quarter of the subjects recorded show the EEG/EOG patterns of fighting with sleep. This is clearly a larger proportion than what is found in the subjective reports of episodes of falling asleep.

As may be expected, sleepiness on the night shift is reflected in performance. One of the classics in this area is the study by Bjerner, Holm and Swensson (1955) who showed that errors in meter readings over a period of 20 years in a gas works had a pronounced peak on the night shift. There was also a secondary peak during the afternoons. Similarly, Brown (1949) demonstrated that telephone operators connected calls considerably slower at night. Hildebrandt, Rohmert and Rutenfranz (1974) found that train drivers failed to operate their alerting safety device more often at night than during the day. Most other studies of performance have used laboratory type tests and demonstrated, for example, reduced reaction time or poorer mental arithmetic on the night shift (Tepas et al., 1981; Tilley et al., 1982). Flight simulation studies have furthermore shown that the ability to "fly" a simulator (Klein, Bruner and Holtman, 1970), or to carry out a performance test (Dawson and Reid, 1997) at night may decrease to a level corresponding to that after moderate alcohol consumption (>0.05% blood alcohol) Interestingly, Wilkinson et al. (1989) demonstrated that reaction time performance on the night shift (nurses) was better in permanent than rotating shift workers.

If sleepiness is severe enough, interaction with the environment will cease and if this coincides with a critical need for action an accident may ensue. Such potential performance lapses due to night work sleepiness were seen in several of the train drivers discussed earlier (Torsvall and Åkerstedt, 1987). The transport area is where most of the available accident data on night shift sleepiness has been obtained (Lauber and Kayten, 1988). Thus, Harris (1977) and Hamelin (1987) demonstrated that single vehicle accidents have by far the greatest probability of occurring at night.

So do fatigue-related accidents (Reyner and Horne, 1995) but also most other types of accidents, for example head-on collisions and rear-end collisions (Åkerstedt, Kecklund and Horte, 2001). The National Transportation Safety Board ranks fatigue as one of the major causes of heavy vehicle accidents (NTSB, 1995).

For conventional industrial operations very little relevant data is available but fatal work accidents show a higher risk in shift workers (Åkerstedt et al., 2002a) and accidents in the automotive industry may exhibit night shift effects (Smith, Folkard and Poole, 1994). An interesting analysis has been put forward by the Association of Professional Sleep Societies' Committee on Catastrophes, Sleep and Public Policy (Mitler et al., 1988). Their consensus report notes that the nuclear plant meltdown at Chernobyl occurred at 01.35 and was due to human error (apparently related to work scheduling). Similarly, the Three Mile Island reactor accident occurred between 04.00 and 06.00 and was due not only to the stuck valve that caused a loss of coolant water but, more importantly, to the failure to recognize this event, leading to the near meltdown of the reactor. Similar incidents, although with the ultimate stage being prevented, occurred in 1985 at the Davis Besse reactor in Ohio and at the Rancho Seco reactor in California. Finally, the committee also states that the NASA Challenger space shuttle disaster stemmed from errors in judgement made in the early morning hours by people who had had insufficient sleep (through partial night work) for days prior to the launch. Still, there is very limited support for the notion that shift work outside the transport area actually carries a higher overall accident risk.

As with sleep, the two main factors behind sleepiness and performance impairment are circadian and homeostatic factors. Their effects may be difficult to separate in field studies but are clearly discernible in laboratory sleep deprivation studies (Fröberg et al., 1975) as well as in studies of forced desynchronization (Dijk, Duffy and Czeisler, 1992). Alertness falls rapidly after awakening but gradually levels out as wakefulness is extended. The circadian influence appears as a sine-shaped superimposition upon this exponential fall in alertness. Space does not permit a discussion of the derivation of these functions, but the reader is referred to Folkard and Åkerstedt (1991) in which the "three-process model of alertness regulation" is described. This model has been turned into computer software for predicting alertness and performance and to some extent accident risk.

2.3.4.2 *Health effects*

Gastrointestinal complaints are more common among night shift workers than among day workers. A review of a number of reports covering 34 047 persons with day or shift work found that ulcers occurred in 0.3–0.7% of day workers, in 5% of people with morning and afternoon shifts, in 2.515% of persons with rotating shift systems with night shifts, and in 10–30% of ex-shift workers (Angersbach et al., 1980). Several other studies have come to similar conclusions (Thiis-Evensen, 1958; Segawa et al., 1987; Harrington, 1994). Other gastrointestinal disorders, including gastritis, duodenitis and dysfunction of the digestive system are more common in shift workers than in day workers (Koller, 1983).

The pathophysiologic mechanism underlying gastrointestinal disease in shift workers is unclear, but one possible explanation is that intestinal enzymes and intestinal mobility are not synchronized with the sleep/wake pattern. Intestinal enzymes are secreted according to the circadian rhythm, and shift workers' intake of food is irregular compared with intestinal function (Suda and Saito, 1979; Smith, Colligan and Tasto, 1982). A high nightly intake of food may be related to increased lipid levels (Lennernäs, Åkerstedt and Hambraeus, 1994) and eating at the circadian low point

may be associated with altered metabolic responses (Hampton et al., 1996). In addition, reduced sleep affects lipid and glucose metabolism (Spiegel, Leproult and van Cauter, 1999).

A number of studies have reported a higher incidence of cardiovascular disease, especially coronary heart disease, in male shift workers than in men who work days (for review see Kristensen, 1989; Boggild and Knutsson, 1999). A study of 504 paper mill workers followed for 15 years found a dose–response relationship between years of shift work and incidence of coronary heart disease in the exposure interval 1–20 years of shift work (Knutsson et al., 1986). A study of 79 000 female nurses in the United States gave similar results (Kawachi et al., 1995) as did a study with more than 1 million Danish men (Tüchsen, 1993) and a cohort of Finnish workers (Tenkanen et al.,1997). As with gastrointestinal disease, a high prevalence of smoking among shift workers might contribute to the increased risk of coronary heart disease, but smoking alone cannot explain the observed excess risk (Knutsson, 1989b). Another possibility is disturbances of metabolic parameters such as lipids and glucose for which there is some support as discussed above.

Only a few studies have addressed the issue of pregnancy outcome in shift workers. In one study of laboratory employees, shift work during pregnancy was related to a significantly increased risk of miscarriage (RR: 3.2) (Axelsson, Lutz and Rylander, 1984). Another study of hospital employees also demonstrated an increased risk of miscarriage (RR: 1.44, 95% CI: 0.83–2.51) (Axelsson and Rylander, 1989). Lower birth weight in infants of mothers who worked irregular hours has been reported (Axelsson and Rylander, 1989; Nurminen, 1989). No teratogenic risk associated with shift work was reported (Nurminen, 1989).

The mortality of shift and day workers was researched by Taylor and Pocock (1972), who studied 8603 male manual workers in England and Wales between 1956 and 1968. Day, shift, and ex-shift workers were compared with national figures. The Standardized Mortality Ratio (SMR) can be calculated from observed and expected deaths reported in the paper. SMRs for deaths from all causes were 97, 101 and 119 for day, shift, and ex-shift workers respectively. Although the figures might indicate an increasing trend, the differences were not statistically significant. However, the reported SMR close to 100 is remarkable because the reference population was the general male population. Most mortality studies concerned with occupational cohorts reveal SMRs lower than 100, implying a healthy workers' effect (Harrington, 1978). The same study showed a significantly increased incidence of neoplastic disease in shift workers (SMR 116). A Danish study of 6000 shift workers failed to demonstrate any excess mortality in shift workers (Boggild et al., 1999). Not much evidence exists on the connection between shift work and cancer. The mortality study by Taylor and Pocock (1972) reported an increased incidence of neoplasms in shift workers compared with the general population. A recent Danish case–control study reported an increased risk of breast cancer among 30–45-year-old women who worked mainly nights (Hansen, 2001). Among 75 000 nurses those with more than 15 years of night work showed an increased risk of colorectal cancer (Schernhammer et al., 2003). If the results are confirmed, a possible mechanism may be the low levels of the hormone melatonin, due to light exposure during the night with a subsequent suppression of melatonin.

Very few studies are available but Koller, Kundi and Cervinka (1978) found a prevalence of endocrine and metabolic disease of 3.5% in shift workers and 1.5% in day workers. Kawachi et al. (1995) found in a prospective study of shift work-

ers that the age-standardized prevalence was 5.6% at 15 years of shift work experience compared with 3.5% for no exposure. Nagaya et al. (2002) found that markers of insulin resistance were more frequent in shift workers above the age of 50 than in day workers. Other indicators, such as body mass index, glucose levels and so forth, give a rather inconclusive impression as indicated in a review by Boggild and Knutsson (1999).

Another contributing factor to gastrointestinal diseases might be the association between shift work and smoking. A number of studies have reported that smoking is more common among shift workers (Angersbach et al., 1980; Knutsson, Åkerstedt and Jonsson, 1988). Studies concerned with alcohol consumption comparing day workers and shift workers have produced conflicting results (Smith, Colligan and Tasto, 1982; Knutsson, 1989a; Romon, Nuttens and Fievet, 1992), probably due to local cultural habits. One study, which used g-glutamyltransferase as a marker of alcohol intake, did not indicate that the shift workers had a higher intake of alcohol than the day workers (Knutsson, 1989a).

Sickness absence is often used as a measure of occupational health risks. However, sickness leave is influenced by many irrelevant factors and cannot be considered as a reliable measure of true morbidity. Studies on sickness absence in day and shift workers have revealed conflicting results and there is no evidence that shift workers have more sickness absence than day workers (for review, see Harrington, 1978).

2.3.4.3 Conclusion

Shift work or similar arrangements of work hours clearly affects sleep and alertness and there is a moderate risk of cardiovascular and gastrointestinal disease. Other diseases such as cancer or diabetes may be related to shift work but the evidence is as yet rather weak.

The present review suggests that the risk of disturbed sleep increases with age but there also seems to be a recent stress-related increase in sleep disturbance in young adults. The long-term health consequences are not yet understood.

The relation between gender and disturbed sleep is confusing. Females, as a rule, complain more of sleep problems, but do not exhibit any objective indications of more disturbed sleep, at least not among otherwise healthy women. With increasing age the sleep of males deteriorates whereas that of women is relatively well upheld. Pregnancy, however, is a period of increased risk of disturbed sleep, whereas the menstrual cycle and menopause show less evidence of sleep disturbance. Clearly there is a great need for longitudinal research on gender and sleep and, in particular, on the possible health consequences connected with pregnancy.

Stress due to work or family seems to be one of the major causes of disturbed sleep. The link to the risk of insomnia is well-established, but reduced sleep in itself seems to yield the same physiological changes as stress. This suggests that several of the major civilization diseases in Europe and the United States (diabetes, cardiovascular diseases and burnout) could be mediated via disturbed sleep. This link clearly warrants longitudinal studies with interventions.

Shift workers constitute a group that suffers from disturbed sleep for most of their occupational life. The reason is the interference of work hours with the normal timing of sleep. This leads to an increased risk of accidents, directly due to excessive sleepiness, but also to cardiovascular and gastrointestinal diseases, although it is

not clear whether the latter effects are sleep related or due to circadian factors – or to a combination. Recent studies also suggest that breast cancer may result from shift work due to the effects of light on melatonin secretion. This still needs verification, however. Future research needs to identify countermeasures, the reasons for large individual differences in tolerance and the possible carcinogenic and other effects.

The conclusions above should be seen against the profound effects of reduced or fragmented sleep on the neuroendocrine (including glucose and lipid regulation) and immune systems as well as the effects on mortality, diabetes and cardiovascular disease.

2.3.5 CONCLUSION

Children, the elderly, pregnant women, people under stress and shift workers are vulnerable to (noise) disturbance of their sleep.

2.4 ACCIDENTS RELATED TO SLEEP QUALITY

As already stated in the earlier section on cardiovascular complications, children with disturbed sleep present cognitive dysfunction and behavioural disturbances, abnormal growth hormone release, increase of diastolic BP and an increased risk of accidents and use of sleeping pills.

Regarding sleep disturbance and accidents in adults, data show that 15–45% of all patients suffering from sleep apnoea, 12–30% of all patients suffering from narcolepsy and 2–8% of all patients suffering from insomnia have at least one accident (in a lifetime) related to sleepiness (statistics from the Stanford Sleep Disorders Clinic).

As already discussed in section 2.3.4, the biggest industrial catastrophes, such as the Three Mile Island, Bhopal, Chernobyl and Exxon Valdez disasters, have occurred during the night shift. The shift schedules, fatigue and sleepiness were cited as major contributing factors to each incident.

The LARES study (Large Analysis and Review of European housing and health Status) is one of the few studies analysing this issue directly. The results show that the likelihood of home accidents is significantly greater when the individual is tired all the time or most the time and there is an association between sleep disturbance and accidents, with 22% of those reporting an accident also reporting having their sleep disturbed during the previous four weeks.

The data available to document the impact of environmental noise on sleep deprivation and accidents are largely inadequate. There is no estimation of relative risk. Further research is needed in order to identify the accident-related burden of diseases attributable to noise during the night-time.

2.5 ANIMAL STUDIES

As pet owners know, cats sleep (most of the time it seems) and so do dogs. But do fish sleep? And flies? Yes, most animals sleep, and they even show the same phenomena as in humans; from deep sleep, dream sleep to sleep disorders. There are also many differences and weird behaviour, such as sleeping with only one half of the brain at a time (dolphins and ducks).

As Ising points out (Appendix 3), in animal experiments it is possible to assess the complete causal chain from noise exposure via physiological reactions and biological risk factors to morbidity or even mortality. However, a quantitative application of the results to humans is not possible. Instead, the method is useful in studying the pathomechanisms qualitatively. Rechtschaffen and Bergmann (1965) studied sleep deprivation in rats, showing that total sleep deprivation leads to mortality in 16 to 20 days. As the animals in the last stage died from microbial infection, Everson and Toth (2000) proceeded to show early infection of the lymph nodes and other tissues and hypothesized that daily sleep of some amount is necessary to maintain an intact immune system that will prevent bacterial invasion, a view that has been challenged.

Surprisingly, sleep in the common fruit fly – *Drosophila melanogaster* – has many similarities with mammalian sleep, including sleep deprivation leading to impaired performance. Genetic studies in fruit flies (Cirelli et al., 2005) led to mutant flies that can get by on 30% less sleep than their normal counterparts, thanks to a single mutation in one gene. While they sleep 30% less they show no immediate ill effects. The lifespan of the flies is, however, reduced by 30%.

These animal models certainly lead one to believe that sleep is a biological necessity, and tampering with it is dangerous for survival.

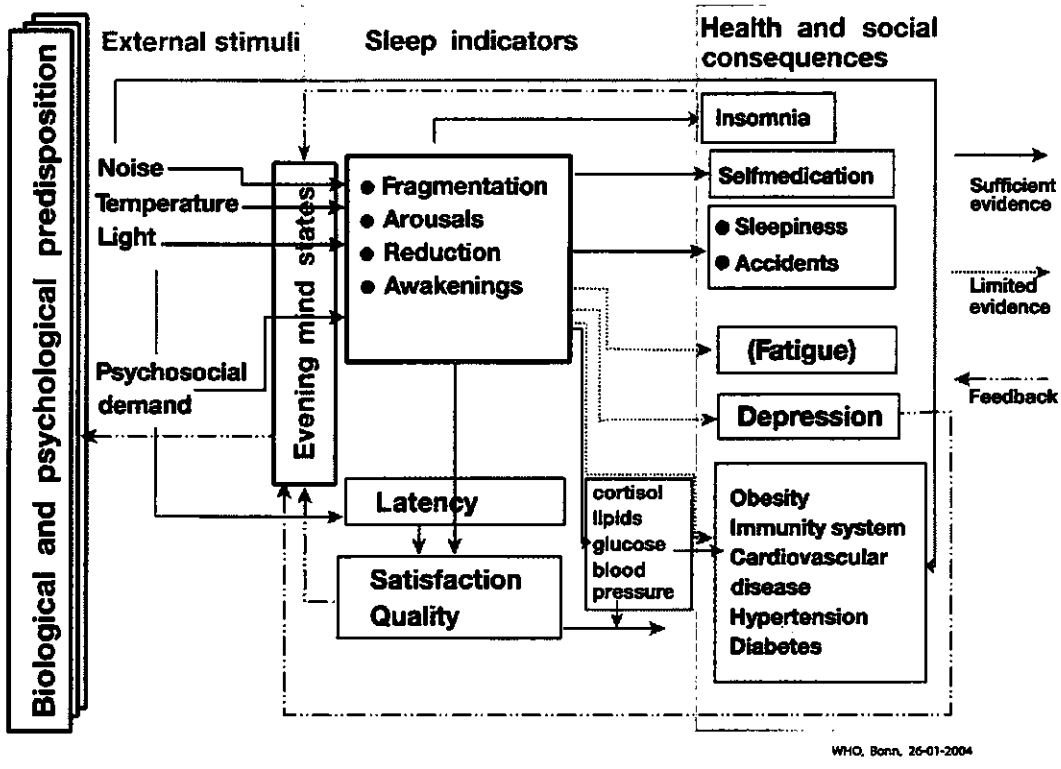
As Ising shows (Appendix 3) noise may play a role in this. Under stressful circumstances the death rate of rats is increased when noise levels are increased from "ambient" to L_{eq}=69 dB(A). Are noise and sleep deprivation stressors that both lead to early death? Is the noise effect due to sleep deprivation? A carefully planned study may sort this out. The question still remains, however, as to how far this is relevant to humans.

2.6 CONCLUSIONS

From the evidence presented so far it can be deduced that sleep is important for human functioning. Why exactly is less evident, but it is clear that disturbed sleep (either from internal factors or from external factors) leads to or is at least associated with fatigue, lower cognitive performance, depression, viral illness, accidents, diabetes, obesity and cardiovascular diseases. Animal experiments show that sleep deprivation shortens lifespan. The fact that – in comparison– relatively mild effects turn up in human sleep deprivation experiments could be due to the short period (about 10 days in controlled experiments) and the limitation to young and healthy adults. The central position of sleep in human functioning is summarized in Fig. 2.1. In this figure relations with sufficient evidence are indicated with solid lines, while relations for which limited evidence exists are indicated with interrupted lines. Feedback connections are in red and double-dotted.

The presence of feedback loops in the system is an indication that it may be difficult to prove direct cause–effect relations. One example is the relation between sleep quality and depression. They are strongly associated, but it is uncertain if depression causes bad sleep, or bad sleep causes depression (see also Chapter 4, section 4.8.11). This may also depend on one of the many other factors, so it could be different for different personality types.

Fig.2.1
Expert view on the relations between sleep and health



Impaired sleep is widely considered as a health problem per se, and this chapter has shown that there are many internal and external causes. In the next chapter the relation between noise and sleep quality is further unravelled.

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CHAPTER 3

EFFECTS OF NIGHT-TIME NOISE ON SLEEP

Best travel tip: Never ever forget to pack ear plugs.
(Virginia Jealous, Lonely Planet author)

3.1 SHORT-TERM EFFECTS OF TRANSPORTATION NOISE ON SLEEP WITH SPECIFIC ATTENTION TO MECHANISMS AND POSSIBLE HEALTH IMPACT

3.1.1 INTRODUCTION

In this section reactions to single events are presented. In Chapter 2 normal sleep and sleep disorders are described in medical terms, but here the focus is on the mechanisms underlying the relation between noise and sleep quality. How does a sound penetrate the brain and cause a disruption of sleep?

3.1.2 HOW NOISE INFLUENCES NORMAL SLEEP

Noise can induce changes in the EEG or in autonomic variables that are called arousals or phasic activations. Similar brief episodes of activity also occur without noise in normal sleep, and, more frequently, in sleep that is otherwise disturbed, for example by apnoea. Arousal during sleep is not a uniform concept and has been defined differently by different researchers. Commonly, the occurrence of alpha rhythms is required for EEG arousal. Depending on the additional requirements and on the length of time that the slower cortical rhythms are interrupted, arousals have been called, for instance, micro-arousal, minor arousal, EEG awakening or transient activation phases. EEG awakening requires an interruption of the sleep patterns of at least 15 seconds (half the period) when sleep staging is scored by periods of 30 seconds, but need not be experienced consciously. Because normal REM sleep is a state characterized by cerebral arousal with frequently occurring alpha rhythms, additional criteria are needed to define arousal from REM. The criteria used are increased heart rate, EMG, or irregular respiration. However, since the mechanisms of such autonomic responses appear to be at least partly different from the causal mechanisms of EEG arousal, such definitions seem to make arousal from sleep a heterogeneous concept that may not have simple relationships with noise exposure.

EEG arousals lasting at least 30 seconds have been found to occur as often as 4 times (95% CI: 1–15) per hour during sleep on average, while micro-arousals occurred 21 times (95% CI: 7–56) per hour (Mathur and Douglas, 1995). Since these figures are from a laboratory study, they almost certainly are higher than the figures that hold for the natural situation at home. Sleep pressure decreases the density of micro-arousals (Sforza, Jouny and Ibanez, 2000). While the number of EEG arousals (dur-

ing sleep stages 1 and 2) increases with age (Mathur and Douglas, 1995; Boselli et al., 1998) possibly only for men (Hume, Van and Watson, 2003), their average length is stable and circa 15 seconds (Boselli et al., 1998). Also the threshold for auditory arousal decreases with age (Zepelin, McDonald and Zammit, 1984; Busby and Pivik, 1985; Busby, Mercier and Pivik, 1994) and towards the end of the night (Basner et al., 2004). Recovery after EEG awakenings takes longer for noise-induced awakenings than for spontaneous awakenings (Basner et al., 2004). The time required for falling asleep again depends on the sound level and especially for loud events this latency is considerably longer than after spontaneous awakenings. Thus, in general, noise-induced EEG awakenings are more disruptive than spontaneous awakenings and therefore will more often be experienced consciously and remembered afterwards. In common situations with aircraft overflight noise at home, (minor) arousals were found in 10.3% of the 64-second intervals without aircraft noise and this percentage was found to be increased by circa 4% up to 14.3% in intervals with an aircraft noise event (Hume, Van and Watson, 2003). Thus, in that particular (exposure) situation, about 1 in 24 aircraft overflights caused a (minor) arousal.

3.1.3 MECHANISMS

Activity in the auditory system up to the brainstem nucleus inferior colliculus occurs within 10 milliseconds after the onset of a sound. This early activity appears to be obligatory and is hardly affected by the state (sleeping or awake). Being asleep or awake does influence later activity. The auditory pathways proceed from the inferior colliculus to the thalamus and from there to the auditory cortex. The state (asleep or awake) affects the activity in the thalamocortical circuits, which occurs after 10–80 milliseconds. In particular, during SWS the transmission of auditory information through the thalamus is suppressed. This is not the case during REM sleep or when awake.

Thus, while in all (sleep) stages, sound activates the auditory system up to the inferior colliculus, the sound-induced activation of higher areas is suppressed in SWS. Therefore, further activation depending on those higher areas (for example, extracting meaning) is not likely to occur as a primary reaction to sound during SWS. For understanding arousal during SWS, it is important that the inferior colliculus and the earlier auditory nucleus of the lateral lemniscus, and also the (dorsal and ventral) cochlear nuclei project to reticular arousal system. Presumably, sound is always capable of arousing the sleeping subject through these connections. The ascending arousal system is heterogeneous and encompasses mono-aminergic, glutamatergic, and cholinergic nuclei that can directly or indirectly activate the thalamus and cortex. An important indirect route is the activation of the basal forebrain, which can activate the cerebral cortex through widespread, mainly cholinergic projections. The activation of the thalamus and cortex is indicated by an increase in EEG rhythm frequency and a reduction of the inhibition in the thalamic sensory relay nuclei. As a consequence of the latter effect, subsequent sound-induced activation may pass the thalamus and may be subject to more elaborate processing than initial sound. It can be speculated that sound in this way also reduces the threshold for somatosensory information that initiates body movements so that more body movements are observed when exposed to sound. The occurrence of habituation of cortical responses suggests an active role played by a part of the brain that blocks or at least limits the impact of the activated ascending pathways.

The parasympathetic autonomic nervous system seems to be responsible for the bradycardia observed in non-REM sleep and mainly in tonic REM sleep through the increase in vagal activity (Guazzi et al., 1968). The variability of heart rate in REM sleep could be placed under the same control, as vagotomy strongly reduced the heart rate instability (Baust and Bohnert, 1969). During falling asleep, respiration is unstable and alternates between hypo- and hyperventilation episodes. This respiration, called "periodic respiration" (Mosso, 1886), disappears when stable sleep occurs (stage 2). The main hypotheses concerning this periodic ventilation refer to metabolic control and chemoreceptor responses to levels of $PaCO_2$ and PaO_2 (Chapman et al., 1988). In stable non-REM sleep, respiration is regular in amplitude and frequency, although ventilation per minute is lower than during awakening. In REM sleep, respiration appears irregular with sudden variations in amplitude and frequency. This irregularity appears to be not modifiable by metabolic factors and, therefore, it is possibly linked to mechanisms leading to REM expression. The non-habituation of the cardiovascular responses would be explained by the absence of an inhibitory influence on the part of the arousal system that affects the centres regulating the autonomous response.

3.1.4 EEG RESPONSE

The sleep polygraph continuously records EEG activity, eye movement (EOG) and muscle tone (EMG). These data are used to classify sleep into various stages, and to assess times of falling asleep and waking up. Also, sleep variables such as total sleep time and total time spent in SWS (consisting of sleep stages 3 and 4, the stages of deep sleep) and in the REM stage (also called dream or paradoxical sleep) can be assessed on the basis of sleep polygraph recordings. Polygraphic indicators of responses to individual noise events are changes from a deeper to a less deep sleep and EEG awakening. Several field studies (Pearsons, Bennett and Fidell, 1973; Vernet, 1979; Vallet et al., 1983; Hume, Van and Watson, 2003; Basner et al., 2004) have been conducted regarding noise-induced changes in sleep stage and awakening using EEG recordings. Transition from a deep stage of sleep to a shallower sleep stage can be the direct consequence of a nocturnal noise event. Although not perceived by the sleeper, these transitions modify the sleep architecture and may reduce the amount of SWS (Carter, 1996; Basner et al., 2004) and the amount and rhythmicity of REM sleep may be markedly affected (Naitoh, Muzet and Lienhard, 1975; Thiessen, 1988). In addition to their results from a laboratory study, Basner et al. (2004) present results from a field study with valid data for 63 subjects (aged 18–65 years) with 15 556 aircraft noise events included in the final analyses. They established a curve that gives the probability of awakening as a function of L_{Amax} with a model that assumed a background noise level just prior to the aircraft noise event of 27 dB(A). The L_{Amax} threshold for noise-induced awakenings was found to be about 35 dB(A). Above this threshold the probability of noise-induced awakenings increases monotonically up to circa 10% when L_{Amax} = 73 dB(A). This is the extra probability of awakening associated with the aircraft noise event, on top of the probability of awakening spontaneously in a 90 second interval.

Some arousals provoked by noise events are so intense that they induce awakening. Frequent awakening leads to sleep fragmentation and overall sleep disturbance. The noise threshold for awakening is particularly high in deep SWS (stages 3 and 4) while it is much lower in shallower sleep stages (stages 1 and 2). In REM sleep the awakening threshold is variable and depends on the significance of the stimulus. Total

sleep time can be reduced by both a longer time to fall asleep and premature final awakening. It has been reported that intermittent noises with maximum noise levels of 45 dB(A) and above can increase the time taken to fall asleep by a few to 20 minutes (Öhrström, 1993). In the morning hours, the sleeper can be more easily awakened by ambient noise and has more difficulty going back to sleep because sleep pressure is progressively reduced with time (Rechtschaffen, Hauri and Zeitlin, 1966; Keefe, Johnson and Hunter, 1971).

Terzano et al. (1990, 1993) showed that with increasing intensity of sound pressure level (white noise at 45, 55, 65 and 75 dB(A), white noise induced a remarkable enhancement of cyclic alternating patterns (CAP)/non-REM, characterized by a linear trend from the lowest to the highest intensities, revealed by a significant increase in the CAP rate already at 45 dB(A). Noise decreased mainly SWS, REM and total sleep time, and increased waking after sleep onset, stage 1 non-REM and CAP rate (Terzano et al., 1993). For CAP/non-REM values between 45% and 60%, subjects generally recalled a moderate nocturnal discomfort and values of CAP/non-REM over 60% corresponded to a severe complaint.

This result corroborates previous findings described by Lukas (1972a) who reported that reactions less intense than a sleep stage change correlate better to the noise intensity than awakening reactions.

3.1.5 CARDIOVASCULAR RESPONSE

For sleeping persons, mean heart rate, mean systolic and diastolic BP and variability in heart rate are usually assessed. Indicators of responses to individual noise events are instantaneous changes in (variability of) heart rate and changes in systolic BP. Several field studies (Carter et al., 1994) have been conducted regarding momentary change in heart rate. Intermittent noise during sleep has been found to induce a biphasic cardiac response and a transient constriction of peripheral vessels together with a short phasic activation in the EEG, while no other behavioural effect can be seen (Muzet and Ehrhart, 1978). This biphasic cardiac response starts with an increase in heart rate, probably due to a phasic inhibition of the parasympathetic cardio-inhibitory centre, followed by a compensatory decrease due to a phasic decrease in orthosympathetic activity (Keefe, Johnson and Hunter, 1971; Muzet and Ehrhart, 1980). The vasoconstrictive response was reported to be due to the sympathetic peripheral stimulation provoked by the auditory reflex (Kryter and Poza, 1980). More recently, Carter et al. (2002) have shown that beat-by-beat BP changes can be induced by suddenly occurring noises. Although habituation in some effect parameters can occur in a few days or weeks, this habituation is not complete and the measured modifications of the cardiovascular functions remain unchanged over long periods of exposure time (Muzet and Ehrhart, 1980; Vallet et al., 1983). Most striking is that none of the cardiovascular responses show habituation to noise after a prolonged exposure, while subjective habituation occurs within a few days. In people that are used to sleep in a noisy surrounding, noise-induced changes in heart rate are dependent on the maximum sound level of a noise event, but not on the EEG sleep stage).

3.1.6 BODY MOVEMENT

Motility is the term used for accelerations of the body or body parts during movement. It is measured with actimeters, usually worn on the wrist in field research and

the laboratory. Brink, Müller and Schierz (2006) describe a more sophisticated method which is based on the bed being placed on accelerometers. This allows the tracking of whole body movements.

Motility is related to many variables of sleep and health (Reyner, 1995; Reyner et al., 1995; Passchier-Vermeer et al., 2002). Clinical research shows that the sleep/wake cycle (assessed by polysomnography, EEG, EOG, EMG) passes through the 24-hour period synchronously with the rest/activity cycle (assessed by actimetry) (Borbely et al., 1981). A number of investigations have compared the results of polysomnographic recordings (number of EEG-awakenings during sleep period, duration of sleep period, sleep onset time, wake-up time) with results of actimetry. The correlation between actimetrically assessed duration of sleep period, sleep onset time, wake-up time and similar variables assessed with polysomnography was found to be very high (correlation coefficients between individual test results in the order of 0.8–0.9). Measures of instantaneous motility are the probability of motility and the probability of onset of motility in a fixed time interval, for example a 15-, 30- or 60-second interval. Increased instantaneous motility during sleep is considered to be a sensitive behavioural marker of arousal, but the relation with arousal is not simple. Also other factors, such as the need to relieve the pressure on body parts for better blood circulation, cause motility, and spontaneously occurring arousals are part of the normal sleep process. The noise-induced probability of (onset of) motility is the difference between the probability of (onset of) motility during noise events minus the probability in the absence of noise.

Onset of motility and minor arousal found on the basis of EEG recordings are highly correlated. In the United Kingdom sleep disturbance study, Ollerhead et al. (1992) found for their study population that during sleep there is on average an EEG (minor) arousal in 40% of the 30-second intervals with onset of motility. Unfortunately, it is unknown whether this 40% is also valid for noise-induced awakenings. In 12% of the 30-second intervals with an EEG (minor) arousal, motility does not occur. Several field studies (Horne et al., 1994; Fidell et al., 1998, 2000; Flindell, Bullmore and Robertson, 2000; Griefahn et al., 2000; Passchier-Vermeer et al., 2002, 2004) have been conducted regarding noise-induced instantaneous motility. For this effect, relationships have been established with SEL or L_{Amax}, for aircraft noise only. In Passchier-Vermeer et al. (2002) relationships between noise-induced increase in motility or noise-induced increase in onset of motility in the 15-second interval with the maximum noise level of an overflight, and L_{Amax} or SEL have been approximated by quadratic functions (see, for instance, Fig. 3.2). It may be noted that the threshold of motility (L_{Amax} = 32 dB(A)) is in the same range as the threshold found by Basner et al. (2004) for EEG awakenings, with a definition that also encompassed transitions to steep stage 1 (L_{Amax} = 35 dB(A)). The probability of motility at 70 dB(A) of about 0.07 is lower than the probability of noise-induced EEG awakening at L_{Amax} = 73 dB(A) of about 0.10. There is no a priori reason to expect the above threshold probabilities to be the same for these two measures of sleep disturbance, but, taking into account that motility is assessed for shorter intervals (15 seconds vs. 90 seconds), the differences in probabilities above threshold appear to be limited.

One of the variables influencing the relationships between noise-induced instantaneous motility and L_{Amax} or SEL, is long-term aircraft noise exposure during sleep. The probability of instantaneous aircraft noise-induced motility is lower when the long-term exposure is higher. This may be partly due to the higher base rate motility in quiet intervals in higher long-term exposure, which is used as a reference for

the instantaneous noise-induced motility. Other factors influencing the relationships between instantaneous motility and L_{Amax} or SEL are the point of time in the night, and time since sleep onset. For example, after 7 hours of sleep, noise-induced motility is about 1.3 larger than in the first hour of sleep. Age has only a slight effect on noise-induced motility, with younger and older people showing a lower motility response than persons in the age range of 40–50 years.

3.1.7 BEHAVIOURAL AWAKENING IN ADULTS

Passchier-Vermeer (2003a) published a review of nine studies on awakening by noise. It was found that these studies had different definitions of what constituted an "awakening". In this review, however, all awakening data were collected on "behavioural awakening": these are awakenings that were followed by an action (like pressing a button) from the sleeper. The number of awakenings defined in this manner is much smaller than the number of sleep stage changes which lead to EEG patterns similar to wakefulness.

Data were available for rail traffic noise, ambient (probably road) noise, civil aviation noise and military aviation noise.

The rail traffic noise study is very small (only 20 subject nights), but showed no awakenings. The study states that "there is some evidence, be it very limited, that railway noise events, in the range of SEL_{indoor} considered (up to 80 dB(A)), do not increase [the] probability of awakening".

Ambient noise also showed no effect on the probability of awakening, but as it is uncertain exactly what noise is meant, no firm conclusions could be drawn.

Military aircraft noise showed a very strong effect, but this study is of limited applicability since the few subjects (military) lived near the end of the runway.

For civil aviation noise there were sufficient data to derive a dose-effect relation:

$$\text{Percentage of noise-induced awakenings} = -0.564 + 1.909 * 10^{-4} * (SEL_{inside})^2 \quad [4],$$

where SELinside is the sound exposure level of an aircraft noise event in the bedroom.

This relation is confined to commercial aircraft noise over the intervals $54<SEL<90$ ($37<L_{Amax}<82$) and the number of events per night $1< N< 10$.

With this relation, it is possible to calculate for an individual L_{night} the expected number of noise-induced behavioural awakenings. This requires all single contributions over the year to this L_{night} to be known. Alternatively (if, for instance, a future situation has to be estimated for which no exact data are available) a worst case scenario can be calculated. Fig. 3.1 represents the results of this worst case approach (converted to L_{night}, see Chapter 1, section 1.3.4), and so gives the maximum number of awakenings n_{max} that may be expected.

$$n_{max} = 0.3504 * 10^{(L_{night}-35.2)/10} \quad [5].$$

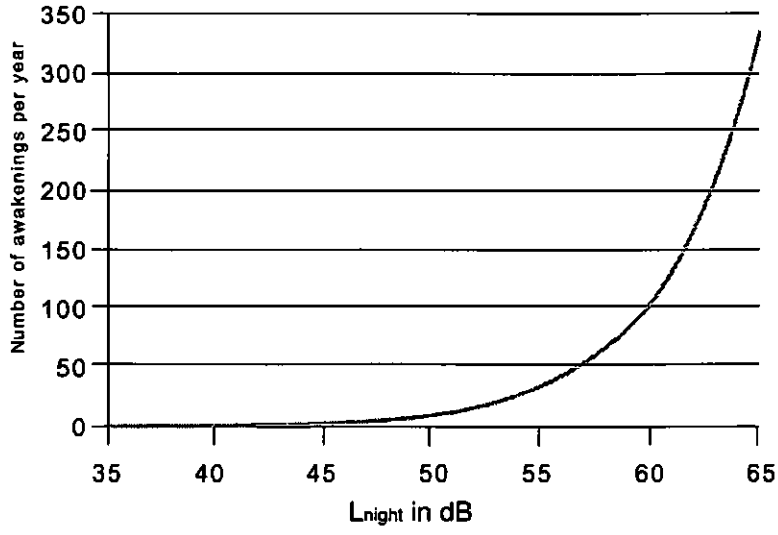


Fig. 3.1
Worst case prediction of noise-induced behavioural awakenings.
L_{night}, converted from inside relation with [3] on page 10.

Source: Miedema, Passchier-Vermeer and Vos, 2003

It can be demonstrated that the number of awakenings reaches a maximum when the SEL_{inside} value is 58.8 dB(A).

It should be noted that, on average, 600 spontaneous awakenings per person are reported per year. This also explains why so many more awakenings are reported than can be attributed directly to aircraft noise. At 55 L_{night}, nearly 100 overflights per night with SEL_{inside} = 58.8, or 1 every 5 minutes are possible. It is, therefore, very likely that an overflight coincides with a spontaneous awakening.

3.1.8 DOSE–EFFECT RELATIONS FOR BODY MOVEMENTS DURING SLEEP

In Passchier-Vermeer et al. (2002) motility is registered in 15-second intervals. A distinction is made between two variables:

- the *presence of motility* in the interval (indicated by m) and
- the onset *of motility*, meaning the presence of motility when there was no motility in the preceding interval (indicated by k).

Relations between a noise-induced increase in motility (m) or a noise-induced increase in the onset of motility (k) in the 15-second interval with the maximum sound level of an overflight, and $L_{Amax,inside}$ or SEL_{inside} have been approximated by quadratic functions with the following format:

$$m = b^*(L_{Amax,inside} - a) + c^*(L_{Amax,inside} - a)^2 \quad [6].$$

The coefficients a, b and c are given in Table 3.1. The value of a is the value below which m or k is zero. Fig. 3.2 shows the relationship between m and $L_{Amax,inside}$ together with the 95% confidence interval. Relations apply to $L_{Amax,inside}$ and SEL_{inside} values of at most 70 and 80 dB(A), respectively.

Table 3.1.
Coefficients of the quadratic equation (formula [6]) of m and k as a function of $L_{Amax,inside}$ or SEL_{inside} for the 15-second interval in which an indoor maximum sound level of an aircraft noise event occurs. The equations are applicable in the $L_{Amax,inside}$ range from a up to 70 dB(A), or SEL_{inside} range from a up to 80 dB(A). Below a, m and k are zero.

	(Aircraft) noise-induced increase of probability of motility (m)	(Aircraft) noise-induced increase of probability of onset of motility (k)
range	$32 < L_{Amax,inside} < 70$ dB(A) (see Fig. 3.2)	$32 < L_{Amax,inside} < 70$ dB(A)
a	32	32
b	0.000633	0.000415
c	3.14×10^{-5}	8.84×10^{-6}
range	$38 < SEL_{inside} < 80$ dB(A)	$40 < SEL_{inside} < 80$ dB(A)
a	38	40
b	0,000532	0.000273
c	2.68×10^{-5}	3.57×10^{-6}

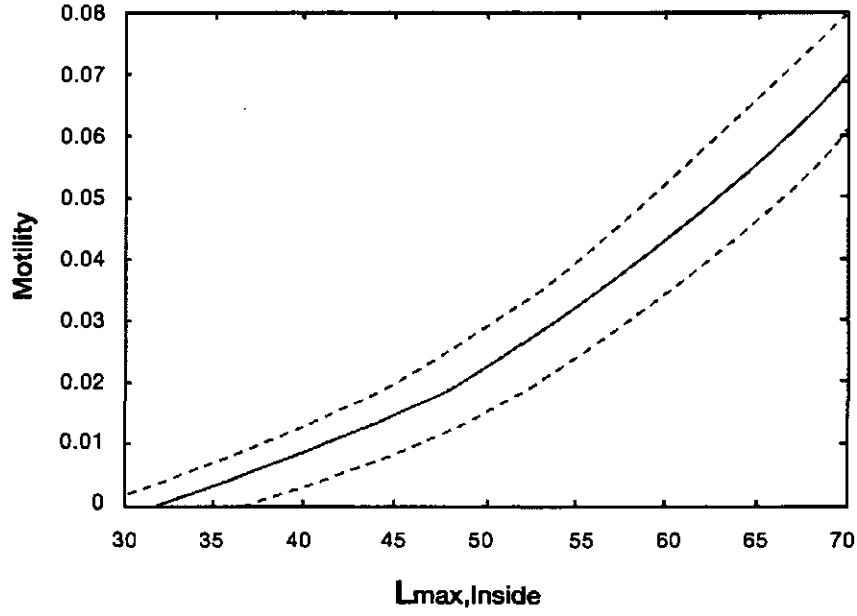


Fig. 3.2
Probability of (aircraft) noise-induced motility (m) at the 15-second interval in which the indoor maximum sound level occurs (solid line) and the 95% confidence interval, as a function of $L_{Amax,inside}$ bedroom

Source: Miedema, Passchier-Vermeer and Vos, 2003.

The study report also gives the upper boundaries for motility, based on the relationship between L_{Amax}, SEL and L_{night} (Fig. 3.3). This figure is mathematically derived from relation [6] as described in Appendix 2.

This area of study is still under development. Miedema, Passchier-Vermeer and Vos (2003) give a detailed account in their study report of the relation between the study used for the data presented here (Passchier-Vermeer et al., 2002), earlier studies like the much quoted Civil Aviation Authority study (Ollerhead et al., 1992; Ollerhead, 1994) and earlier work done in the United States.

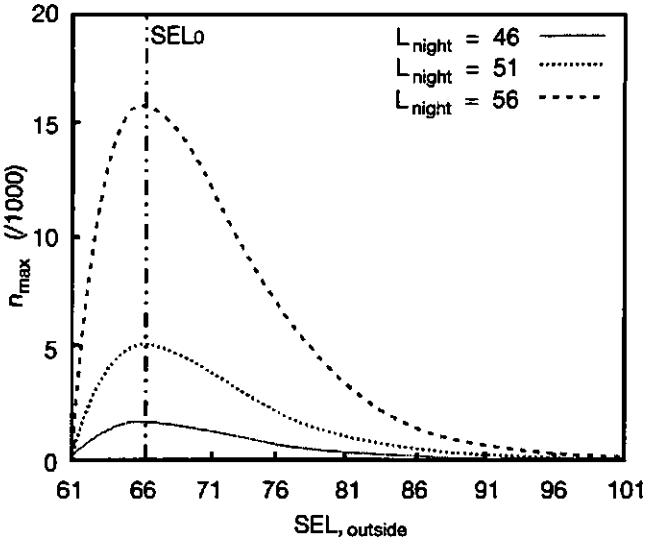


Fig. 3.3
Maximum number of noise-induced motility for three values of L_{night}. Converted from inside relation with [3] on page 10

Source: Miedema, Passchier-Vermeer and Vos, 2003.

3.1.9 INDIVIDUAL SENSITIVITY

Sensitivity to noise may vary greatly from one individual to another. Primary self-evaluation of sensitivity to noise has been used as a factor to evaluate highly sensitive and non-sensitive groups and to compare their reactions to noise exposure during daytime and night-time (Di Nisi et al., 1990). In this study, self-declared highly sensitive individuals had a higher cardiovascular response rate to noise than non-sensitive people during their waking exposure, while there was no difference in sensitivity to noise between these two groups during their night-time exposure while they were asleep.

The physiological sensitivity to noise depends also on the age of the sleeper. Thus, while EEG modifications and awakening thresholds are, on average, 10 dB(A) higher in children than in adults, their cardiovascular sensitivity to noise is similar, if not higher, than the older group (Muzet and Ehrhart, 1980; see also Appendix 4). Elderly people complain much more than younger adults about environmental noise. However, their spontaneous awakenings occurring during night sleep are also much more numerous. Therefore, it is difficult to conclude if elderly people are more sensitive to noise or if they hear noise because they are often awake during the night. This natural fragmentation of their night sleep tends also to lengthen their return to the sleeping state and this accounts for a significant part of their subjective complaints.

Differences in sensitivity to noise have been found between the sexes. Thus, young men seem to complain more about noise-disturbed sleep than young females (Muzet et al., 1973). However, this difference seems to reverse for populations over 30 years of age and then females (often mothers) appear to be more sensitive to noise than males (Lukas, 1972b).

3.1.10 USE OF INSTANTANEOUS EFFECTS IN PREDICTIONS OVER A LONGER TIME PERIOD

It is tempting to use the relations between single exposures and measured effects in long-term predictions. Although this is perhaps possible, a word of caution is appropriate.

In general, the reactions are calculated by looking at a certain time frame around an exposure, usually in the order of a few minutes. The second limitation is that order and follow-up effects are neglected. Time and order effects of identical events on

motility have been described by Brink, Wirth and Schierz (2006). Only if the situation that is modelled resembles the one that was used in the single exposure analysis, are no major deviations to be expected. Reactions to noise events are generally not independent from each other. Each event may alter a subject's tendency to awake at the next event, even if no awakening reaction is detected for that particular event. If, for example, each event would additionally increase the probability of awakening at the next event, the total probability of awakening per night would be greater than predicted by mere summation of the single event probabilities. Most likely, this underestimation of probability will occur when events in the real situation follow in close succession, whereas events in the single exposure analysis did not. Such limitations can to some degree be overcome through applying advanced statistical methods such as those put forward by Basner (2006). A third limitation is that an overall increase in the base line could go undetected.

If the situation that is calculated resembles the one that was used in the single exposure analysis, probably no major deviations are to be expected. Care should be taken to extrapolate outside the boundaries given in the number of events or L_{Amax}. Calculations for Amsterdam Schiphol Airport show a good agreement between the number of calculated awakenings per year (based on the actual SEL data) and the self-reported number of awakenings. This number is a factor 2 lower than the worst case scenario presented in section 3.1.7 above.

3.2 CHRONIC EFFECTS: CHRONIC INCREASE OF MOTILITY

Mean motility – all body movements counted together – during sleep is strongly related to age and is also a function of noise exposure during the sleep period. The relationships between mean motility and $L_{night,inside}$ are shown in Fig. 3.4. Mean motility during sleep is lowest at the age of 45 years, and greater at higher and lower ages. The relation between mean motility, $L_{night,inside}$ and age is:

$$\text{Mean motility} = 0.0587 + 0.000192 * L_{night,inside} - 0.00133 * age + 0.0000148 * age^2 \quad [7].$$

The relation between the increase in mean noise-induced motility, m_{night}, and $L_{night,inside}$ is:

$$m_{night} = 0.000192 * L_{night,inside} \quad [8],$$

assuming, as described in Chapter 1, section 1.3.4, that $L_{night,inside} = L_{night}-21$:

$$m_{night} = 0.000192 * L_{night} - 0.004032 \quad [8a].$$

Source: Miedema, Passchier-Vermeer and Vos, 2003.

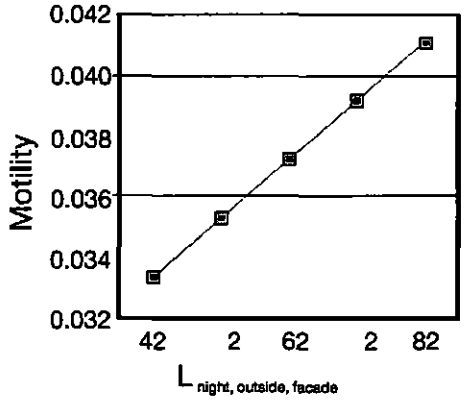


Fig. 3.4
Increase in mean motility (body movements during sleep). Converted from inside relation with [3]

The increase in m_{night} is 22% over the baseline motility (0.03 on average) if indoor $L_{night,inside}$ increases from 0 (absence of aircraft noise) to 35 dB(A) (living close to a runway). This increase is independent of age, although the absolute level varies.

Other chronic effects like the use of sleeping pills, changes in BP and changes in levels of stress hormones are discussed in the next chapter.

3.3 CONCLUSIONS

During sleep the auditory system remains fully functional. Incoming sounds are processed and evaluated and although physiological changes continue to take place, sleep itself is protected because awakening is a relatively rare occurrence. Adaptation to a new noise or to a new sleeping environment (for instance in a sleep laboratory) is rapid, demonstrating this active protection. The physiological reactions do not adapt, as is shown by the heart rate reaction and the increase of average motility with sound level. The autonomous physiological reactions are a normal reaction to these stimuli, but the question is whether prolonged "abuse" of this system leads to adverse consequences for the organism. The next chapter tries to answer that.

CHAPTER 4

EFFECTS OF NIGHT-TIME NOISE ON HEALTH AND WELL-BEING

The sick die here because they can't sleep,

For when does sleep come in rented rooms?
It costs a lot merely to sleep in this city!
That's why everyone's sick: carts clattering
Through the winding streets, curses hurled
At some herd standing still in the middle of the road,
Could rob Claudius or a seal of their sleep!

(Juvenal, 1st century AD)

4.1 INTRODUCTION

In Chapters 2 and 3, sufficient evidence was presented to support the hypothesis for the simplified model presented in Chapter 1: sleep disturbance is connected to health impairment, and noise is an important factor that causes sleep disturbance. The full model (Fig. 2.1, Chapter 2) showed why it is difficult to find evidence for a direct relation between noise exposure at night and health outcomes. Noise is but one of the internal and external factors that cause sleep disturbance and feedback loops obscure the view of the cause and effect chain. In this chapter the evidence for the direct relation is presented.

4.2 SELF-REPORTED (CHRONIC) SLEEP DISTURBANCES

Self-reported sleep disturbance is investigated by means of a questionnaire containing questions regarding sleep disturbance. Often, sleep disturbance is not the main focus of the questionnaires used in studies of self-reported noise effects. This means that considerable effort is needed to harmonize the different response categories. The relationships for self-reported sleep disturbance are based on analyses of the 15 data sets with more than 12 000 individual observations of exposure–response combinations, from 12 field studies (Miedema, 2003; Miedema, Passchier-Vermeer and Vos, 2003).

The curves are based on data in the L_{night} (outside, most exposed facade) range 45–65 dB(A). 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)). The formulae of these polynomial approximations are as follows (SD = sleep disturbance; H = high; L = low):

for road traffic:

$$\%HSD = 20.8 - 1.05*L_{night} + 0.01486*(L_{night})^2 \quad [9]$$

$$\%SD = 13.8 - 0.85*L_{night} + 0.01670*(L_{night})^2 \quad [10]$$

$$\%LSD = -8.4 + 0.16*L_{night} + 0.01081*(L_{night})^2 \quad [11]$$

for aircraft:

$$\%HSD = 18.147 - 0.956*L_{night} + 0.01482*(L_{night})^2 \quad [12]$$

$$\%SD = 13.714 - 0.807*L_{night} + 0.01555*(L_{night})^2 \quad [13]$$

$$\%LSD = 4.465 - 0.411*L_{night} + 0.01395*(L_{night})^2 \quad [14]$$

and for railways:

$$\%HSD = 11.3 - 0.55*L_{night} + 0.00759*(L_{night})^2 \quad [15]$$

$$\%SD = 12.5 - 0.66*L_{night} + 0.01121*(L_{night})^2 \quad [16]$$

$$\%LSD = 4.7 - 0.31*L_{night} + 0.01125*(L_{night})^2 \quad [17].$$

The above relations represent the current best estimates of the influences of L_{night} on self-reported sleep disturbance for road traffic noise and for railway noise, when no other factors are taken into account. Fig. 4.1 illustrates the relations [9] [12] and [15] for persons highly disturbed by road, aircraft and rail noise.

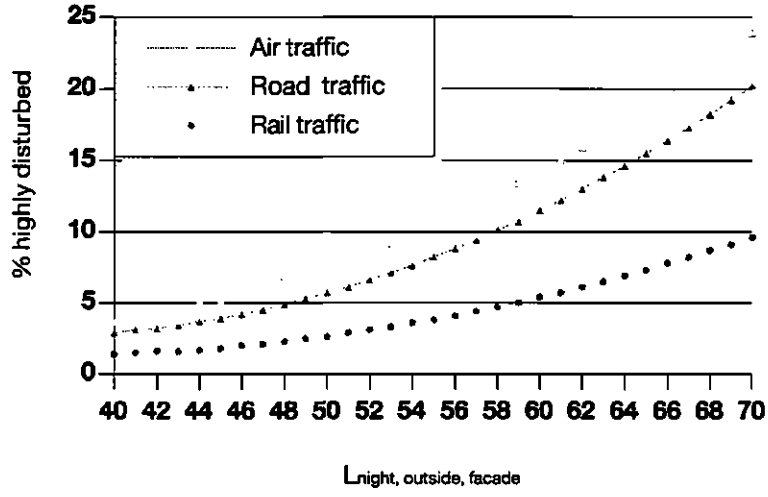


Fig. 4.1
High sleep disturbance by noise at night

Source:
European Commission, 2004.

With regard to the relations for aircraft noise it should be noted that the variance in the responses is large compared to the variance found for rail and road traffic. This means that the uncertainty regarding the responses for night-time aircraft noise is large, and such responses can be considered as indicative only. Miedema (2003) suggests the following causes.

- The time pattern of noise exposures around different airports varies considerably due to specific night-time regulations.
- The sleep disturbance questions for aircraft noise show a large variation.
- The most recent studies show the highest self-reported sleep disturbance at the same L_{night} level. This suggests a time trend.

For industrial noise there is an almost complete lack of information, although there are some indications (Vos, 2003) that impulse noise may cause considerable disturbance at night.

4.3 COMPLAINTS

According to the Health Council of the Netherlands (2004), the submission of a complaint about noise is symptomatic of reduced well-being.

Complaints about noise are widespread, and night noise seems to cause more complaints than daytime noise at the same level. Hume, Morley and Thomas (2003) found that around Manchester Airport complaints per 1000 aircraft traffic movements rose from an average of 10 in daytime hours to up to 80 in the night. When linking part of the complaints to measured noise levels, they found an increase from an average of 1 complaint at 70 PNLdB (circa 58 L_{Amax}) to 2 at 114 PNLdB (circa 102 L_{Amax}).

Due to differences in complaint cultures and registration practices, it is difficult to make comparisons between complaint registrations. Around Amsterdam Schiphol Airport a relation between complaints and L_{Aeq} was found (Ministerie Verkeer en Waterstaat, 2005). The threshold for complaints is around 45 L_{den}, and increases to 7% of the population at 72 L_{den}. Night-time complaints follow the same pattern, and the threshold for night complaints is 35 L_{night}. In Fig. 4.2 the mean percentage shows a definite relationship with L_{night}. The 95 percentile indicates that the threshold is 35 L_{night}.

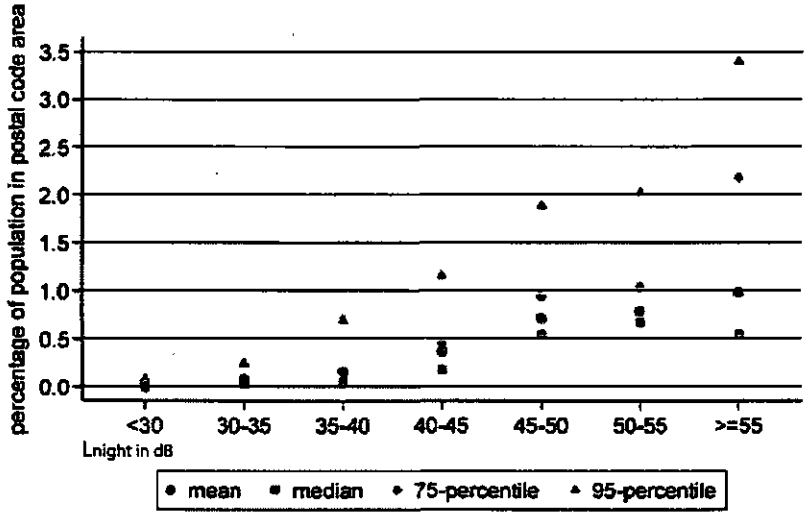


Fig. 4.2
Dose-effect relation for persons having complained at least once during a year between 1994 and 2004

Source: Ministerie Verkeer en Waterstaat (2005).

4.4 NEIGHBOURHOOD NOISE AND NOISE FROM NEIGHBOURS

Inventory studies in the Netherlands indicate that sleep disturbance attributable to the most annoying forms of neighbourhood noise and noise from neighbours (contact noise and human noises in the environment) is on a similar scale to disturbance attributable to the most annoying sources of road traffic noise (mopeds and passenger cars). It is reasonable to assume that chronic sleep disturbance is, in the long term, liable to have consequences for health and well-being. The sound pressure level and other noise characteristics are liable to determine the nature of the influence to some extent, but certain other factors play a more prominent role than is the case with traffic noise. These factors include appreciation of the noise and of the party responsible for the noise, as well as the hearer's personal circumstances. However, scientific understanding of the relative importance of and interaction between

acoustic and non-acoustic factors is not sufficient for the committee to draw any definitive conclusions regarding the relationship between, on the one hand, exposure to night-time neighbourhood noise and noise from neighbours and, on the other, health and well-being.

Leidelmeijer and Marsman (1997) carried out an interview-based study of 1242 households in the Netherlands, in which subjects were asked about daytime and night-time noise from neighbours and any associated annoyance. A distinction was made on the basis of the part of the house in which the noises were audible and any associated annoyance was experienced. Subjects proved least tolerant of noise from their neighbours that was audible in the master bedroom. The researchers distinguished five types of noise, which are listed below (Table 4.1), along with the percentage of subjects who indicated hearing the relevant type of noise from a neighbouring dwelling at night in the master bedroom.

Table 4.1 Daytime and night-time noise from neighbours

Source: Leidelmeijer and Marsman, 1997.

Type of noise	% subjects hearing noises at night in the bedroom
Contact noise	22%
Noise from sanitary fittings, central heating, etc.	19%
Noise from radio, TV and hi-fi	12%
Do-it-yourself noises	8%
Pets	6%

Where each of the five investigated types of noise was concerned, roughly 10–15% of subjects indicated that they felt it was unacceptable for the noise to be audible during the day. Overall, nearly 30% of subjects said that sanitary fittings should not be audible at night, while approximately 50% felt each of the other four types of noise was unacceptable at night.

In 1993, Kranendonk, Gerretsen and van Luxemburg produced a synthesis of the research conducted up to that point in time into the annoyance associated with noise from neighbours. Subsequently, in 1998, van Dongen et al. published a report on the relationship between noise from neighbouring dwellings and the airborne and contact noise attenuating indices I_{lu}, $I_{lu;k}$, and I_{co}, drawing on data from a questionnaire-based survey of the residents of 600 dwellings, whose acoustic quality was determined in 202 cases. The results of the two studies are reasonably consistent. Both found that the chief causes of annoyance were loud radios, hi-fis and TVs, audible and sometimes intelligible voices, the slamming of doors and footsteps on floors and staircases. In both cases, it proved that, when Ilu had a value of 0 (the minimum requirement for new homes), 10% of subjects reported high annoyance and 15% reported annoyance caused by noise from neighbouring dwellings. These figures are not specific to night-time noise, but apply to annoyance over a 24-hour period.

On the basis of the findings outlined above, the committee concludes that the standard of inter-dwelling sound attenuation presently required does not provide sufficient protection to prevent annoyance caused by noise from neighbours. Since people are less tolerant of the noise their neighbours make at night-time than of their neighbours' evening or daytime noise, it may be assumed that much of the annoyance associated with noise from neighbours relates to the influence of such noise on sleep.

4.5 CARDIOVASCULAR EFFECTS OF NOISE – FINDINGS FROM EPIDEMIOLOGICAL STUDIES

4.5.1 INTRODUCTION

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 (WHO Regional Office for Europe, 2000; Schwela, 2000). Besides the psychosocial effects of community noise, there is concern about the impact of noise on public health, particularly regarding cardiovascular outcomes (Suter, 1992; Passchier-Vermeer and Passchier, 2000; Stansfeld, Haines and Brown, 2000). Non-auditory health effects of noise have been studied in humans for a couple of decades using laboratory and empirical methods. Biological reaction models have been derived, which are based on the general stress concept (Selye, 1956; Henry and Stephens, 1977; Ising et al., 1980; Lercher, 1996). Amongst other non-auditory health end points, short-term changes in circulation including BP, heart rate, cardiac output and vasoconstriction, as well as stress hormones (epinephrine, norepinephrine and corticosteroids) have been studied in experimental settings for many years (Berglund and Lindvall, 1995; Babisch, 2003). Various studies have shown that classical biological risk factors are higher in subjects who were exposed to high levels of traffic noise (Arguelles et al., 1970; Eiff et al., 1974; Verdun di Cantogno et al., 1976; Algers, Ekesbo and Strömberg, 1978; Knipschild and Sallé, 1979; Manninen and Aro, 1979; Eiff et al., 1981a; Rai et al., 1981; Marth et al., 1988; Babisch and Gallacher, 1990; Babisch et al., 1990; Lercher and Kofler, 1993; Schulte and Otten, 1993; Dugué Leppänen and Gräsbeck, 1994; Yoshida et al., 1997; Goto and Kaneko, 2002). Although controls for other risk factors were not consistent in all these studies, the hypothesis emerged that persistent noise stress increases the risk of cardiovascular disorders including high BP (hypertension) and IHD.

- Sound/noise is a psychosocial stressor that activates the sympathetic and endocrine system.
- Acute noise effects do not only occur at high sound levels in occupational settings, but also at relatively low environmental sound levels when, more importantly, intended activities such as concentration, relaxation or sleep are disturbed.

The following questions need to be answered.

- Do these changes observed in the laboratory habituate or persist under chronic noise exposure?
- If they habituate, what are the physiological costs? If they persist, what are the long-term health effects?

The answers to these questions come from epidemiological noise research. Large-scale epidemiological studies have been carried out for a long time (Babisch, 2000). The studies suggest that transportation noise is associated with adverse cardiovascular effects, in particular IHD. The epidemiological evidence is constantly increasing (Babisch, 2002, 2004a). The biological plausibility of the association derives from the numerous noise experiments that have been carried out in the laboratory. There is no longer any need to prove the noise hypothesis as such. Decision-making and risk management, however, rely on a quantitative risk assessment which requires an established dose–response relationship. Since many of the stress indicators and risk factors that have been investigated in relation to

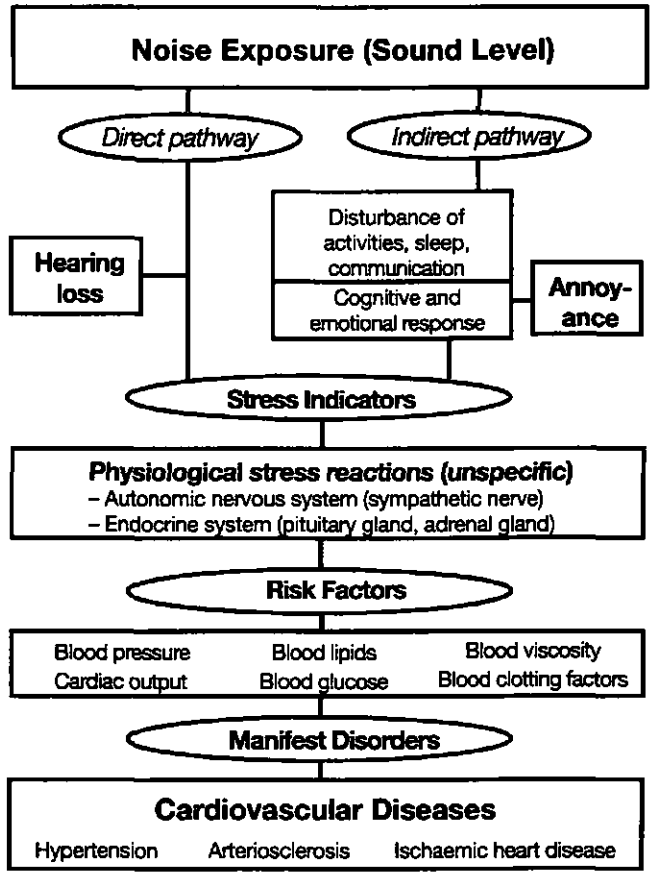


Fig. 4.3
Noise effects reaction scheme

Source:
Babisch, 2002.

noise are known to be classical cardiovascular risk factors, the hypothesis has emerged that chronic noise exposure increases the risk of hypertension, arteriosclerosis and IHD. Its relevance for public health comes from the high prevalence of cardiovascular diseases in developed and industrialized countries. It is unclear as to what extent chronically repeated noise-induced sleep disturbance contributes to the development of somatic health disorders. Only a few epidemiological studies address this particular issue. Epidemiological noise research has seldom distinguished between day and night exposures, or between the exposure of the living room and the bedroom. However, some deduction can be made from daytime to night-time exposure.

4.5.2 NOISE AND STRESS-REACTION MODEL

The auditory system is continuously analysing acoustic information, which is filtered and interpreted by different cortical and subcortical brain structures. The limbic system, including the hippocampus and the amygdala, plays an important role in the emotional processing pathways (Spreng, 2000). It has a close connection to the hypothalamus that controls the autonomic nervous system and the hormonal balance of the body. Laboratory studies found changes in blood flow, BP and heart rate in reaction to noise stimuli as well as increases in the release of stress hormones including the catecholamines adrenaline and noradrenaline, and the corticosteroid

cortisol (Berglund and Lindvall, 1995; Maschke, Rupp and Hecht, 2000; Babisch, 2003). Such changes also occur during sleep without the involvement of cortical structures. The amygdala has the capacity to learn due to its plasticity, particularly with respect to the meaning of sound stimuli (for example, danger of an approaching lorry) (Spreng, 2000, 2004). Acoustic stimulation may act as an unspecific stressor that arouses the autonomous nervous system and the endocrine system. The generalized psychophysiological concept given by Henry and Stephens can be applied directly to noise-induced stress reaction (Henry, 1992). The stress mechanism as such is genetically determined but it may be modified by experience and environmental factors. Its biological function is to prepare the organism to cope with a demanding stressor. The arousal of the sympathetic and endocrine system is associated with changes in physiological functions and the metabolism of the organism, including BP, cardiac output, blood lipids (cholesterol, triglycerides, free fatty acids, phosphatides), carbohydrates (glucose), electrolytes (magnesium, calcium), blood clotting factors (thrombocyte aggregation, blood viscosity, leukocyte count) and others (Friedman and Rosenman, 1975; Cohen, Kessler and Underwood Gordon, 1995; Lundberg, 1999). In the long term, functional changes and dysregulation may occur, thus increasing the risk of manifest diseases.

Fig. 4.3 shows the principal reaction schema used in epidemiological noise research for hypothesis testing (Babisch, 2002). It simplifies the cause–effect chain, that is: sound – annoyance (noise) – physiological arousal (stress indicators) – (biological) risk factors – disease – and mortality (the latter is not explicitly considered in the graph). The mechanism works "directly" through synaptic nervous interactions and "indirectly" through the emotional and the cognitive perception of the sound. It should be noted that the "direct" pathway is relevant even at low sound levels particularly during sleep, when the organism is at its nadir of arousal. The objective noise exposure (sound level) and the subjective noise "exposure" (annoyance) may serve independently as exposure variables in the statistical analyses of the relationship between noise and health end points.

Principally, the effects of environmental noise cannot directly be extrapolated from results of occupational noise studies. The two noise environments cannot simply be merged into one sound energy-related dose–response model (for example, a simple 24-hour average noise level measured with a dose-meter). Noise effects are not only dependent on the sound intensity but also on the frequency spectrum, the time pattern of the sound and the individuals' activities which are disturbed. Therefore, epidemiological studies carried out under real-life conditions can provide the basis for a quantitative risk assessment provided that there is adequate control over confounding and exposure variables. Other noise sources might act as confounders and/or effect modifiers on the association of interest. The effects of road traffic noise (at home) were shown to be stronger in subjects who were also exposed to high noise levels at work (Babisch et al., 1990).

4.5.3 PREVIOUS REVIEWS ON ENVIRONMENTAL NOISE AND CARDIOVASCULAR RISK

Causality in epidemiology can never be completely proven (Schlesselman, 1987; Christoffel and Teret, 1991; Weed, 2000). It is a gradual term for which evidence is increasing with the increasing number of facts. However, the magnitude of effect, the presence of a dose–response relationship and consistency with other studies in different populations and with different methodology and biological plausibility are

commonly accepted arguments for a causal relationship (Bradford Hill, 1965; Evans, 1976; Morabia, 1991; Weed and Hursting, 1998). Classical, systematic and quantitative reviews have been published in the past, summarizing the results of studies that have been carried out up to the end of the last century, and assessing the evidence of the relationship between community noise and cardiovascular disease outcomes (Health Council of the Netherlands, 1994, 1999, 2004; Berglund and Lindvall, 1995; IEH, 1997; Morrell, Taylor and Lyle, 1997; Porter, Flindell and Berry, 1998; Babisch, 2000; Passchier-Vermeer and Passchier, 2000), including a classical review and synthesis report by Babisch (2000) and a systematic review (meta-analysis) by van Kempen et al. (2002).

In a meta-analysis it was concluded that the risk of hypertension due to aircraft noise was 1.26 per increase of 5 dB(A) (95% CI: 1.14–1.39, L_{day} = 55–72 dB(A)) (van Kempen et al., 2002). But, only one study (Knipschild, 1977a) was considered in the meta-analysis. With respect to road traffic noise and hypertension a pooled estimate of 0.95 per 5 dB(A) (95% CI: 0.84–1.08, L_{day} = <55–80 dB(A)) was calculated (van Kempen et al., 2002). Two cross-sectional studies (Knipschild and Sallé, 1979; Knipschild, Meijer and Sallé, 1984) were considered in this calculation. The highest degree of evidence was for the association between community noise and IHD. Across the studies there was not much indication of an increased risk for subjects who lived in areas with a daytime average sound pressure level of less than 60 dB(A). For higher noise categories, however, higher risks were relatively consistently found amongst the studies (Babisch, 2004a). Statistical significance was rarely achieved.

Some studies permit reflections on dose–response relationships. These mostly prospective studies suggest an increase in risk for outdoor noise levels above 65–70 dB(A) during the daytime, the relative risks ranging from 1.1 to 1.5. Noise effects were larger when mediating factors like years in residence, room orientation and window-opening habits were considered in the analyses. In a meta-analysis it was concluded that the risk of IHD increased by 1.09 per 5 dB(A) of the road traffic noise level (95% CI: 1.05–1.13, L_{day} = 51–70 dB(A)) (van Kempen et al., 2002), when two cross-sectional studies (Babisch et al., 1993a) were considered. However, the pooled estimate of two prospective studies (Babisch et al., 1999) was calculated to be 0.97 per 5 dB(A) (95% CI: 0.90–1.04, L_{day} = 51–70 dB(A)) (van Kempen et al., 2002). When the diagnosis of IHD was limited to myocardial infarction, three studies (Babisch et al., 1999, 1994) were considered in this meta-analysis. Then the linear effect estimate was 1.03 per 5 dB(A) increase in road traffic noise level (95% CI: 0.99–1.09, L_{day} = 51–80 dB(A)). New studies have appeared in the meantime which are included in the present updated review (Matsui et al., 2001; Bluhm, Nordling and Berglind, 2001; Evans et al., 2001; Rosenlund et al., 2001; Belojevic and Saric-Tanaskovic, 2002; Goto and Kaneko, 2002; Lercher et al., 2002; Maschke, 2003; Franssen et al., 2004; Matsui et al., 2004; Niemann and Maschke, 2004; Babisch et al., 2005). Others are on their way or have not yet been finalized and published, for instance the pan-European HYENA project (Jarup et al., 2003).

4.5.4 UPDATED REVIEW OF EPIDEMIOLOGICAL STUDIES

Sixty epidemiological studies were recognized as having either objectively or subjectively assessed the relationship between transportation noise and cardiovascular end points. The identification of studies was based on the author's expert

knowledge of the topic and respective literature. Details are given in the major report (Babisch, 2006). Information particularly on night-time exposure (L_{night}: 22.00–06.00 or 23.00–07.00) was seldom available. Newer studies used non-weighted or weighted averages of the 24-hour exposure (L_{eq}, L_{dn}, L_{den}). Some aircraft noise studies used national calculation methods (for example, Dutch Kosten Units). For comparisons of study results and the pooling of data (meta-analysis), sound levels were converted on the basis of best guess approximations to L_{day} (Matschat and Müller, 1984; Passchier-Vermeer, 1993; Bite and Bite, 2004; Franssen et al., 2004). It should be noted in this context that doubling/halving of road traffic volume results in a 3 dB(A) higher/lower average sound pressure level. Not all studies allowed dose–response reflections because some of them considered very broad exposure categories. Besides objective noise measurements, subjective measurements of exposure have been used in some epidemiological noise studies, which is in accordance with the noise–stress model. Type of road (for example, busy street, side street, etc.), disturbances and annoyance were rated by the study subjects from given scales.

4.5.5 MEAN BP

Table A2 of the major report (Babisch, 2006) lists the major findings of epidemiological traffic noise studies in which mean BP was considered as the outcome. It indicates mean systolic and diastolic BP differences as obtained from extreme group comparisons of noise exposure. The effects in children and in adults are discussed separately. The findings in children are difficult to interpret with regard to possible health risks in their later life. The effect may be of a temporary nature and may not be relevant to permanent health damage. There is evidence during childhood (Gillman et al., 1992), adolescence (Yong et al., 1993) and adulthood (Tate et al., 1995) that the BP level at an early age is an important predictor of the BP level at a later age. Studies over the full age range are missing (tracking). Growth and body weight are important factors in BP development. The impact of body size was not adequately considered in some of the studies. A crude hint regarding reversible effects on BP came from one study (Morrell et al., 2000). Results of the Munich intervention study on the effects of a reduction of aircraft noise have only been reported regarding cognitive performance but not with respect to change of BP (Hygge, Evans and Bullinger, 2002). It was concluded from the available data on the length of exposure that children do not seem to adapt to high levels of road traffic noise but to some extent to aircraft noise (Passchier-Vermeer, 2000; Bistrup et al., 2001). However, the database appears to be too poor to draw final conclusions. Aircraft noise studies focused on exposure at school, while road traffic noise studies mostly considered noise exposure at home. The conclusions given by Evans and Lepore (1993) seem still to hold true:

> "We know essentially nothing about the long-term consequences of early noise exposure on developing cardiovascular systems. The degree of blood pressure elevations is small. The clinical significance of such changes in childhood blood pressure is difficult to determine. The ranges of blood pressure among noise-exposed children are within the normal levels and do not suggest hypertension. The extent of BP elevations found from chronic exposure are probably not significant for children during their youth, but could portend elevations later in life that might be health damaging."

Regarding mean BP, no consistent findings in the relationship between traffic noise level and mean systolic or diastolic BP can be seen in adults across the studies. In longitudinal studies, problems arose from migration of subjects, which had a considerable impact on sample size. The latter problem also applies to cross-sectional studies, in general. Sensitive subjects may tend to move out of the polluted areas, which dilutes the effect of interest. Medication due to high BP may affect the BP readings. However, the exclusion of subjects with hypertension or hypertension treatment dilutes the true effect on BP differences, if the hypothesis (noise causes high BP) is true. In principle, hypotension – a fall in BP – can also be a stress reaction. All this makes it more reasonable to look at manifest hypertension (defined by a cut-off criterion) as a clinical outcome rather than at mean BP readings (Ising, 1983; Winkleby, Ragland and Syme, 1988). To date, there is no evidence from epidemiological data that community noise increases mean BP readings in the adult population. However, this does not discard the noise hypothesis as such. Studies suffered from insufficient power, narrow exposure range or other difficulties in the study design.

4.5.6 HYPERTENSION

Table A3 of the major report (Babisch, 2006) gives the results of epidemiological traffic noise studies on the relationship between community noise level and the prevalence or incidence of hypertension. Hypertension in these studies was either defined by WHO criteria (WHO-ISH Guidelines Subcommittee, 1999), similar criteria based on measurements of systolic and diastolic BP, from information which was obtained from a clinical interview, or a social survey questionnaire about hypertension diagnosed by a doctor. Most studies refer to road traffic noise. However, in recent years some new aircraft noise studies entered the database. The subjects studied were the adult male and female population, sometimes restricted to certain age ranges. With regard to the association between community noise and hypertension, the picture is heterogeneous. With respect to aircraft noise and hypertension, studies consistently show higher risks in higher exposed areas. The evidence has improved since a previous review (Babisch, 2000). The relative risks found in four significantly positive studies range between 1.4 and 2.1 for subjects who live in high exposed areas, with approximate daytime average sound pressure level in the range of 60–70 dB(A) or more. Swedish studies found a relative risk of 1.6 at even lower levels >55 dB(A). With respect to road traffic noise, the picture remains unclear. New studies, more than older studies, tend to suggest a higher risk of hypertension in subjects exposed to high levels of road traffic noise, showing relative risks between 1.5 and 3.0. However, the earlier studies cannot be neglected in the overall judgement process. Across all studies no consistent pattern of the relationship between community noise and prevalence of hypertension can be seen. Dose–response relationships were considered in new studies. Subjective ratings of noise or disturbances due to traffic noise seem to consistently show a positive association with prevalence of hypertension. The relative risks found here range from 0.8 to 2.3. These studies, however, are of lower validity due to principal methodological issues regarding over-reporting (Babisch et al., 2003).

4.5.7 IHD

Table A5 of the major report (Babisch, 2006) gives the results of cross-sectional epidemiological traffic noise studies on the relationship between noise level and prevalence of IHD. Table A6 of the major report gives the results of case-control

and cohort studies on the association between noise level and incidence of IHD. In cross-sectional studies, IHD prevalence was assessed by clinical symptoms of angina pectoris, myocardial infarction, ECG abnormalities as defined by WHO criteria (Rose and Blackburn, 1968), or from self-reported questionnaires regarding doctor-diagnosed heart attack. In longitudinal studies, IHD incidence was assessed by clinical myocardial infarction as obtained from hospital records, ECG measurements or clinical interviews. The majority of studies refer to road traffic noise. With regard to IHD, the evidence of an association between community noise and IHD risk has increased since a previous review (Babisch, 2000). There is not much indication of a higher IHD risk for subjects who live in areas with a daytime average sound pressure level of less than 60 dB(A) across the studies. For higher noise categories, a higher IHD risk was relatively consistently found amongst the studies. Statistical significance was rarely achieved. Some studies permit reflections on dose–response relationships. These mostly prospective studies suggest an increase in IHD risk at noise levels above 65–70 dB(A), the relative risks ranging from 1.1 to 1.5 when the higher exposure categories were grouped together. Noise effects were larger when mediating factors like residence time, room orientation and window-opening habits were considered in the analyses. This accounts for an induction period (Rose, 2005) and improves exposure assessment. The results appear as consistent when subjective responses of disturbance and annoyance are considered, showing relative risks ranging from 0.8 to 2.7 in highly annoyed/disturbed/affected subjects. However, these findings may be of lower validity due to methodological issues.

4.5.8 MEDICATION AND DRUG CONSUMPTION

Table A8 of the major report (Babisch, 2006) gives the results of studies on the relationship between drug consumption and community noise. Medication was primarily investigated with respect to aircraft noise. A significant prevalence ratio for medication with cardiovascular drugs of 1.4 was found in the sample of Amsterdam Schiphol Airport (Knipschild, 1977a). The results of the "drug survey", where the annual data of the pharmacies regarding the purchase of cardiovascular drugs were analysed (repeated cross-sectional survey), supported this finding. An increase in drug purchase over time in the exposed areas and not in the less exposed was found. This refers to the purchase of cardiovascular and antihypertensive drugs, as well as the purchase of hypnotics, sedatives and antacids (Knipschild and Oudshoorn, 1977). Furthermore a dependency with changes in night flight regulations was found (decrease after reduction of night flights). A large recent study around Amsterdam Schiphol Airport found only a slightly higher risk of self-reported medication with cardiovascular drugs, including antihypertensive drugs (relative risk 1.2), in subjects exposed to aircraft noise where the noise level L_{den} exceeded 50 dB(A) (Franssen et al., 2004). Dose–response relationships across noise levels (L_{den} = <50–65 dB(A)) with respect to prescribed and non-prescribed sedatives/sleeping pills were found (relative risk 1.5 and 2.0, respectively) in the highest noise category of L_{den} = 61–65 dB(A). The preliminary results of an ongoing aircraft noise study from Sweden carried out around Stockholm's airport are in line with the Dutch studies (Bluhm et al., 2004). A significant relative risk of 1.6 for the use of antihypertensive drugs was found in male subjects, where the noise level according to the Swedish calculation standard exceeded FBM = 55 dB(A). The road traffic noise studies, where medication/purchase of drugs was investigated also tend to show a higher use in higher exposed subjects (Eiff and Neus, 1980; Schulze et al., 1983; Lercher,

1992). The relative risk for cardiovascular drugs was 1.3 in the Bonn study and 5.0 in the Erfurt study. The results for other drugs including sleeping pills, sedatives, tranquillizers and hypnotics ranged between 1.2 and 3.8 in these studies. All in all, the studies on the relationship between the use of medication or purchase of drugs and community noise support the general hypothesis of an increase in sleep disturbance and cardiovascular risk in noise-exposed subjects.

4.5.9 EVALUATION OF STUDIES

This section refers only to studies where the prevalence or the incidence of manifest cardiovascular diseases was considered as a potential health outcome of chronic exposure to environmental noise. The focus here is on a quantitative risk assessment with respect to manifest diseases. Furthermore, studies on the effects of low-altitude jet-fighter noise are also excluded, because this type of noise includes other dimensions of stress (for instance, fear). Thirty-seven studies have assessed the prevalence or incidence of manifest diseases, including hypertension and IHD (angina pectoris, myocardial infarction, ECG abnormalities).

4.5.9.1 Criteria

Epidemiological reasoning is largely based on the magnitude of effect estimates, dose–response relationships, consistency of findings, biological plausibility of the effects and exclusion of possible bias. Internal (the role of chance) and external validity (absence of bias and confounding) are important issues in the evaluation of studies (Bradford Hill, 1965). Analytic studies (for example, cohort or case-control studies) are usually considered as having a higher validity and credibility than descriptive studies (for example, cross-sectional or ecological studies) (Hennekens and Buring, 1987), although many of the reservations against cross-sectional studies seem to be of minor importance when considering noise. For example, it does not appear to be very likely that diseased subjects tend to move differentially more often into exposed areas. Rather the opposite may be true, if noise stress is recognized as a potential cause of the individual's health problem. Thus, a cross-sectional study design may act conservatively on the results. The presence of a dose–response relationship is not a necessary criterion of causality. Non-linear relationships, including "u-" or "j-" shaped, saturation and threshold effects may reflect true associations (Calabrese and Baldwin, 2003; Rockhill, 2005). With respect to the derivation of guideline values in public health policy, the assessment of a dose–response relationship enables a quantitative risk assessment on the basis of continuous or semi-continuous (for instance 5 dB(A) categories) exposure data. Dichotomous exposure data, on the other hand, that refer to a cut-off criterion which splits the entire exposure range into two halves, can be used to evaluate the hypothesis of an association (qualitative interpretation), but not a quantitative assessment. The objective or subjective assessment of exposure and/or health outcomes is an important issue when judging the validity of a study (Malmström, Sundquist and Johansson, 1999; Cartwright and Flindell, 2000; Hatfield et al., 2001). The objective prevalence of hypertension was found to be higher in a population sample than the subjective prevalence of hypertension (Schulte and Otten, 1993). In a telephone survey more than half of the hypertensives classified themselves as normotensive (sensitivity 40% for men and 46% for women) (Bowlin et al., 1993). In a representative health survey, the validity of the self-reported assessment of morbidity (subjective morbidity) was found to be "low" with respect to hypercholesterolaemia, "intermediate" with respect to angina pectoris, hypertension and stroke and "high" with respect to

myocardial infarction (Bormann et al., 1990). Myocardial infarction is a very definite and severe health outcome which subjects would clearly know about if they had experienced it. Its assessment by questionnaire tends to be more credible than that regarding hypertension. Test–retest reliability was found to be good with respect to "harder" outcomes, including high BP and heart attack (Lundberg and Manderbacka, 1996; Lipworth et al., 2001). Over-reporting, on the other hand, may be a source of potential bias, particularly when both exposure and outcome are assessed on a subjective basis (Winkleby, Ragland and Syme, 1988; Babisch et al., 2003). The subjects may be more prone to blame their environment for their health problems, or may even tend to exaggerate adverse effects or exposure in order to influence noise policy. Therefore, a higher credibility and ranking was given to studies where exposure and outcome were assessed objectively (for example sound level versus subjective ratings, and measurement of BP or a clinical interview versus self-reported hypertension in a self-administered questionnaire). This means that the sound level must have been measured or calculated on the basis of the traffic counts, and clinical interviews or measurements must have been carried out by medically trained personnel (no self-administered questionnaire data) to give a study a high ranking. Studies which have been adequately controlled (for instance stratification, model adjustment (regression), matching) for a reasonable set of confounding variables in the statistical analyses, besides age and sex, were given a high ranking.

4.5.9.2 Assessment

The evaluation concerning the epidemiological studies was made with respect to the identification of good quality studies that can be feasibly considered for the derivation of guideline values. These studies can either be used for a statistical meta-analysis, for a combined interpretation (synthesis) or for singular interpretations. All the studies were evaluated with respect to the following criteria for inclusion or exclusion in the synthesis process. Necessary criteria were: (1) peer-reviewed in the international literature; (2) reasonable control of possible confounding; (3) objective assessment of exposure; (4) objective assessment of outcome; (5) type of study; and (6) dose–response assessment. All six criteria were fulfilled by the two prospective cohort studies carried out in Caerphilly and Speedwell (Babisch et al., 1999; Babisch, Ising and Gallacher, 2003), the two prospective case-control studies carried out in the western part of Berlin ("Berlin I" and "Berlin II") (Babisch et al., 1992, 1994), and the new prospective case-control study carried out in the whole of Berlin ("NaRoMI" = "Berlin III") (Babisch, 2004b; Babisch et al., 2005). The studies refer to road traffic noise and the incidence of myocardial infarction. They were also the only ones considered in an earlier meta-analysis on this issue (van Kempen et al., 2002), with the exception of the "NaRoMI" study, which was not available at that time. All these studies are observational analytic studies (Hennekens and Buring, 1987). If descriptive studies on individuals – namely cross-sectional studies – are allowed, another two studies from Caerphilly and Speedwell on the association between road traffic noise and the prevalence of IHD, myocardial infarction and angina pectoris can be taken into account (Babisch et al., 1988, 1993a, 1993b). These studies were also considered in the meta-analysis by van Kempen et al. (2002). However, the results of the Berlin study on the prevalence of myocardial infarction (Babisch et al., 1994) – which was also considered in that meta-analysis – are not considered here, because the outcome was assessed subjectively with a self-administered questionnaire (an exclusion criterion). All the studies suggest an increase in IHD, in particular myocardial infarction. These studies are used for a new meta-analysis (section 4.5.10).

Regarding aircraft noise, the cross-sectional Okinawa study (Matsui et al., 2001; Matsui et al., 2004) on the association between aircraft noise and hypertension fulfils the inclusion criteria. When studies are included that did not assess dose–response relationships but only compared dichotomous categories of exposure in the analyses, two more studies appear on the list. The studies were carried out in the vicinity of Amsterdam Schiphol Airport. They suggest a higher risk of cardiovascular diseases in general (Knipschild, 1977b), and – specifically – for hypertension and IHD (angina pectoris, ECG abnormalities, heart trouble) (Knipschild, 1977a) in subjects from areas exposed to high aircraft noise. These studies were considered in the meta-analysis by van Kempen et al. (2002). However, they do not fulfil the strict criteria set here. Finally, if the inclusion criteria are widened to include peer-reviewed studies that assessed dose–response relationships between objective indicators of exposure and the subjective (self-reported) prevalence of diseases, a further two studies can be considered. These are the cross-sectional study carried out in Stockholm regarding the association between aircraft noise and hypertension (Rosenlund et al., 2001), and the cross-sectional part of the study in Berlin regarding the association between road traffic noise and myocardial infarction (Babisch et al., 1994). Fig. 4.4 shows the results of the three aircraft noise studies carried out in Amsterdam, Okinawa and Stockholm (Knipschild, 1977a; Rosenlund et al., 2001; Matsui et al., 2004). The graph clearly indicates that the results are too heterogeneous to derive a pooled dose–response curve. However, all three studies show an increase in risk with increasing noise level.

Studies that are not given a high ranking according to the above mentioned criteria, however, may serve as additional sources of information to support the evidence of the conclusions being made on the basis of this review. This is illustrated in Fig. 4.5. The entries are relative risks (centre of the bars) with 95% confidence intervals (the bars) for dichotomous comparisons of noise exposure (extreme groups or high vs. low). A relative consistent shift of the bars to relative risks greater than 1 can be seen. The dark-shaded bars in the diagram refer to studies where the noise exposure was determined objectively (noise levels), the

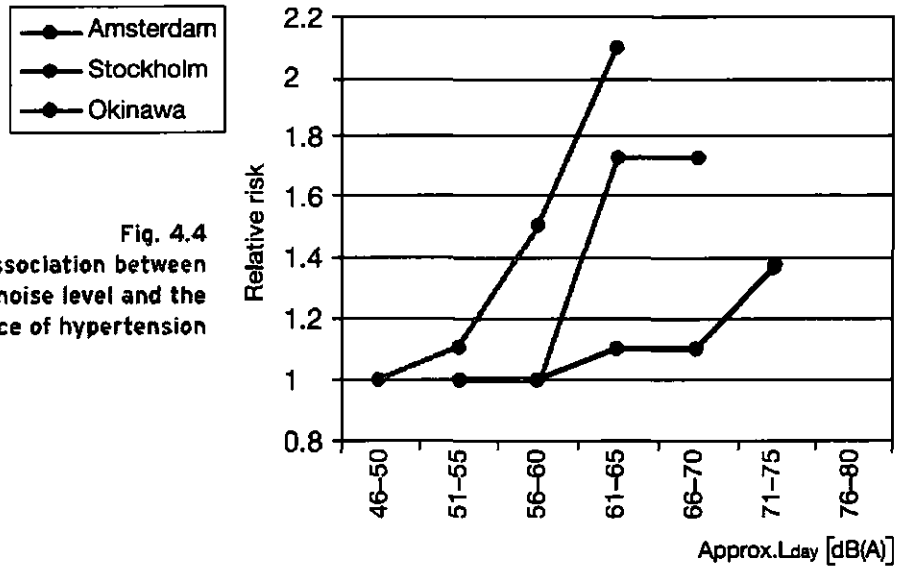


Fig. 4.4
Association between aircraft noise level and the prevalence of hypertension

light-shaded bars where it was determined subjectively (annoyance). Road traffic and aircraft noise studies are here viewed together. No corresponding results are available for rail traffic studies. If different subgroups of the population (males/females) or different health end points were taken into account, specific studies appear more than once in the illustration.

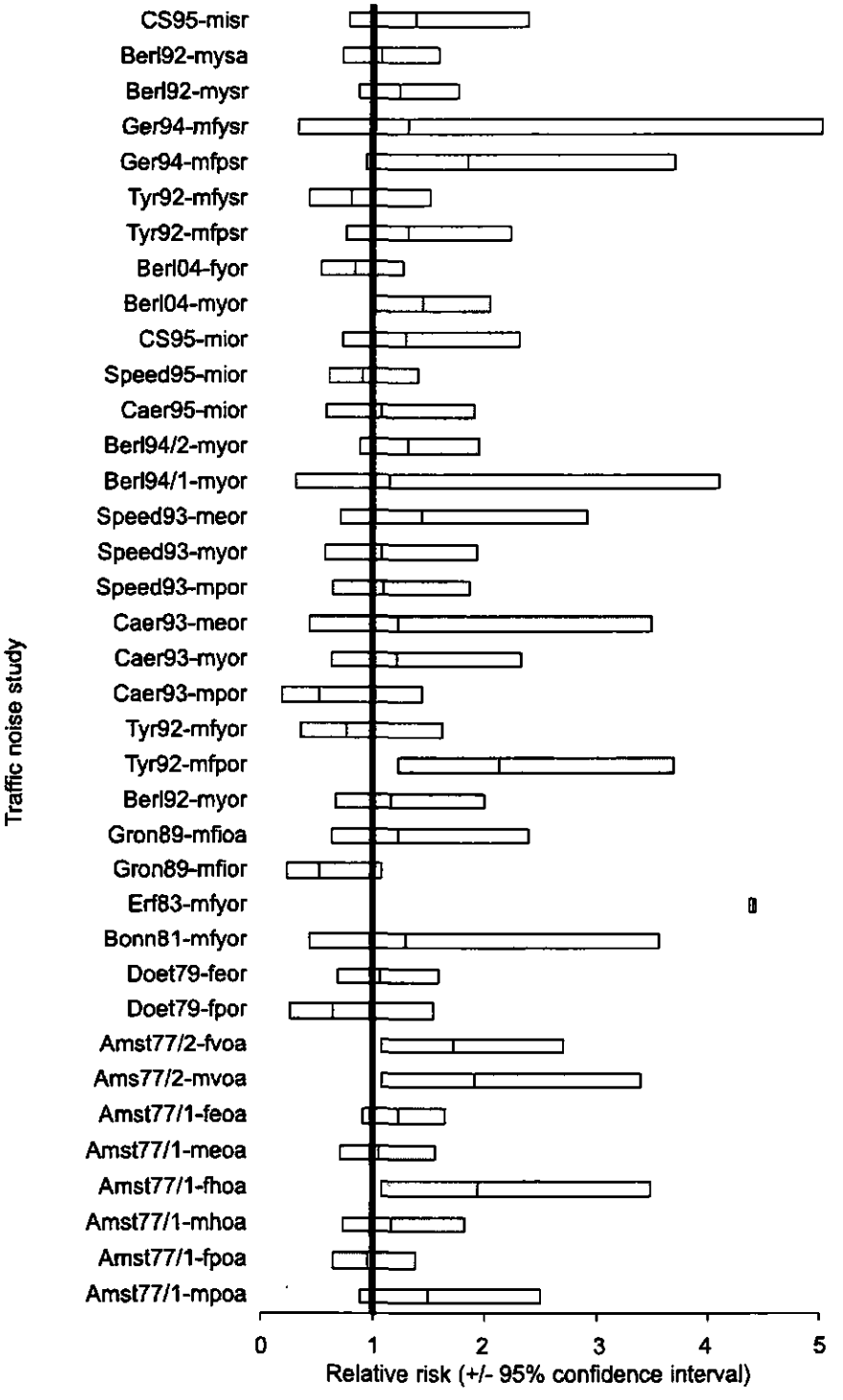


Fig. 4.5
Results of epidemiological studies on the association between traffic noise and ischaemic heart disease

Captions: sex:
f = female,
m = male;
noise measurement:
o = objective (sound level), dark-shaded beam;
s = subjective (annoyance), light-shaded beam;
type of noise:
a = aircraft noise;
r = road traffic noise;
ischaemic heart disease:
e = ECG-ischaemic signs,
h = heart complaints,
i = ischaemic heart disease,
p = angina pectoris,
v = cardiovascular complaints in general, y = heart attack.

Source: Babisch, 2002, modified according to the results of Babisch, 2004b.

4.5.10 DOSE-RESPONSE CURVE: META-ANALYSIS

For a quantitative risk assessment and the derivation of guidelines for public health noise policy a common dose–response curve is required. The risk estimates obtained from different noise studies can be summarized using the statistical approach of a meta-analysis. Based on the judgement criteria discussed in section 4.5.9.2, five analytic and two descriptive studies emerged that can be used to derive a common dose–response curve for the association between road traffic noise and the risk of myocardial infarction. Two separate meta-analyses were made by considering the analytic studies that were carried out in Caerphilly and Speedwell (cohort studies) and Berlin (case-control studies) on the one hand, and the descriptive studies that were carried out in Caerphilly and Speedwell (cross-sectional studies) on the other hand. It turned out, as a result of the evaluation, that all these studies referred to road traffic noise during the day (L_{day}: 06.00–22.00) and the incidence or prevalence of myocardial infarction as the outcome. Study subjects were men. In all analytic studies the orientation of rooms was considered for the exposure assessment (facing the street or not). With respect to the Caerphilly and Speedwell studies, the six years of pooled follow-up data provided the respective information. In all descriptive studies the traffic noise level referred to the facades that were facing the street and did not consider the orientation of rooms/windows. All individual effect estimates were adjusted for the covariates considered in each of the studies. Different sets of covariates were considered in each study. However, this pragmatic approach accounts best for possible confounding in each study and provides the most reliable effect estimates derived from each study. The concept of meta-analysis was used to aggregate and summarize the findings of the different studies (Olkin, 1995; Blettner et al., 1999). The program "meta" was downloaded from the "STATA" web site for use in the statistical package STATA (version 8.0), and for calculating the pooled random effect estimates.

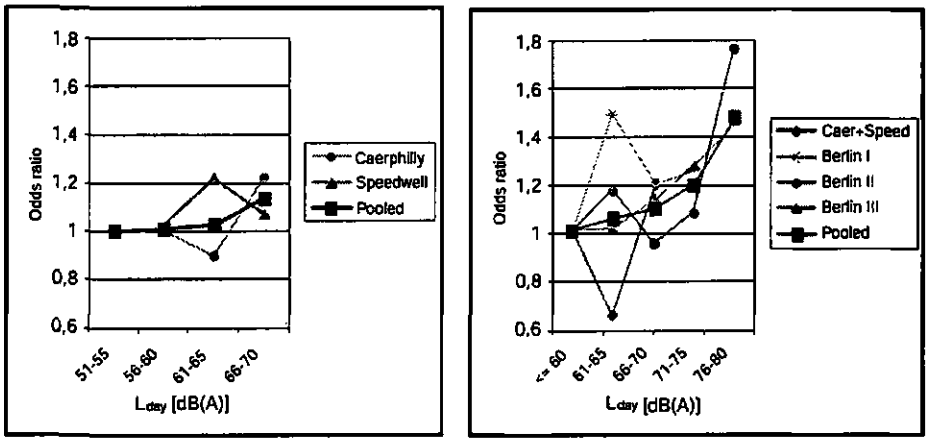
Table 4.2

Single and pooled (meta-analysis) effect estimates (odds ratios and 95% confidence intervals) of descriptive and analytic studies on the relationship between road traffic noise level (L_{day}) and the incidence/prevalence of myocardial infarction

	Road traffic noise level - L_{day} [dB(A)]					
Descriptive studies	51-55	56-60	61-65	66-70		N
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 (0.70-2.21), [12.62]	1.07 (0.59-1.94), [10.94]		2348
Pooled	1.00	1.01 (0.68-1.50)	1.02 (072-1.47)	1.14 (0.73-1.76)		
Q-Test		p=0.96	p=0.41	p=0.77		
Analytic 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 (0.69-1.68), [18.92]	1.46 (0.77-2.78), [9.27]	6035
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	

Table 4.2 shows individual and pooled effect estimates with confidence intervals (rounded brackets), statistical weights (square brackets) for the individual studies, and the Q-test of heterogeneity between studies. According to the Q-test, the nil hypothesis of non-heterogeneity was never discarded. Figs 4.6 and 4.7 show odds ratios of individual studies and the pooled estimates for the descriptive and analytic studies.

Fig. 4.6 and Fig. 4.7
Single and pooled effect estimates (odds ratios) for the descriptive and analytic studies of the association between road traffic noise level and the prevalence (left graph) and incidence (right graph), respectively, of myocardial infarction



4.5.11 EFFECT MODIFICATION

Support for any noise–effect relationship may come from subgroup analyses that are in line with the noise hypothesis. This refers to effect modification with respect to residence time, window-opening behaviour and other determinants that affect the noise exposure and cumulative noise dose. In the Amsterdam aircraft noise studies, a steady increase in the purchase of cardiovascular and antihypertensive drugs at local pharmacies was found over the period of eight years in a community newly exposed to aircraft noise. No such increase was found in a control community that was not exposed to aircraft noise (Knipschild and Oudshoorn, 1977). Positive associations between the prevalence of cardiovascular diseases and residence time in exposed areas (but not in unexposed) were also found in the road traffic noise studies carried out in Bonn with respect to hypertension (Eiff and Neus, 1980; Neus et al., 1983) and in Caerphilly and Speedwell with respect to IHD (Babisch et al., 1999; Babisch, Ising and Gallacher, 2003). When the analyses of the road traffic noise studies carried out in Berlin, Caerphilly and Speedwell were restricted to subjects who had not moved within a retrospective period of 10–15 years, the effect estimates turned out to be larger than for the total samples of each study (Babisch et al., 1994, 1999, 2005). Similarly, a larger effect was found in the study in Sollentuna with respect to hypertension (Bluhm, Nordling and Berglind, 2001). No such effect was found in the Lübeck study (Hense, Herbold and Honig, 1989; Herbold, Hense and Keil, 1989). The cross-sectional data of the study carried out in Los Angeles on children regarding mean BP indicated some habituation to aircraft noise (Cohen et al., 1980). The longer the children were enrolled in the school, the smaller was the difference in BP between exposed and non-exposed children.

However, the follow-up study suggested that this may also be an effect of attrition (Cohen et al., 1981). The longer the families experienced the noise, the more likely that they moved away from the exposed areas (selection bias). In contradiction to this, BP differences between children exposed and not exposed to road traffic noise increased with school grade (Karsdorf and Klappach, 1968). Intervention studies were conducted with respect to changes in BP and changes in air traffic operation (for example the opening/closing of airports or runways). In the Munich study, a

larger increase in BP was found in children from a noisy area (Evans, Bullinger and Hygge, 1998). Other studies suggested reversible effects on BP when the exposure was lowered (Wölke et al., 1990; Morrell et al., 1998, 2000). In the Tyrol study, significantly lower BP readings were found in subjects who kept the windows closed throughout the night (Lercher and Kofler, 1993, 1996). When the subjects lived close to the highway (within a distance of approximately 500 metres), the prevalence of hypertension was higher in subjects whose bedroom was facing the main road than in those whose bedroom was not facing the main road. The orientation of rooms and window opening was also found to be an effect modifier of the association between road traffic noise and IHD in the Caerphilly and Speedwell studies (Babisch et al., 1999). The relative risk with respect to the noise level was slightly higher in subjects with rooms facing the street and subjects keeping the windows usually open when spending time in the room. A much greater relative risk of hypertension was found in subjects who slept with open bedroom windows in the Spandau Health Survey (Maschke, 2003; Maschke, Wolf and Leitmann, 2003). Hearing impairment was found to be an effect modifier on the association between aircraft noise and hypertension (Rosenlund et al., 2001). Amongst the exposed subjects, a higher risk associated with the noise was only found in subjects without hearing loss.

4.5.12 EXPOSURE DURING THE NIGHT

Unfortunately, epidemiological noise research provides nearly no information regarding the particular impact of noise exposure during the night on cardiovascular health outcomes. The Spandau Health Survey explicitly distinguished between the exposure of the living room (during the day) and the exposure of the bedroom (during the night). There, a slightly higher relative risk of hypertension was found with respect to the traffic noise level during the night (relative risk 1.9 vs. 1.5) compared with the noise level during the day (Maschke, 2003; Maschke, Wolf and Leitmann, 2003). Furthermore, sleeping with open bedroom windows was associated with a large increase in risk. However, due to the small sample size, the confidence intervals were very large. In the drug survey of the Amsterdam aircraft noise studies, a steady increase in purchase of hypnotics (sleeping pills) and sedatives was found (Knipschild and Oudshoorn, 1977). This trend decreased considerably when night flights were largely banned. Such a decrease was not found regarding cardiovascular drugs for which the purchase also increased over time. However, this may partly be due to the fact that atherosclerotic manifestations of high BP were less reversible (in contrast to vasoconstriction, which is more related to acute or semi-acute effects, for instance in children). It was mentioned in the previous section that closing the windows had a *protective effect on BP readings in the Tyrol study (Lercher and Kofler, 1993).* This was only found regarding closing the windows during the night and not during the day. Furthermore, subjects who had switched the bedroom and the living room because of the noise had a significantly lower BP than those who did not do so. The findings are discussed in a broader context of coping strategies (Lercher, 1996). When subjective responses to community noise were considered, higher relative risks of cardiovascular diseases were found for noise-related disturbances of sleep and relaxation, rather than for other disturbances or subjective descriptors of noise exposure, which did not refer to the night-time. This was found in the Caerphilly and Speedwell studies (Babisch, Ising and Gallacher, 2003), the NaRoMI study (Babisch et al., 2005), the Spandau Health Survey (Maschke, Wolf and Leitmann, 2003) and a general population sample of Germany (Bellach et al., 1995). The LARES study (Niemann and Maschke,

2004), in which noise-induced sleep disturbance was assessed, did not show a higher relative risk compared with the general annoyance.

4.5.13 RISK GROUPS

Most epidemiological noise studies looked at the cardiovascular effects of community noise in men. This may simply be due to the fact that the prevalence of cardiovascular diseases in middle-aged subjects is higher in men than in women. Statistical power is an important issue for the design of a study. Furthermore, in noise experiments, physiological reactions controlled by the autonomic nervous system were less pronounced in females than in males (Neus et al., 1980; Ising and Braun, 2000). Improper control for possible differential effects of the intake of sex hormones, including contraceptives, which may prevent or promote adverse (noise) stress effects, may act conservatively on the results (Cairns et al., 1985; Eiff, 1993; Farley et al., 1998). The studies carried out in Lübeck (Hense, Herbold and Honig, 1989; Herbold, Hense and Keil, 1989), Pancevo (Belojevic and Saric-Tanaskovic, 2002), Berlin (Babisch et al., 2005), Stockholm (Rosenlund et al., 2001), a German population sample (Bellach et al., 1995), Bonn (residence time) (Eiff and Neus, 1980; Eiff et al., 1981b) and in Amsterdam (angina pectoris) (Knipschild, 1977a) found higher prevalences of hypertension, IHD and the use of cardiovascular drugs in noise-exposed men than in women. The studies carried out in Bonn (sound level) (Eiff and Neus, 1980; Eiff et al., 1981b), Sollentuna and Amsterdam (heart trouble) (Knipschild, 1977a; Bluhm, Nordling and Berglind, 2001) found the opposite. In the studies carried out in the former Soviet Union, it was reported that noise effects on the cardiovascular system were more pronounced in young and middle-aged subjects (Karagodina et al., 1969). Swedish noise studies (Bluhm, Nordling and Berglind, 2001, 2004) and the LARES study (Niemann and Maschke, 2004) found similar results. The opposite (larger effects in elderly subjects) was reported from the Amsterdam study (Knipschild, 1977a) and the Stockholm study (Rosenlund et al., 2001). The available database on cardiovascular effects of noise in children is poor. No data are available that refer, in particular, to noise and sleep. The quantitative impact of transportation noise on the cardiovascular system is still a matter of research. A quantitative health risk assessment for children cannot be made at the moment.

Based on the available information from noise studies, it must be concluded that children do not appear to be a particular risk group with respect to cardiovascular outcomes, especially BP. This does not mean that the literature does not suggest higher BP readings in children. It only means that the effect in children does not appear to be different than that in adults. However, children may be exposed longer to noise throughout their lifetime than the adults that have been studied. No long-term follow-up studies are known that focus on noise exposure. Most studies on children considered noise in schools rather than noise at home, which implies different mechanisms about how noise could contribute to a rise in BP (raised effort in learning/speech perception vs. disturbed relaxation/sleep). The prospective part of the Caerphilly and Speedwell studies gave a small hint that health status could be a modifying factor. In subjects with prevalent chronic diseases, road traffic noise was associated with a slightly larger increase in the incidence (new cases) of IHD than in subjects without prevalent diseases – when the objective noise level was considered (Babisch et al., 2003). Surprisingly, when annoyance and disturbances due to traffic noise were considered for exposure, the opposite was found. Noise effects were only seen in subjects without prevalent diseases. This was discussed with respect to reporting bias.

4.5.14 RISK EVALUATION

The process of risk assessment (risk evaluation) comprises hazard identification ("Which health outcome is relevant for the exposure?"), exposure assessment ("How many are affected?") and dose–response assessment ("threshold of effect"). This information is summarized in "risk characterization" ("health hazard characterization"). It involves the interpretation of the available evidence from the available data and other scientific disciplines, and is subject to discussion of the uncertainties. These include chance, bias and validity of studies as well as transparency, replicability and comprehensiveness of reviews. As a result of the risk evaluation process, a quantitative estimate about the likelihood that the hazard will affect exposed people will be derived. Usually, attributable risk percentages are calculated (Walter, 1998). This will serve as key information for any kind of risk management including regulatory options (Jasanoff, 1993). The term "adverse" is essential in this context of environmental standard setting. Risk management should ensure that "adverse" health effects do not occur. The fact that an organism responds to noise does not have to be per se "adverse". The severity of a health outcome is an important determinant of the adversity of an effect and implies variable action levels for public health policy (Babisch, 2002, 2004a; Griefahn et al., 2002; Health Council of the Netherlands, 2003). Since considerable parts of the population are exposed to high noise levels (EEA, 2004), noise policy can have a significant impact on public health (Neus and Boikat, 2000). Due to the increasing number of people affected with the decreasing severity of the effect, even small individual risks and less severe health outcomes can be relevant for public health and decision-making. It has been shown that moderate noise exposures implying a small individual risk may cause more noise-induced cases of health-impaired subjects than higher noise exposures. Franssen et al. (2004) pointed out that the number of people suffering from poor health due to aircraft noise is dominated by the larger number of people that is exposed to relatively moderate-to-low noise levels and not by those exposed to high noise levels. This means that more emphasis should be put on the reduction of noise in moderately exposed areas. However, public health policy cannot only consider population attributable risks (risk percentages), but must also consider individual risks (lifetime risk).

In practice, it seems to be reasonable that noise policy should reduce noise, beginning with the highest exposures and ending with the lowest ones. Decision-making will have to find common standards of acceptable risks, which may vary according to the cost–benefit considerations within and between communities and countries. Such practical standards may, however, vary due to economic development and abilities, cost–benefit considerations and priority settings of a community or country. Health quality targets derived from scientific research are usually intended to minimize risks; decision-making in the political process is only partly scientifically based due to economic limitations and concurring interests (Nijland et al., 2003). Different health outcomes or indicators of well-being and quality of life imply different action levels. Environment and health policy must determine acceptable noise standards that consider the whole spectrum from subjective well-being to somatic health (for example annoyance, physiological arousal, health risk). The evidence for a causal relationship between community or transportation noise and cardiovascular risk appears to have increased over recent years due to new studies that accomplish the database.

4.5.15 CONCLUSIONS

The evaluation process used in this paper considered the "necessary" criteria: peer-reviewed publication in an international journal, reasonable quantitative control of possible confounding, objective assessment of exposure and outcome, type of study (analytic vs. descriptive), and dose–response assessment (not only dichotomous "high" vs. "low"). The approach differs from that of an earlier meta-analysis (van Kempen et al., 2002) in that there regression coefficients were calculated for the entire dose–response curve within a single study (for instance the increase in risk per 5 dB(A)), which were then pooled between studies. Since higher exposure categories usually consist of smaller numbers of subjects than the lower categories, regression coefficients across noise levels tend to be influenced by the lower categories. This may lead to an underestimation of the risk in higher noise categories. The approach presented here 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.

Fig. 4.8 and Fig. 4.9
Pooled effect estimates (meta analysis) of descriptive and analytic noise studies of the association between road traffic noise level and the prevalence (left graph) and incidence (right graph), respectively, of myocardial infarction (odds ratio ± 95% confidence interval).

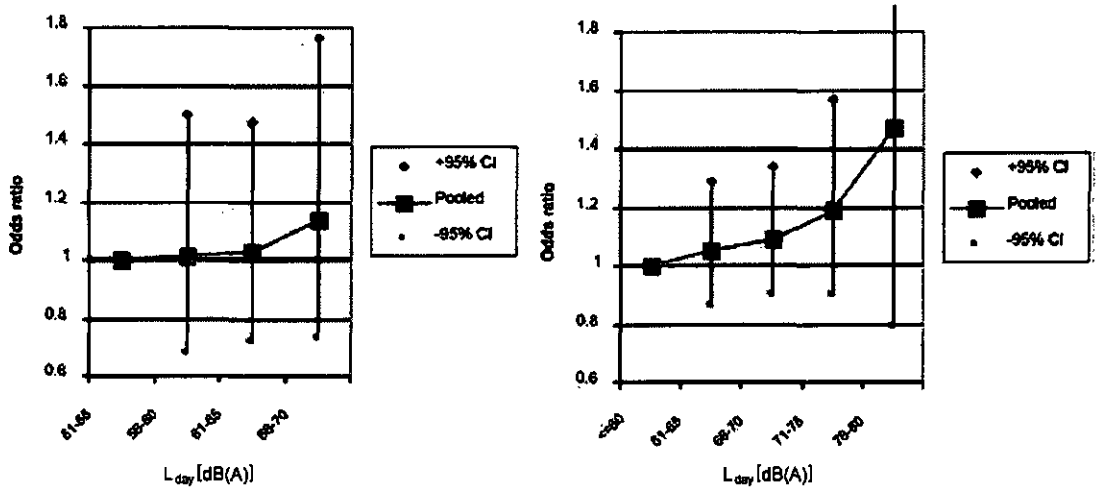


Fig. 4.8 and Fig. 4.9 show the two risk curves for descriptive and analytic studies (Hennekens and Buring, 1987). The graphs show the pooled effect estimates (odds ratios) and the 95% confidence intervals for each noise category. Whereas the cross-sectional studies (Fig. 4.9) cover the sound level range of L_{day} from >50 to 70 dB(A), the cohort and case-control studies (Fig. 4.8) cover the range from ≤60 to 80 dB(A). Both curves together can serve as a basis for a quantitative risk assessment. From Fig. 4.8 it can be seen that below 60 dB(A) for L_{day} no noticeable increase in risk of myocardial infarction is to be detected. Therefore for the time being, L_{day} = 60 dB(A) can be seen as NOAEL (no observed adverse effect level) for the relationship between road traffic noise and myocardial infarction (Babisch, 2002). For noise levels greater than 60 dB(A), the risk of myocardial infarction increases continuously, and is greater than 1.2 for noise levels of 70 dB(A). This can be seen in Fig. 4.9. It should be mentioned that the risk estimates, in general, were found to be higher in subjects that had lived in the exposed areas for a longer time (Babisch et al., 1994, 1999, 2005). This is in accordance with the noise hypothesis and the effects of chronic noise stress (Lercher and Kofler, 1996; Thompson, 1997). However, for the calculation of population attributable risks the figures for the whole population are relevant due to unknown information about residence time.

No particular risk groups could be identified on the basis of epidemiological research on cardiovascular effects of community noise. The assessment of dose-effect relationships sometimes suggested a cut-off level, above which the risk tends to increase. From a biological point of view, one would expect a continuous increase in risk with increasing noise level. However, adaptation, habituation and coping may be reasons for an empirical threshold of effect. Decisions with respect to guideline values usually refer to a quantitative risk assessment of populations (for example population attributable risk percentage). However, prevention strategies – for ethical reasons – should not ignore the individual risks of highly exposed subjects, even if their number may be small.

With respect to night noise exposure, nearly no information is available from epidemiological studies on the cardiovascular effects of long-term noise exposure of the bedroom during the night. Only one study distinguished between the exposures of the bedroom and the living room in the statistical analyses (Maschke, Wolf and Leitmann, 2003). The results suggested slightly higher effect estimates for the prevalence of hypertension with respect to the noise exposure of the bedroom (during the night) compared with the exposure of the living room (during the day). However, the difference was small (odds ration 1.9 vs. 1.5), which means that it still remains an open question whether the night exposure or the overall exposure throughout the whole day is the driving force. The study has some methodological limitations that were addressed in the summary of the major technical report and in a recent advisory report of the Health Council of the Netherlands (2004). They are mainly concerned with the fact that the study population consisted of a selected, predominantly older and health conscious group of persons that might have already suffered from regular health problems (risk group). A few studies that looked at the association between subjective responses to community noise and cardiovascular outcomes suggest a closer relationship with sleep-related annoyance/disturbance reaction rather than with non-sleep-related annoyance/disturbance (Bellach et al., 1995; Babisch et al., 1999, 2005; Maschke, Wolf and Leitmann, 2003; Niemann and Maschke, 2004). Closing the bedroom window or, vice versa, sleeping with the bedroom window open, was associated with a lower or higher risk, respectively (Lercher, 1996). The same was found with respect to changing the bedroom to the living room because of noise. These findings may indicate that night-time noise may be more a determinant of noise-induced cardiovascular effects than daytime exposure. However, daytime activity patterns and expectations of the individuals are much more inhomogeneous than during the night, which tends to dilute the statistical association of true effects with the day noise exposure.

Given the situation that only a few data are available from epidemiological studies with respect to effects on sleep (exposure of the bedroom during the night), there does not seem to be any other way of reasoning than inferring night noise recommendations or guidelines from the results of studies that refer to noise exposure during the daytime period (L_{day}) or the whole day (L_{dn}, L_{24h}). L_{den}, in this context, appears to be a useful noise indicator for decision-making and regulatory purposes. Penalties of 5 dB(A) and 10 dB(A) are usually given to the evening period and the night period, respectively. It can be used for noise mapping and refers normally to the most exposed facade, which incorporates a certain degree of exposure misclassification regarding cause–effect relationships. This weighted indicator was introduced to assess the relationship between sound level and noise annoyance (European Commission, 2002a). However, it may not be adequate for research into (somatic) health-related noise effects. Non-weighted separate exposure indicators, such as L_{day}, $L_{evening}$ or L_{night}, may be more appropriate when assessing physiological responses to the noise. In urban settings, night-time average noise levels (22.00–06.00) for road traffic tend to be approximately 7–10 dB(A) lower than daytime average noise levels – relatively independent (no freeways) of the traffic volume of the street (Utley, 1985; Ullrich, 1998; Evans et al., 2001). In such cases, L_{den} is approximate-

ly 2–3 dB(A) higher than L_{day} (Bite and Bite, 2004). Therefore, in epidemiological studies in which the relative effects of road traffic noise are studied, the sound emission during the daytime can as well be viewed as an approximate relative measure of the overall sound emission including the night. This seems to be further justified because existing noise regulations usually consider a 10 dB(A) difference between the day and the night. The NOAEL of 60 dB(A) for L_{day} corresponds, in this respect, with 50 dB(A) for L_{night}. This approximation can only be made with respect to road traffic noise.

Aircraft noise has been less intensively studied in noise epidemiology. The studies focused on high BP. Dose–response curves were hardly considered. A large European study on the association between aircraft noise and road traffic noise on BP is currently being conducted (Jarup et al., 2003). Regarding aircraft noise – and particularly the ongoing debate on night flight restrictions in the vicinity of busy airports – no other alternative exists at present than to take the myocardial infarction risk curves derived from road traffic noise studies as an approximate for aircraft noise. Since aircraft noise acts on all sides of a building, that is, different to road traffic noise, the suspicion exists that the effects induced by aircraft noise could be greater than those induced by road traffic (Ortscheid and Wende, 2000; Babisch, 2004a). This may be due to the lack of evasive possibilities within the home and the greater annoyance reactions to aircraft noise, which are usually expressed in social surveys (Miedema and Vos, 1998). More research is needed regarding the association between aircraft noise and cardiovascular end points.

This section is clearly focused on ill health as an outcome of the adverse effect of noise. A common dose-effect curve for the relationship between road traffic noise (outdoors) and the risk of myocardial infarction was developed. This curve can be used for a quantitative risk assessment and the calculation of attributable cases in a community. However, decisions regarding limit values have to be made within the spectrum between discomfort (annoyance) and ill health (disease) (Lindström, 1992; Babisch, 2002). Whereas quality targets at the lower end of the effects scale may be more flexible, quality targets at the upper end may be more obligatory. For example, for ethical reasons (equality principle) it does not seem to be justified if (ill) health-based limit values are varied according to the type of living area as expressed in land development plans (for example residential, mixed or commercial).

4.6 INSOMNIA

A group of Japanese researchers carried out a questionnaire-based survey of 3600 adult Japanese women (aged between 20 and 80) to gather information about the factors that contribute to insomnia (Kageyama et al., 1997). Some 11% of subjects were found to be affected by insomnia (as defined on the basis of WHO's International Statistical Classification and Related Health Problems, 10th revision – ICD10). Analysis of the survey data took account of various distorting variables, such as age, number of (small) children in the family, social status, receipt of medical treatment, regularity of bedtimes, apnoea-like problems and serious unpleasant experiences in the six months prior to completing the questionnaire. When the percentage of insomniacs in each of the three areas with the highest exposures was compared with the percentage in the low-exposure areas, the ratios worked out at, respectively, 1.4 (2100 vehicles per hour, L_{night} of around 65 dB(A)), 2.1 (2400 vehicles per hour, L_{night} of around 67 dB(A)) and 2.8 (6000 vehicles per hour, L_{night} of around 70 dB(A)). The most frequently reported problem was difficulty getting to sleep.

Research into the effects of exposure to air and road traffic noise has shown that increases in night-time noise exposure or in noise exposure during the sleep latency period have a statistically significant adverse impact on subjects' ability to get off to sleep and on sleep inception periods.

4.7 EFFECTS ON PERFORMANCE

4.7.1 COGNITION AND SWS

Jan Born and co-workers at the University of Lübeck (Wagner, Gais and Born, 2001; Benedict et al., 2004; Born and Wagner, 2004; Gais and Born, 2004; Drosopoulos, Wagner and Born, 2005) have reported interesting research and put forward intriguing hypotheses on the relation between noise exposure, sleep loss and subsequent cognitive performance. They conclude that declarative memory benefits mainly from sleep periods dominated by SWS, while there is no consistent benefit for this memory from periods rich in REM sleep. This points to the importance of SWS for declarative memory.

Since sleep in the early night is dominated by SWS, in contrast to late night when REM sleep dominates, this would imply that noise in the early night, for example aircraft noise before midnight, would be particularly damaging to memory and related cognitive functions. However, this implication has not yet been explicitly tested. That is, there seems to be a certain risk for impoverished memory due to noise in the early night, but there is as yet no graded quantification about whether ordinary pre-midnight noise levels around large airports are sufficient to make a difference to SWS. We also lack graded quantification about the relationship between impoverished SWS and the resulting effect on different aspects of declarative memory.

Thus, in terms of Fig. 1.1 we have evidence for the arrow marked (b), but we do not have enough information to say whether the strength of arrow (a) is sufficient to cause reduced SWS in field settings.

Furthermore, since children's memory systems pass through developmental changes and are not structured in the same way as for adults, it would be interesting to know to what extent the Born group results are also valid for children, and whether the depth of children's sleep counteracts or enhances SWS dominance in the early night.

4.7.2 COMPARING DAYTIME AND NIGHT-TIME NOISE EXPOSURE

As implied by Fig. 1.1, the relation between noise exposure and resulting effects on cognition should be analysed somewhat differently depending on whether the noise exposure takes place during the day or night. Analysing the cognitive effects of daytime noise exposure is fairly straightforward. For night-time noise exposure, however, any effects on cognition can either be a more or less direct effect of the noise exposure, or an indirect effect mediated by reduced sleep or sleep quality.

Also, comparing, for example, memory and learning functions when exposed to

night-time noise, in contrast to daytime noise, shifts the focus of analysis away from encoding (in memory) or acquisition (in learning) while experiencing noise, to a focus on storing the material to be remembered or learnt while asleep (compare to daytime noise effects on cognition as reported by Hygge, Evans and Bullinger, 2002; Stansfeld et al., 2005). Thus, assuming that people are mainly asleep at night, all cognitive work that relies on the intake of information, listening or reading is not relevant. In all, this suggests that studies of daytime noise levels cannot be used much to give rough estimates of the effects of night-time exposure.

4.7.3 COMPARING CHILDREN AND ADULTS

How far can effects of daytime noise levels on children be generalized to give a rough estimate of the effects on adults? Are children more sensitive? Judging from earlier daytime studies of children and adults doing the same cognitive tasks while exposed to noise, children are not more sensitive than adults to noise (Boman, Enmarker and Hygge, 2004), but they perform at a lower level than the adults both in noisy and in quiet environments. Thus, it could be said that children are not more vulnerable to (daytime) noise in relation to cognitive performance, but since so much more cognitive work is expected from children while in school, their learning environment and their cognitive tasks can be said to be more noise vulnerable than corresponding environments for adults.

4.7.4 NOISE AND AFTER-EFFECTS

An argument can be made for noise as a stressor leading to reduced motivation (Glass and Singer, 1972), which in turn may act as a mediator of impaired cognitive performance. Along this line of reasoning, night-time noise may be more potent in inducing reduced motivation than daytime noise, but for the time being this is only a conjecture and has not been tested.

4.7.4.1 The role of restoration

Noise can be viewed both as a source of stressful demands and as a constraint on restoration. Noise levels and noise sources that are not by themselves particularly demanding during the waking hours of the day, may nevertheless be quite effective in blocking and constraining when they appear in periods meant to be restorative, such as sleep (Hartig, 2004). To what extent this idea is applicable to night-time noise exposure has not yet been explored.

4.7.4.2 Noise and communication

Some of the difficulties with children's responses to noise are related to problems in speech perception. A metric that weights night-time exposure more heavily is, in fact, less useful since children's auditory processing with parents and teachers is obviously more critical during waking hours.

4.8 EFFECTS ON PSYCHIC DISORDERS

Noise exposure at night may be more disturbing than daytime noise because it interferes with rest and sleep at a time when people want to relax. It seems plausible that night-time noise might have a particular effect on mental health. However, there is lit-

tle direct research into night-time noise and mental health and it is first necessary to consider the evidence for environmental noise and mental health in general. The association between noise and mental health has been examined using a variety of outcomes including (at the simplest level) individual symptoms, as well as psychiatric hospital admission rates, use of health services and psychotropic medication, and community surveys.

4.8.1 TRANSPORTATION NOISE AND MENTAL HEALTH

Sources of transportation noise that have been studied in relation to mental health include road traffic noise and aircraft noise. Studies relating to each type of noise will be considered in turn.

4.8.1.1 Road traffic noise

The association between road traffic noise exposure and psychological distress has been studied in the small town of Caerphilly, South Wales. In the cross-sectional results, no association was found between the initial level of road traffic noise based on traffic noise maps, in terms of L_{eq} referring to the period 06.00–22.00, and minor psychological distress, measured by the General Health Questionnaire (GHQ), a screening questionnaire for depression and anxiety, even after adjustment for sociodemographic factors (Stansfeld et al, 1993). In longitudinal analyses in the Caerphilly Study, no association was found between road traffic noise and psychological distress, even after adjustment for sociodemographic factors and baseline psychological distress, although there was a small non-linear association of noise with increased anxiety scores (Stansfeld et al, 1996).

The disadvantage of the Caerphilly study is that it relied on one location with not very high levels of traffic noise. In a secondary analysis of a large British road traffic noise study, which took into account multiple noise exposure sites, the noise level in dB(A) exceeded for 10% of the time was weakly associated with a mental health symptoms scale of five items adjusting for age, sex, income and length of residence (Halpern, 1995). Weaker associations between traffic density and the mental health symptoms scale may relate to the skewed distribution of this traffic density variable. It seemed that traffic noise was more important than traffic flow. The scale used included some clear mental health items but also some that were less obviously related to mental health. It may be questioned whether the reported association between noise level and mental health symptoms was actually due to noise exposure; adjustment for the amount of "noise heard" reduced the association very little, suggesting no causal association with noise, but it is likely that there was a good deal of error in the measurement of this variable, reducing its validity.

It may be that the peak noise level is a better indicator of environmental noise heard indoors than noise measures averaged over time and that peak levels are a crucial indicator for mental health. Furthermore, in a road traffic noise study in Belgrade, 253 residents exposed to road traffic noise levels of >65dB(A), with high levels both day and night (L_{eq} 76.5 in the day, 69.5 at night in the noise-exposed area), experienced significantly more fatigue, depression, nervousness and headaches, compared to residents exposed to <55dB(A) (Belojevic and Jakovljevic, 1997). Sleep quality was also found to be worse among the inhabitants of noisy streets, compared to inhabitants of quiet streets, and those living in noisy streets had more difficulties falling asleep, more night awakenings and more pronounced tiredness after sleep. However, there were no differences in time taken to fall asleep or to go back to sleep,

duration of sleep or consumption of sleeping pills between noise-exposed and non-exposed residents. A great methodological advantage of this study was that the high and low noise exposure areas were homogenous for age, sex, employment and subjective noise sensitivity. A community study in 366 Japanese women suggests that road traffic noise only has effects on depression, fatigue and irritability above a threshold of 70 dB(A) (Yoshida et al., 1997). However, it is difficult to be confident of the results of these analyses as they were unadjusted for age or social deprivation. Milder psychological states such as health functioning and well-being have also been examined in the first stage of an intervention study on the effect of introducing a bypass to relieve traffic congestion in a small town in North Wales (Stansfeld, Haines and Brown, 2000). Health functioning was measured by the SF-36 General Health Survey (Ware and Sherbourne, 1992), including dimensions of general health status, physical functioning, general mental health and social functioning. Ninety-eight respondents were studied who lived on a busy high street with traffic noise levels varying between 72 and 75 dBA outdoor L_{eq}. These respondents were compared with 239 control subjects living in adjacent quieter streets (noise level 55–63 dB(A) outdoor L_{eq}). Although subjects were well-matched on age, sex, housing insulation, car ownership and employment status, they were not so well-matched on proportion of manual workers, household crowding, deprivation and home ownership. There was no evidence that respondents exposed to higher levels of road traffic noise had worse health functioning than those exposed to lower levels of the noise, adjusting for levels of deprivation.

Another method of assessing mental health effects related to noise exposure is to use an indirect indicator such as medication use. In five rural Austrian communities exposed to road traffic noise, noise levels above 55 dB(A), including increasing night-time exposure to noise from trucks, were associated with increased risk of taking sleeping tablets (OR = 2.22 [CI: 1.13–4.38]) and overall prescriptions (OR = 3.65 [CI: 2.13–6.26]) relative to road traffic noise exposure less than 55 dB(A) (Lercher, 1996). This suggested effects at fairly low noise levels. In this case mental ill health may be secondary to sleep disturbance, which is likely to occur at lower nocturnal noise levels than mental health symptoms resulting from daytime noise exposure. As this occurred in a rural setting where road traffic was the predominant source of noise it would be interesting to replicate these findings in other settings.

4.8.1.2 Road traffic noise and mental health in children

Noise exposure and mental health has also been studied in children where child self-reported mental health on a standard scale and teacher ratings of classroom adjustment in response to motorway, road and rail noise were measured in a large sample of 8–11-year-old Austrian primary school children and in a second stage sample of extreme noise-exposed groups. Noise exposure was significantly associated with classroom adjustment scores but, intriguingly, child self-reported mental ill health was only impaired in noisy settings for children of low birth weight and preterm birth (Lercher et al., 2002).

4.8.1.3 Aircraft noise

Community surveys have found that high percentages of people reported "headaches", "restless nights", and "being tense and edgy" in high aircraft noise areas (Kokokusha, 1973; Finke et al., 1974; Öhrström, 1989). An explicit link between aircraft noise and symptoms emerging in such studies raises the possibility of a bias towards over-reporting of symptoms (Barker and Tarnopolsky, 1978). Notably, a study around three Swiss airports (Grandjean et al., 1973), did not mention that it was related to aircraft noise and did not find any association between the

level of exposure to aircraft noise and symptoms. In the West London Survey, "tinnitus", "burns, cuts and minor accidents", "ear problems" and "skin troubles" were all more common in areas of high noise exposure (Tarnopolsky, Watkins and Hand, 1980). Acute symptoms such as "depression", "irritability", "difficulty getting off to sleep", "night waking", "skin troubles", "swollen ankles" and "burns, cuts and minor accidents" were particularly common in high noise areas. However, apart from "ear problems" and "tinnitus", 20 out of 23 chronic symptoms were more common in low noise environments. Symptoms did not increase with increasing levels of noise. This is possibly related both to more social disadvantage and associated ill health among residents in low aircraft noise exposure areas and the possible unwillingness of chronically unhealthy individuals to move into potentially stressful high noise exposure areas. Nevertheless, it would not exclude an effect of noise in causing some acute psychological symptoms. As the majority of aircraft noise exposure is during the day, daytime exposure is likely to have greater effects than nighttime exposure. Many of the effects of noise in industrial and teaching settings may be related primarily to disturbances in communication.

4.8.2 NOISE EXPOSURE AND MENTAL HOSPITAL ADMISSION RATES

Much of the concern with the possible effects of noise on mental health began with the study of admissions to psychiatric hospitals from noisy areas. Early studies found associations between the level of aircraft noise and psychiatric hospital admissions, both in London (Abey Wickrama et al., 1969) and Los Angeles (Meecham and Smith, 1977). These results have been criticized on methodological grounds (Chowns, 1970; Frerichs, Beeman and Coulson, 1980) and a replication study by Gattoni and Tarnopolsky (1973) failed to confirm these findings. Jenkins et al., (1979) found that age-standardized admission rates to a London psychiatric hospital over four years were higher as the level of noise of an area decreased, but lower noise areas were also central urban districts, where high admission rates would be expected. In a further extensive study of three hospitals (Jenkins, Tarnopolsky and Hand, 1981), high aircraft noise was associated with higher admission rates in two hospitals, but in all three of them, admission rates seemed to follow non-noise factors more closely; the effect of noise, if any, could only be moderating that of other causal variables but not overriding them. Kryter (1990), in a re-analysis of the data, found "a more consistently positive relation between level of exposure to aircraft noise and admissions rates". Undoubtedly, the route to hospital admission is influenced by many psychosocial variables that are more potent than exposure to noise. Therefore, whether or not noise causes psychiatric disorder is more suitably answered by studying a community sample.

4.8.3 NOISE EXPOSURE AND PSYCHIATRIC MORBIDITY IN THE COMMUNITY

In a community pilot study carried out in West London, Tarnopolsky et al. (1978) found no association between aircraft noise exposure and either GHQ scores (Goldberg, 1972) (dichotomized 4/5, low scorers/high scorers) or estimated psychiatric cases (Goldberg et al., 1970). This was the case even when exposure to road traffic noise was controlled, except in three subgroups: persons "aged 15–44 of high education" (41%, 14% $p<0.05$), "women aged 15–44" (30%, 13% n.s.), and those in "professional or managerial occupations". The authors expressed the guarded opinion that

noise might have an effect in causing morbidity within certain vulnerable subgroups. In the subsequent West London Survey of Psychiatric Morbidity (Tarnopolsky, Morton-Williams and Barker, 1980), 5885 adults were randomly selected from within four aircraft noise zones, according to the Noise and Number Index. No overall relationship was found between aircraft noise and the prevalence of psychiatric morbidity either for GHQ scores or for estimated numbers of psychiatric cases, using various indices of noise exposure. However, there was an association between noise and psychiatric morbidity in two subgroups: "finished full-time education at age 19 years +", and "professionals". These two categories, which had a strong association with each other, were combined and then showed a significant association between noise and psychiatric morbidity ($X2 = 8.18$, df 3 $p<0.05$), but only for the proportion of high GHQ scorers. Tarnopolsky, Morton-Williams and Barker (1980) concluded that their results "show so far that noise per se in the community at large, does not seem to be a frequent, severe, pathogenic factor in causing mental illness but that it is associated with symptomatic response in selected subgroups of the population".

More recent studies have examined the effects of higher levels of military aircraft noise. Exposure to higher levels of military aircraft noise around the busy Kadena military airport in Japan was related in an exposure–effect association to depressiveness and nervousness measured by questionnaire using the Todai Health Index, based on the Cornell Medical Index (Ito et al., 1994; Hiramatsu et al., 1997). Mental health subscales included in this study measured depressiveness, nervousness, neurosis, and mental instability. Noise level was expressed as WECPNL (the power average of the maximum perceived noise exposure level in dB(A)) from 75–79, 80–84, 85–89, 90–94 and over 95). In unadjusted analyses, statistically significant differences were found in scores of depressiveness, nervousness and neurosis between the non-noise exposed control group and the pooled group exposed to 75–95 WECPNL. In multivariate analysis adjusting for age, sex, marital status, type of house and length of residence, noise exposure greater than 95 WECPNL was associated with higher scores on depressiveness and neurosis (Hiramatsu et al., 1997). Clear exposure–effect relationships were not found between scale scores and noise exposure, as expressed in five unit steps. However, using more broadly defined groups, an exposure–effect association was evident. This highlighted differences between the highest noise exposure group and lower exposure groups and indicated a threshold effect rather than a linear relationship – that mental health effects are more likely to be found at higher noise levels. In general, psychological rather than somatic symptoms were more related to noise in this study. Further analyses of the Japanese studies suggest that high levels of military aircraft noise may have effects on mental health. In a cross-sectional study of 5963 inhabitants around two air bases in Okinawa, those exposed to noise levels of L_{dn} 70 or above had higher rates of "mental instability" and depressiveness (Hiramatsu et al., 2000). Those who were more annoyed showed a higher risk of mental or somatic symptoms. A further survey using similar methodology on 6486 respondents found exposure–effect associations between aircraft noise exposure, nervousness and mental health (Miyakita et al., 1998). These are important studies because of the opportunity to examine the effect of high noise-exposure levels and the probability that vulnerable people migrating out of noisy areas and thus biasing the sample was small.

The use of health services has also been taken as a measure of the relationship between noise and psychiatric disorder. Grandjean et al. (1973) reported that the proportion of the Swiss population taking drugs was higher in areas with high levels of aircraft noise and Knipschild and Oudshoorn (1977) found that the purchase of sleeping pills, antacids, sedatives and antihypertensive drugs all increased in a vil-

lage newly exposed to aircraft noise, but not in a "control" village where the noise level remained unchanged. In both studies, there was also an association between the rate of contact with general practitioners and level of noise exposure. In the Heathrow study (Watkins, Tarnopolsky and Jenkins, 1981), various health care indicators were used – use of drugs, particularly psychiatric or self-prescribed, visits to the GP, attendance at hospital, and contact with various community services – but none of these showed any clear trend in relation to levels of noise. A recent study found that the use of sleeping tablets and sedatives was elevated with increasing night-time noise exposure, especially in the elderly (Passchier-Vermeer et al., 2002). This has been judged to be "sufficient" evidence of a noise effect (Health Council of the Netherlands, 2004).

4.8.4 AIRCRAFT NOISE EXPOSURE AND MENTAL HEALTH IN CHILDREN

Poustka, Eckermann and Schmeck (1992) studied the psychiatric and psychosomatic health of 1636 children aged 4–16 in two geographical regions that differed according to the noise made by jet fighters frequently exercising at low altitude. Psychological and neurological outcomes were not related to noise exposure. They found that associations between noise exposure and depression and anxiety could be demonstrated, but only beneath the threshold of clinical significance. These results are less convincing because the areas differed socioeconomically and the results were not adjusted for these factors and also because of lack of precision of the measures of noise exposure. However, in Munich, children living in areas exposed to high aircraft noise had lower levels of psychological well-being than children living in quieter environments (Evans, Hygge and Bullinger, 1995). The longitudinal data from around Munich showed that after the inauguration of the new airport, the newly noise-exposed communities demonstrated a significant decline in self-reported quality of life measured on the Kindl scale, after being exposed to the increased aircraft noise for 18 months (third wave of testing), compared with a control sample (Evans, Bullinger and Hygge, 1998). Impairment of "quality of life" is a less severe disturbance than impairment of mental health. Further studies have examined the effects of noise on child psychiatric disorders.

Chronic aircraft noise exposure was not associated with anxiety and depression (measured with psychometrically valid scales), after adjustment for socioeconomic factors, in the Schools Health and Environment Study around Heathrow Airport (Haines et al., 2001a). In a further larger study of children's health around Heathrow Airport – the West London Schools Study (Haines et al., 2001b) – an association was found between aircraft noise exposure level and increased hyperactivity scores on the hyperactivity subscale of the Strength and Difficulties Questionnaire (Goodman, 1997). These studies suggest that noise influences child mental health in terms of hyperactivity and that it may affect child stress responses and sense of well-being.

4.8.5 NEIGHBOURHOOD NOISE AND MENTAL HEALTH

Noise from neighbours is the commonest source of noise complaints to local authorities in the United Kingdom (Chartered Institute of Environmental Health, 1999). Noise which is continuous, apparently indefinite, of uncertain cause or source, emotive or frightening or apparently due to thoughtlessness or lack of consideration is most likely to elicit an adverse reaction (Grimwood, 1993). In the 1991 BRE survey,

people most objected to barking dogs, banging doors, noise from radio, television, or hi-fi and human voices (Grimwood, 1993). In this survey, two types of emotional response to noise were observed: outwardly directed aggression, characterized by feelings of annoyance, aggravation, bitterness and anger towards the source of the noise, and a more emotional response of tension, anxiety and feelings of pressure. These responses are reminiscent of the distinction between internalizing and externalizing disorders. Whether noise from neighbours can induce psychiatric disorder has been little studied in community research, but this is an area that deserves further study (Stansfeld, Haines and Brown, 2000).

Undoubtedly, prolonged exposure to noise can be very upsetting, intrusive and interfering for sleep and everyday activities. In poorly built dwellings, especially apartments, even low intensity noises may be clearly audible through walls, floors, or ceilings (Raw and Oseland, 1991). In this situation, noise is destructive of privacy, especially for those living alone, and may be associated with perceptions of threat or increase a sense of isolation. This may be especially the case among people who are chronically anxious and likely to complain of sensitivity to noise; prolonged noise exposure may make them more anxious and unhappy. Often, this leads to arguments with neighbours, leading to a breakdown of neighbourly relationships and further isolation which may well in itself have a bad effect on mental health. Occasionally, this may be a sign of feelings of persecution associated with psychotic illness in which noise exposure is just an external trigger of an internally generated condition.

4.8.6 MECHANISMS FOR CAUSAL LINKS BETWEEN NOISE AND MENTAL HEALTH

What might the mechanism be for the effects of noise on mental health? One way to approach this is through the effects of noise on cognitive performance where the laboratory evidence of effects is fairly robust (Smith and Broadbent, 1992). Effects of noise on mental health might be expected because there is evidence that noise impairs other aspects of human functioning, such as performance (Loeb, 1986) and sleep, that are important in maintaining normal functioning, and that noise causes adverse emotional reactions such as annoyance. In general, it seems that noise exposure increases arousal, and decreases attention through distraction (Broadbent, 1953), increases the need for focusing attention to cut out irrelevant stimuli (Cohen and Spacapan, 1978), as well as altering choice of task strategy (Smith and Broadbent, 1981). Even relatively low levels of noise may have subtle ill effects, and in this respect, the state of the person at the time of performance may be as important as the noise itself (Broadbent, 1983). Individuals' perception of their degree of control over noise may also influence whether it impairs memory (Willner and Neiva, 1986) while perception of lack of control over environmental conditions may be an important mediator of health effects.

Additionally, noise may also affect social performance as: (1) a stressor causing unwanted aversive changes in affective state; (2) by masking speech and impairing communication; and (3) by distracting attention from relevant cues in the immediate social environment (Jones, Chapman and Auburn, 1981). It may be that people whose performance strategies are already limited for other reasons (for instance through high anxiety) and who are faced with multiple tasks may be more vulnerable to the masking and distracting effects of noise.

The mechanism for the effects of noise on health is generally conceptualized as fit-

ting the stress–diathesis model, in which noise exposure increases arousal, and chronic exposure leads to chronic physiological change and subsequent health effects. It is not clear, however, whether this model is appropriate for mental health effects. A more sophisticated model (Biesiot, Pulles and Stewart, 1989; Passchier-Vermeer, 1993) incorporates the interaction between the person and their environment. In this model, the person readjusts their behaviour in noisy conditions to reduce exposure. An important addition is the inclusion of the appraisal of noise (in terms of danger, loss of quality, meaning of the noise, challenges for environmental control, etc.) and coping (the ability to alter behaviour to deal with the stressor). This model emphasizes that dealing with noise is an active not a passive process.

4.8.7 HABITUATION TO NOISE AND MENTAL HEALTH

It is likely that mental health effects arise from persistent exposure to noise over a long period of time. But do people habituate or adapt to noise over time? In some studies people do seem to adapt to noise and no longer notice noise that they are frequently exposed to. On the other hand, in some studies of annoyance there seems to be little evidence of adaptation (Cohen and Weinstein, 1981). It may be that, as in physiological studies, a failure of adaptation occurs if the stimulus is novel, salient or implies threat. The development of mental health symptoms implies a failure to habituate to noise, or at least to adapt to noise. In some studies control over noise or active coping with noise rather than passive emotion-focused coping is related to lower levels of symptom (van Kamp, 1990). Habituation has not been formally studied in relation to noise and mental health.

4.8.8 RISK GROUPS FOR MENTAL HEALTH EFFECTS FROM NOISE

One way to look at susceptibility to noise is to think about groups in the population who may be more susceptible to noise, for instance people with existing physical or mental illness tend to be more highly annoyed by noise and potentially could be vulnerable to mental health effects. Similarly, people with hearing impairment may be vulnerable to communication difficulties in noisy environments that could increase the risk of mental health symptoms. People who report that they are sensitive to noise tend to be more prone to noise annoyance and may be more at risk for common mental disorders (Stansfeld et al., 2002).

4.8.9 POPULATION GROUPS AT RISK FOR MENTAL HEALTH EFFECTS FROM NOISE

There is some evidence that children are more vulnerable to the mental health effects of noise than adults in terms of prematurity, low birth weight and through scoring higher on hyperactivity. There is no consistent evidence of age, social class, ethnic or gender differences in susceptibility to mental health effects from environmental noise.

4.8.10 NOISE SENSITIVITY

Noise sensitivity, based on attitudes to noise in general (Anderson, 1971; Stansfeld, 1992), is an intervening variable which explains much of the variance between exposure and individual annoyance responses (Weinstein, 1978; Langdon, Buller and

Scholes, 1981; Fields, 1993). Individuals who are noise-sensitive are also likely to be sensitive to other aspects of the environment (Broadbent, 1972; Weinstein, 1978; Thomas and Jones, 1982; Stansfeld et al., 1985a). This raises the question as to whether noise-sensitive individuals are simply those who complain more about their environment. Certainly, there is an association between noise sensitivity and neuroticism (Thomas and Jones, 1982; Öhrström, Bjorkman and Rylander, 1988; Jelinkova, 1988; Belojevic and Jakovljevic, 1997; Smith, 2003), although it has not been found in all studies (Broadbent, 1972). On the other hand, Weinstein (1980) hypothesized that noise sensitivity is part of a critical/uncritical dimension, showing the same association as noise sensitivity to measures of noise, privacy, air pollution and neighbourhood reactions. He suggested that the most critical subjects, including noise-sensitive people are not uniformly negative about their environment, but more discriminating than the uncritical group, who comment uniformly on their environment.

Noise sensitivity has also been related to current psychiatric disorder (Bennett, 1945; Tarnopolsky, Morton-Williams and Barker, 1980; Iwata, 1984). Stansfeld et al. (1985) found that high noise sensitivity was particularly associated with phobic disorders and neurotic depression, measured by the Present State Examination (Wing, Cooper and Sartorius, 1974). Similar to this association with phobic symptoms, noise sensitivity has also been linked to a coping style based on avoidance, which may have adverse health consequences (Pulles, Biesiot and Stewart, 1988) and a tendency to report health complaints rather than take a more active coping approach to noise (Lercher and Kofler, 1996). Noise sensitivity may be partly secondary to psychiatric disorder: depressed patients followed over four months became less noise-sensitive as they recovered (Stansfeld, 1992). These "subjective" psychological measurements were complemented by an "objective" psychophysiological laboratory investigation of reactions to noise in a subsample of depressed patients. Noise-sensitive people tended to have higher levels of tonic physiological arousal, more phobic and defence/startle responses and slower habituation to noise (Stansfeld, 1992). Thus, noise-sensitive people attend more to noises, discriminate more between noises, find noises more threatening and out of their control, and adapt to noises more slowly than people who are less sensitive. Through its association with greater perception of environmental threat and its links with negative affectivity and physiological arousal, noise sensitivity may be an indicator of vulnerability to minor psychiatric disorder, although not necessarily psychiatric disorder caused by noise (Stansfeld, 1992).

In analysis of a subset of noise-sensitive women, compared to less sensitive women in the West London survey, there was no evidence that aircraft noise exposure predicted psychiatric disorder in the sensitive women (Stansfeld et al., 1985). In the Caerphilly study, noise sensitivity predicted psychological distress at follow-up after adjusting for baseline psychological distress, but did not interact with the noise level, suggesting that noise sensitivity does not specifically moderate the effect of noise on psychological distress (Stansfeld et al., 1993). However, in further analyses, a statistically significant association between road traffic noise exposure and psychological distress, measured by the General Health Questionnaire (GHQ), was found in noise-sensitive men, that was not found in men of low noise sensitivity (Stansfeld et al., 2002). In the original analyses, after adjusting for trait anxiety at baseline, the effect of noise sensitivity was no longer statistically significant. This suggests that much of the association between noise sensitivity and psychological distress may be accounted for by the confounding association with trait anxiety. Constitutionally anxious people may be both more aware of threatening aspects of their environment and more prone to future psychiatric disorder. It seems possible that these traits might be linked.

In a United Kingdom community study, associations were examined between noise exposure, noise sensitivity, subjective symptoms and sleep disturbance in a random sample of 543 adults (Smith, 2003). Perceived noise exposure was related to subjective health, but this association became non-significant after adjustment for negative affectivity. In a similar way, adjustment for negative affectivity eliminated the association between noise sensitivity and subjective health. Thus, it was suggested that noise sensitivity was merely a proxy measure of negative affectivity or neuroticism. However, although this means that noise sensitivity is not specific to noise, the more recent analyses suggest that high levels of trait anxiety or neuroticism may be an indicator of vulnerability to noise effects and could put people at risk of adverse psychological effects from noise, even if they do not increase the risk of physical ill health.

4.8.11 MENTAL HEALTH CONSEQUENCES OF INSOMNIA

Transient insomnia is usually accompanied by reports of daytime sleepiness and performance impairment the next day. Chronic insomnia is generally associated with poorer emotional and physical health. Several large-scale epidemiological studies of the general adult population have shown that between one third and one half of people who complain of chronic insomnia are also diagnosable with primary psychiatric disorders, mostly anxiety and mood disorders. Mellinger, Balter and Uhlenhuth (1985) found that 17% of adults reported "a lot" of trouble falling asleep or staying asleep over the past year; 47% of them had high levels of psychological distress, with symptom complexes suggestive of depression and anxiety disorders. In contrast, only 11% of individuals with no history of insomnia showed elevated levels of psychiatric symptoms. In a survey of almost 8000 individuals, Ford and Kamerow (1989) reported that 10% had suffered from significant insomnia for at least a two-week period during the previous six months; 40% of the insomniacs met criteria for psychiatric disorders, with the majority being anxiety disorders and depression; only 16% of those with no sleep complaints had psychiatric illness.

Breslau et al. (1996) found a strong correlation between lifetime prevalence of sleep problems and psychiatric disorders, with anxiety, depression, and substance abuse disorders being the most common. Similar results have been found by Vollrath, Wicki and Angst (1989), Chang et al. (1997) and Dryman and Eaton (1991). In a large-scale European population-based study (Ohayon and Roth, 2003), it was found that insomnia more often precedes rather than follows incident cases of mood disorders.

Insomniacs not only have higher rates of psychiatric disorders, but they also have increased rates of various kinds of psychological symptoms: patients with insomnia reported increased psychological stress and/or decreased ability to cope with stress according to surveys of the American (Roth and Ancoli-Israel, 1999) and Japanese (Kim et al., 2000) population. Almost 80% of insomniacs had a significant increase on one or more clinical scales on the Minnesota Multiphasic Personality Inventory (MMPI) (Kalogjera-Sackellares and Cartwright, 1997). Even people whose insomnia was due to identified medical factors showed elevation on the MMPI, suggesting a possible causal relationship or specific association between insomnia and psychiatric symptomatology. Compared to good sleepers, severe insomniacs reported more medical problems, had more GP office visits, were hospitalized twice as often and used more medication. Severe insomniacs had a higher rate of absenteeism, missing work twice as often as did good sleepers. They also had more problems at work (including decreased concentration, difficulty performing duties and more work-related accidents) (Leger et al., 2002).

4.8.12 INSOMNIA AS A MENTAL HEALTH SYMPTOM

Insomnia is a symptom of many psychiatric disorders, especially depression and anxiety. In studies of depressed patients compared to control subjects, there was prolonged latency to sleep, increased wakefulness during sleep, early morning wakening, decreased sleep efficiency and reduced total sleep time. There is also evidence that insomnia may be a risk factor for developing depression (Riemann, Berger and Voderholzer, 2001; Roberts, Roberts and Chen, 2002). This raises the question as to whether prolonged noise exposure leading to insomnia provokes the onset of depression in susceptible people? This seems theoretically possible, but there is little evidence to support it. In a longitudinal study of adolescents, it was the other way round – that depressive symptoms preceded the onset of insomnia (Patton et al, 2000). Delayed sleep latency in children has been linked to increased externalizing symptoms including aggressive behaviour, and impaired attention and social problems (Aronen et al., 2000). In this cross-sectional study, the direction of association was uncertain, but it seems most plausible that the sleep disturbance is a feature of the behavioural disturbance rather than a cause of it. Three criteria have been suggested for sleep disturbance to be environmentally determined: (1) the sleep problem is temporally associated with the introduction of a physically measurable stimulus or definable set of environmental circumstances; (2) the physical rather than the psychological properties of the environmental factors are the critical causative elements; and (3) removal of the responsible factors results in an immediate or gradual return to normal sleep and wakefulness (Kraenz et al., 2004). Most studies do not fulfil these criteria. In a German school-based study of 5–6-year-old children, sleep disturbance by noise, largely from road traffic, was reported "sometimes" in 10% by parents of children and 2% "often". Children's reports were slightly higher: "sometimes" in 12% and 3% "often" (Kraenz et al., 2004). Further longitudinal research is needed to ascertain whether noise-induced insomnia leads on to overt psychiatric disorder.

In summary, population as well as clinic-based studies have demonstrated a high rate of psychiatric morbidities in patients with chronic insomnia. It has traditionally been assumed that insomnia is secondary to the psychiatric disorders; however, it is possible that in some cases the insomnia preceded the psychiatric disorder.

4.8.13 DEPRESSIVE EPISODE AND ANXIETY DISORDERS

A mild depressive episode is diagnosed by clinical interview. The criteria for a mild depressive episode include two or more symptoms of depressed mood, loss of interest or fatigue lasting at least two weeks, with two or three symptoms such as reduced concentration, reduced self-esteem, ideas of guilt, pessimism about the future, suicidal ideas or acts, disturbed sleep, diminished appetite and social impairment, and fewer than four symptoms including lack of normal pleasure/interest, loss of normal emotional reactivity, waking =>2 hours early, loss of libido, diurnal variation in mood, diminished appetite, loss of =>5% body weight, psychomotor agitation or psychomotor retardation.

Anxiety disorders are similarly diagnosed by clinical interview. The criteria for "generalized anxiety disorders" include duration of at least six months of free-floating anxiety and autonomic overactivity.

4.8.14 ASSOCIATIONS BETWEEN INSOMNIA AND PSYCHIATRIC DISORDERS

At the present time, exposure–effect associations have not been established between parameters of sleep disturbance (number of behavioural awakenings, body movements or EEG awakenings) and the onset of depressive and anxiety disorders, although there is some evidence that insomnia is a risk factor for developing depression (Riemann, Berger and Voderholzer, 2001; Roberts, Roberts and Chen, 2002). A number of longitudinal prospective studies in different age groups have found associations between self-reports of insomnia and the subsequent onset of psychiatric disorder, in particular major depression. A selection of the most important studies and their findings are outlined in Table 4.3 below.

Table 4.3 Insomnia as a predictor of psychiatric disorder

Study	Sample size	Sample	Follow-up interval	Depression measure	Results
Ford and Kamerow, 1989	7954	Community sample	1 year	Diagnostic interview schedule	Risk of developing new depression for insomnia on two occasions: [OR=39.8, 95% CI 19.8-80.0]
Breslau et al., 1996	1200	21–30 years members of health maintenance organization	3 years	Diagnostic interview schedule	RR for new onset major depression associated with baseline insomnia [RR=4.0, 95% CI 1.5-5.6]
Chang et al., 1997	1053	Male medical students	34 years (median)	Clinical depression	RR for clinical depression for those who reported insomnia at medical school [RR= 2.0, 95% CI 1.2-3.3]
Roberts, Roberts and Chen, 2002	3136	11–17 years from managed care rosters	1 year	Diagnostic interview schedule for children major depression module	Fully adjusted OR for insomnia in waves 1 and 2 for depression at follow-up [OR=1.92, 95% CI 1.30-2.82]

4.8.15 CONCLUSIONS: ASSOCIATIONS BETWEEN NOISE AND PSYCHIATRIC DISORDERS

The effects of noise are strongest for those outcomes that, like annoyance, can be classified under "quality of life" rather than illness. What they lack in severity is made up for in numbers of people affected, as these responses are very widespread.

Current evidence does seem to suggest that environmental noise exposure, especially at higher levels, is related to mental health symptoms and possibly raised anxiety and consumption of sedative medication, but there is little evidence that it has more serious effects. Further research is needed on mental health effects at very high noise levels. Existing studies may be confounded either by prior selection of subjects out of (or into) noisy areas as a result of noise exposure, or by confounding between noise exposure, socioeconomic deprivation, and psychiatric disorder. It is also possible that people underestimate or minimize the effects of noise on health through optimism bias (Hatfield and Soames Job, 2001) and that this is particularly protective for mental health.

The evidence is not strong for the association between noise exposure and mental ill health. What evidence there is suggests that noise exposure may be responsible for psychological symptoms above 70 dB(A) L_{eq}. Almost all studies have only examined the effects of daytime noise on mental health, but it is possible that night-time noise, during sleep time, may have effects on mental health at lower levels than daytime noise.

The most powerful evidence of noise on mental health comes from studies of military aircraft noise. There is also some evidence that intense road traffic noise may lead to psychological symptoms. There is no evidence of any effects of railway noise on mental health.

4.9 THE SEVERITY OF SELF-REPORTED SLEEP DISTURBANCE

4.9.1 INTRODUCTION

In section 2.1.2 of Chapter 2 of this report, it is stated that sleep disturbance caused by noise may either be diagnosed (Environmental Sleep Disorder: ICSD 780-52-6) or self-reported. Although self-reported sleep disturbance is subjective by definition, its observed occurrence correlates with noise levels as well as with important diagnostic criteria for ICSD 780-52-6. It appears justified to consider self-reported sleep disturbance as an impairment of health, especially if indicated by representative population samples in social surveys. Furthermore, section 4.1 of Chapter 4 of this report gives a quantitative relationship between noise level L_{night} and the percentage of population that reports a disturbed sleep of high, medium or low disturbance intensity.

But an open question concerns severity: even if night-time noise causes large percentages of the population to declare themselves as highly sleep-disturbed, this could nevertheless represent an almost negligible loss of health, if the mean severity of self-reported sleep disturbance were negligible in comparison with commonly accepted diseases. Attempts have been made to give an answer to this important question, using WHO's concept of disability weights (Murray et al., 1996) as a basis for severity comparisons.

4.9.2 AN ASSESSMENT OF DISABILITY WEIGHTS

A Swiss study (Müller-Wenk, 2002) aimed at determining a disability weight for sleep disturbance due to road traffic noise. For this purpose, a description of road-

noise-related sleep disturbance was set up: essentially, this state of health was assumed to be present if a person indicated that, due to traffic noise, he or she, almost every night, had problems with falling asleep, with continuing sleep during the night or with early or non-restorative waking in the morning. In addition, a list was established with already available disability weights (Murray et al., 1996; Stouthard et al., 1997) for a selection of 28 diseases of various types, covering a range from very light severity to high severity (Müller-Wenk 2002:65–66). All 64 members of the medical staff of the Swiss Accident Insurance Institute (SUVA) were then asked in a written questionnaire to determine the hitherto unknown disability weight of sleep disturbance by interpolation, that is, by inserting sleep disturbance at the appropriate place between the presented 28 diseases that were sorted according to ascending disability weight. These participants were chosen because the physicians of the SUVA, besides being medical doctors, have a particularly high professional know-how in comparing the severity of different types of disability. Forty-two questionnaires were completed, of which 41 were usable.

From these questionnaires, an arithmetical mean of 0.055 of the disability weight for sleep disturbance could be calculated, with a 95% confidence limit of 0.039 at the low end and 0.071 at the high end. This result can be illustrated by mentioning diseases from the catalogues of Murray et al. (1996) or Stouthard et al. (1997) with the same disability weight: hence the disability weight of the road-noise-related sleep disturbance is roughly the same as the disability weight of "chronic hepatitis B infection without active viral replication", the latter having a mean disability weight of 0.06 and a 95% confidence interval from 0.034 to 0.087. The low-end estimate of 0.039 for sleep disturbance severity would correspond to the mean disability weight of "benign prostatic hypertrophy (symptomatic cases)", whilst the high estimate of 0.071 would correspond to the mean disability weight of "uncomplicated diabetes mellitus". The conclusion is that the mean disability weight of road-noise-related sleep disturbance is not smaller (= less severe) than the disability weight of health impairments commonly recognized as diseases, and there is a strong overlap amongst the probability distributions of these disability weights. On the basis of the chosen disability weight 0.055 for self-reported sleep disturbance, and taking into account the current traffic noise levels during the night in many European states, it is justified to consider noise-related sleep disturbance as a substantial loss of public health.

4.9.3 COMPARISON BETWEEN INSOMNIA AND SELF-REPORTED SLEEP DISTURBANCE

The original list of disability weights (Murray et al., 1996) did not contain any kind of non-normal sleep. In the meantime, WHO has published an extended list (Mathers et al., 2003, Annex Table 5a) containing a disability weight of 0.100 for insomnia (diagnostic code 307.42). This has opened a way to recheck the disability weight of 0.055 (Müller-Wenk, 2002), by asking a panel of medical professionals to compare, on the basis of disability weights, the mean severity of self-declared sleep disturbance due to road noise at night with the mean severity of insomnia. It may be debated whether it is more straightforward to compare two types of sleep anomalies with similar symptoms, or to compare self-declared sleep disturbance with various types of completely different diseases. But it makes sense anyway to use the comparison with insomnia as a second approach for determining the disability weight of self-reported sleep disturbance.

This severity comparison between different sleep anomalies was made in 2005 by structured oral interviews, executed by a medical staff member of the sleep clinic of Kantonsspital St. Gallen (Switzerland), with 14 GPs selected at random from all GPs who had admitted patients to the sleep clinic during the nine preceding months. These patients were mainly suffering from OSAS. The question was as follows:

> "Could you please give us your opinion on the relative severity of three different cases of insomnia:
>
> 1. (primary) insomnia, in our region usually called psychophysiological insomnia
> 2. Obstructive Sleep Apnoea Syndrome (OSAS)
> 3. traffic-noise-related sleep disturbance, that may occur with persons who are forced to sleep along through roads with nocturnal motor traffic.
>
> Your opinion should be based on the patients you have seen in your office lately, or on other persons of your social environment. When comparing the severity of the health impairment, the focus should be above all on the person's condition during the day after the sleep-disturbed night. The absolute value of the severity is less important for the current study than the relative severity amongst the three cases of insomnia. The opinion of the severity may be expressed on a linear scale from 0 (no impairment at all) to 10 (impairment almost unsupportable). On the scale from 0 to 10, you may give us your mean value of the severity, or you may give us a span from a low to a high for the severity."

All of the interviewed GPs gave their opinions, and the result is presented in Table 4.4.

Table 4.4
Severity ratings (10 = almost insupportably disturbing, 0 = not in the least disturbing) by 14 GPs selected at random

	Primary insomnia				OSAS (sleep apnoea)				Sleep disturb.(noise)				Ratio noise/	Noise/
No	Max	Min	Mean	Rank	Max	Min	Mean	Rank	Max	Min	Mean	Rank	priminsomnia	OSAS
10	6	4	5	3	8	6	7	1	8	6	7	1	1.40	1.00
11	5	3	4	3	9	7	8	1	8	4	6	2	1.50	0.75
12			5	3			10	1	7	8	7.5	2	1.50	0.75
13	2	3	2.5	2	4	5	4.5	1	1	2	1.5	3	0.60	0.33
14			3	2			6	1	1	2	1.5	3	0.50	0.25
15			8	2			9	1			6	3	0.75	0.67
16			8	1			7	2			4	3	0.50	0.57
17			5	1			5	1			3	3	0.60	0.60
18	2	3	2.5	2			6	1	1	2	1.5	3	0.60	0.25
19			8	1			3	2			2	3	0.25	0.67
20			6	2			7	1			4	3	0.67	0.57
21			7	2			8	1			0	3	0.00	0.00
22			4	3			5	2			6	1	1.50	1.20
23			4	3	6	7	6.5	2	8	9	8.5	1	2.13	1.31
Mean			5.143	2.143			6.57	1.286			4.18	2.429	0.89	0.64
Sigma													0.60	
Median													0.63	
Upper value 95% C.I. for mean													1.20	
Lower value 95% C.I. for mean													0.58	

Clearly, the severity judgements vary widely between the participating GPs. Apart from the differences in personal judgement, this variation is certainly influenced by the mix of patients visiting a particular GP. For instance, GP number 15 could have encountered one or two very serious cases of OSAS, whilst his/her experience with noise-related sleep disturbance might refer to persons that were only moderately disturbed by night-time noise in their bedroom. On the other hand, number 22 could

have had experience with persons suffering very much from sleep disturbance due to high traffic noise exposure, whilst his/her OSAS or primary insomnia patients happened to be light cases. One must accept that even GPs have a limited experience with the whole range of cases of each of the three types of insomnia, so that their opinion on the mean severity of noise-related sleep disturbance, compared to the mean severity of OSAS or insomnia, is influenced by the randomness of their patient mix.

Nevertheless, the table supports the following statements.

- With respect to severity, the majority of GPs rank noise-related sleep disturbance lower than insomnia and OSAS, while three of them put noise-related sleep disturbance in the first rank. Only one of the participants (number 21) considers noise-related sleep disturbance as a fully negligible disturbance.
- The severity ratio between noise-related sleep disturbance and insomnia varies between 0 and 2.1. Seven of the fourteen GPs indicate a severity ratio between 0.5 and 0.75, that is to say that half the participants are of the opinion that the severity of noise-related sleep disturbance amounts to 50–75% of the severity of insomnia.
- The mean of this severity ratio is 0.89, with a standard deviation (sigma) of 0.60. The confidence interval (CI) for the mean goes from 0.58 to 1.20. The median of the severity ratio is 0.63. The distribution is skewed to the right.

The severity ratio developed above can be used as a proportionality factor between the known disability weight for insomnia and the required disability weight for self-reported sleep disturbance. Bearing in mind that the already existing WHO disability weight for insomnia is 0.10, a best guess for the mean disability weight for self-reported sleep disturbance due to road traffic noise at night is therefore 0.089, with a CI from 0.058 to 0.12.

4.9.4 CONCLUSIONS

According to the two groups of interviewed medical professionals, persons that declare themselves to be chronically deprived of normal sleep by road traffic noise have a health state whose mean disability weight is comparable to "chronic hepatitis B infection without active viral replication" or higher. Irrespective of the question whether self-reported sleep disturbance is formally recognized as a disease or not, its severity is comparable to commonly accepted diseases.

The best estimate for a mean disability weight for self-reported sleep disturbance due to road traffic noise was 0.055 (CI: 0.039; 0.071) according to Müller-Wenk (2002), whilst our recheck based on a comparison with insomnia resulted in a disability weight of 0.09 (CI: 0.06; 0.12). The higher disability weight according to the second approach might be caused by the fact that in this second approach, there was a stronger focus on "the person's condition during the day after the sleep-disturbed night".

The above figures compare reasonably with a study published by van Kempen (1998), cited in Knol and Staatsen (2005:46), where a severity weight of 0.10 for severe sleep disturbance was found, based on the judgement of 13 medical experts according to the protocol of Stouthard et al. (1997).

In conclusion, a mean disability weight of 0.07 is proposed for self-reported sleep disturbance due to road noise or similar ambient noise. This disability weight can be used in connection with the equations of section 4.1 of this chapter for highly sleep-disturbed persons.

4.10 DISCUSSION: CAN CHRONIC SHORT-TERM EFFECTS CAUSE LONG-TERM EFFECTS ON HEALTH?

EEG modifications, cardiovascular responses, body movements and awakenings due to noise occur within a few seconds after the stimulus. In addition to the instantaneous effects related to single events, large field studies on aircraft (Passchier-Vermeer et al., 2002) and road traffic noise exposure during night-time (Griefahn et al., 2000; Passchier-Vermeer et al., 2004) show that also sleep latency and average motility during the sleep period increased monotonously as a function of the noise exposure level. The increase in average motility was substantially higher than would be expected on the basis of the instantaneous extra motility at the times of the noise events (Passchier-Vermeer et al., 2002) suggesting persistent arousal during the sleep related to aircraft noise. Furthermore, an international field study (Jurriëns et al., 1983) found slightly reduced REM sleep, increased time being awake according to the EEG, increased average heart rate, and reduced performance on a reaction time test in people when exposed during the night to higher road traffic noise levels.

The relationship between instantaneous effects and more global modifications of one night sleep, as well as chronic changes, is not simple, as illustrated by the findings concerning motility. An increase in average motility that is substantially higher than would be expected on the basis of the instantaneous extra motility at the times of the noise events (Passchier-Vermeer et al., 2002) suggests a persistent arousal during sleep related in a dose-dependent way to the aircraft noise.

Since EEG arousal and instantaneous motility are correlated, this finding suggests that also the number of (micro-)arousals may increase during noise exposure more than by the sum of the instantaneous (micro-)arousals that occur contingent upon a noise event.

For overall motility during sleep, clear indications have been found of associations with further effects, although the causal direction is not in all cases clear. Mean (onset of) motility during sleep is associated with the following variables based on questionnaires and diaries (Passchier-Vermeer et al., 2002):

- frequency of conscious awakening during the sleep period: the increase is 0.8 conscious;
- awakenings per night, if motility increases from low to high;
- frequency of awakening remembered next morning: the increase is 0.5 remembered;
- awakenings per night, if motility increases from low to high;
- long-term frequency of awakening attributed to specific noise sources assessed with a questionnaire;
- sleep quality reported in a morning diary;
- long-term sleep quality assessed with a questionnaire;

- number of sleep complaints assessed with a questionnaire;
- number of general health complaints assessed with a questionnaire.

The associations of mean motility with these variables are stronger than the corresponding associations of mean onset of motility.

For evaluating the adverseness of the instantaneous effects, it is important to consider whether they bring the body into a more persistent state of higher arousal or not, although this is not the only criterion. Those effects which are progressively disappearing with the repetition of the stimulus may be less harmful than those which do persist over long exposure time, provided that the suppression of the effects do not require costs in another form. For example, short-term cardiovascular effects that appear not to habituate could lead to permanent cardiovascular system impairment (Carter, 1996, 1998).

The relations presented for motility and conscious awakening imply that motility is sensitive to noise and has a relatively low threshold, while conscious awakening, the strongest instantaneous interference of noise with sleep, has the highest threshold of the instantaneous effects considered.

In one of the most sophisticated field studies (Passchier-Vermeer et al., 2002), increased probability of instantaneous motility was found for events with a maximum sound level L_{Amax} > 32 dB(A), while in a meta-analysis conscious awakening was found for events with L_{Amax} > 42 dB(A) (Passchier-Vermeer, 2003a). Above their threshold, these effects were found to increase monotonously as a function of the maximum sound level during a noise event (aircraft noise). It is important to note that in another recent sophisticated field study (Basner et al., 2004), the threshold found for EEG awakening was L_{Amax} = 35 dB(A), that is, only a little higher than the 32 dB(A) found for noise-induced awakenings. This strengthens the evidence that noise starts to induce arousals at L_{Amax} values in the range 30–35 dB(A). Given the night-time noise levels to which people are exposed, these results imply that instantaneous effects are common. Although most studies concerned aircraft noise, the instantaneous effects can be assumed to occur at similar levels for different types of transportation.

The above observations can be used as a basis for setting limits with respect to night-time transportation noise. For transparency, it is useful to distinguish two steps in choosing actual limits: the first step is the derivation of a health-based limit; the second step is the derivation of an actual limit that takes into account the health-based limit as well as feasibility arguments. Here the concern is with the first step.

When deriving a health-based limit, two points need to be considered: the dose-dependent effects of a single noise event, and the number of events. With respect to the dose-dependent effects of a single event, adverse effects can be distinguished from effects that by themselves need not be adverse but can contribute to an adverse state. It is proposed to classify conscious awakenings as an adverse effect. Conscious awakenings have been estimated to occur at a baseline rate of 1.8 awakening per night. A substantial increment of conscious awakenings over this baseline is thought to be adverse. Since, in general, falling asleep after conscious awakening takes some time, and this latency is longer after noise-induced conscious awakening that will often also induce an emotional reaction (anger, fear), it will also reduce the time asleep and may affect mood and functioning next day. Although additional, more sophisticated analyses could be performed to refine this estimate, we propose L_{Amax}

= 42 dB(A) is proposed as the currently best estimate of the threshold for conscious awakening by transportation noise. This would mean that the no observed effect level ($NOEL_{Amax}$) for transportation noise events is at most 42 dB(A).The most sensitive instantaneous effect that has been studied extensively in field studies is motility. A single interval with (onset of) noise-induced motility by itself cannot be considered to be adverse.

However, noise-induced motility is a sign of arousal, and frequent (micro-)arousal and accompanying sleep fragmentation can affect mood and functioning next day and lead to a lower rating of the sleep quality. Therefore, motility is relevant for adverse health effects, but more than a few intervals with noise-induced motility are needed for inducing such effects. Although additional, more sophisticated analyses could be performed to refine this estimate, we propose L_{Amax} = 32 dB(A) as the currently best estimate of the threshold for motility induced by transportation noise. The threshold found for EEG awakening was L_{Amax} = 35 dB(A), that is, only a little higher than the 32 dB(A) found for noise-induced awakenings. This would mean that the $NOEL_{Amax}$ for transportation noise events is most likely at most 32 dB(A), and definitely not higher than 35 dB(A). It is important to note that the above given $NOEL_{Amax}$ ~ 32 dB(A) and $NOEL_{Amax}$ ~ 42 dB(A) are indoor levels, in the sleeping room. Although events below 32 dB(A) are audible, and, hence, further research may show more sensitive effects than motility, on the basis of the present available evidence we propose to assume that $NOEL_{Amax}$ = 32 dB(A) and set a health-based night-time noise limit that is tolerant for transportation noise events with L_{Amax} ~ 32 dB(A). On the other hand, since adverse health effects need to be prevented by health-based limits and even though vulnerable groups may require lower limits, on the basis of the present available evidence we propose to assume that $NOAEL_{Amax}$ = 42 dB(A) and set a health-based night-time noise limit that does not tolerate transportation noise events with L_{Amax} > 42 dB(A).

On the basis of the above proposal, it would be possible to derive a night-time noise guideline value in terms of L_{night}. Such a guideline value would indicate the level below which no short-term effects are to be expected that would lead to temporary reduced health or chronic disease. Such a guideline value needs to be compared with guideline values derived directly with a view to preventing temporary reduced health and chronic diseases. In particular, for self-reported sleep disturbance, which is an expression of reduced well-being and may be an indication of effects that could contribute to cardiovascular disease, exposure–effect relationships have been derived on the basis of an extensive set of original data from studies from various countries (Miedema, Passchier-Vermeer and Vos, 2003; Miedema, 2004). The percentage of people reporting high noise-induced sleep disturbance (%HS) levels off at 45 dB(A) but at a non-zero effect level. The remaining effect may be caused by events not incorporated in the exposure assessment and it appears that if all noise contributions would be incorporated in the exposure metric, high noise-induced sleep disturbance would vanish between 40 dB(A) and 45 dB(A), say at 42 dB(A). Since values found for other temporary reduced health effects or chronic diseases, in particular cardiovascular diseases, will be higher, and considering self-reported sleep disturbance as an adverse effect, this would suggest L_{night} = 42 dB(A) as the NOAEL to be compared with the value derived from the short-term effects. Note that this is an outdoor level, which would, assuming partly opened windows and an actual insulation of 15 dB(A), correspond to an indoor equivalent night-time sound level of 27 dB(A). The above discussion is based on motility, EEG awakenings, and conscious awakening. In addition, EEG micro-/minor arousals, and autonomic reactions have been discussed above.

Furthermore, there are potential instantaneous effects, such as effects on memory consolidation or restoration of the immune system, for which the information on a possible relation with noise exposure is so limited that they were not considered here. In order to acquire more insight into these effects, more field research is needed. Field research is needed because earlier studies have shown that estimates of effects on the basis of laboratory studies are much higher than estimates from field studies. Methodological differences between the different approaches certainly cannot be the only possible explanation. Research allowing the introduction of some specific but light laboratory technique into the sleeper's own bedroom, should be encouraged, as, for example, used in the Swiss Noise Study 2000 (Brink, Müller, and Schierz, 2006). The key to better insight into effects of night-time noise, leading to mechanistic models describing the relationships between noise exposure, instantaneous effects, effects at the level of a 24-hour period and chronic effects, appears to be epidemiological studies at home with well-designed instrumentation.

The relationships between noise exposure, instantaneous effects, effects at the level of a single 24-hour period and chronic effects is complex because the effects at a smaller time scale do not simply add up to effects at a larger time scale. For example, the noise-related increase in night-time average motility was substantially higher than would be expected on the basis of the instantaneous extra motility at the times of the noise events (Passchier-Vermeer et al., 2002), suggesting persistent arousal during sleep related to aircraft noise. It is likely that such shifts in the basic state are more important for the development of chronic effects than the instantaneous effects per se. A further complication is that some effects habituate. Habituation in some effect parameters can occur in a few days or weeks, but the habituation is not always complete. The measured modifications of the cardiovascular functions remain unchanged over long periods of exposure time (Muzet and Ehrhart, 1980; Vallet et al., 1983). Most striking is that none of the cardiovascular responses show habituation to noise after a prolonged exposure, while subjective habituation occurs within a few days. It appears plausible that, in particular non-habituating effects lead to the development of chronic effects, but also the disappearance of effects with continuing exposure may come at a cost associated with suppressing the effects. A third complication is that daytime noise exposure may contribute to the effects found in relation to night-time noise. Large epidemiological studies are needed that compare populations exposed to similar daytime noise and differ in their night-time noise exposure only. A specific challenge for mechanistic models on the effects of noise on sleep is the identification of factors that make subjects vulnerable to night-time noise. The following groups may be hypothesized to be more vulnerable to noise during sleep: old people, ill people, people with chronic insomnia, shift workers and people resting during daytime, people with a tendency to depression, light sleepers, pregnant women, people with high anxiety and high stress levels. Furthermore, children need attention because of their relatively high exposure during sleep, and because they are in a phase of neurocognitive development for which undisturbed sleep may be particularly important.

CHAPTER 5

GUIDELINES AND RECOMMENDATIONS

5.1 ASSESSMENT

In Chapter 1 the need for a guideline document for night-time exposure to noise was defended on the basis of the lack of existing guidance, the signs that a substantial part of the population could be exposed to levels of noise that might risk their health and well-being and the EU activities that compel the public and authorities to take notice when noise maps showing L_{night} levels are made public.

Where sufficient direct evidence concerning the effects of night-time noise on health could not be collected, indirect evidence was looked at: the effects of noise on sleep (quality) and the relations between sleep and health.

In Chapter 2 the evidence was presented that sleep is a biological necessity and disturbed sleep is associated with a number of health outcomes. Studies of sleep disturbance in children and in shift workers clearly show the adverse effects. Unravelling the relations between sleep and health (Fig. 2.1) shows that sleep is an essential feature of the organism, so that simple direct relations can hardly be expected.

In Chapter 3 it was shown beyond doubt that noise disturbs sleep through a number of direct and indirect pathways. Even at very low levels physiological reactions (heart rate, body movement and arousals) can be reliably measured. It was also shown that awakening reactions are relatively rare, occurring at a much higher level.

Chapter 4 summarized the known evidence for the direct effects of night-time noise on health. The working group agreed that there is sufficient evidence that night noise is related to self-reported sleep disturbance, use of pharmaceuticals, self-reported health problems and insomnia-like symptoms. These effects can lead to a considerable burden of disease in the population. For other effects (hypertension, myocardial infarctions, depression and others), limited evidence was found: although the studies were few or not conclusive, a biologically plausible pathway could be constructed from the evidence.

An example of a health effect with limited evidence is myocardial infarction. Although evidence for increased risk of myocardial infarction related to L_{day} is sufficient according to an updated meta-analysis, the evidence in relation to $L_{night,outside}$ was considered limited. This is because $L_{night,outside}$ is a relatively new exposure indicator, and few field studies have focused on night noise when considering cardiovascular outcomes. Nevertheless, there is evidence from animal and human studies supporting a hypothesis that night noise exposure might be more strongly associated with cardiovascular effects than daytime exposure, highlighting the need for future epidemiological studies on this topic.

The review of available evidence leads to the following conclusions.

- Sleep is a biological necessity and disturbed sleep is associated with a number of health outcomes.

- There is sufficient evidence for biological effects of noise during sleep: increase in heart rate, arousals, sleep stage changes and awakening.
- There is sufficient evidence that night noise exposure causes self-reported sleep disturbance, increase in medicine use, increase in body movements and (environmental) insomnia.
- While noise-induced sleep disturbance is viewed as a health problem in itself (environmental insomnia), it also leads to further consequences for health and well-being.
- There is limited evidence that disturbed sleep causes fatigue, accidents and reduced performance.
- There is limited evidence that noise at night causes hormone level changes and clinical conditions such as cardiovascular illness, depression and other mental illness. It should be stressed that a plausible biological model is available with sufficient evidence for the elements of the causal chain.

In the next section threshold levels are presented for the effects, where these can be derived.

5.2 THRESHOLDS FOR OBSERVED EFFECTS

The NOAEL is a concept from toxicology, and is defined as the greatest concentration which causes no detectable adverse alteration of morphology, functional capacity, growth, development or lifespan of the target organism. For the topic of night-time noise (where the adversity of effects is not always clear) this concept is less useful. Instead, the observed effect thresholds are provided: the level above which an effect starts to occur or shows itself to be dependent on the dose. This can also be an adverse effect (such as myocardial infarcts) or a potentially dangerous increase in a naturally occurring effect such as motility.

Threshold levels are important milestones in the process of evaluating the health consequences of environmental exposure. The threshold levels also delimit the study area, which may lead to a better insight into overall consequences. In Tables 5.1 and 5.2 all effects are summarized for which sufficient or limited evidence exists (see Table 1.2 in Chapter 1 for a definition). For the effects with sufficient evidence the threshold levels are usually well known, and for some the dose-effect relations over a range of exposures could also be established.

5.3 RELATIONS WITH $L_{night,outside}$

Over the next few years, the END will require that night exposures are reported in $L_{night,outside}$. It is therefore interesting to look into the relation between $L_{night,outside}$ and the effects from night-time noise. The relation between the effects listed in Tables 5.1 and 5.2 and $L_{night,outside}$ is, however, not straightforward. Short-term effects are mainly related to maximum levels per event inside the bedroom: $L_{Amax,inside}$. In order to express the (expected) effects in relation to the single EU indicator, some calculation needs to be done.

Table 5.1
Summary of effects and threshold levels for effects where sufficient evidence is available

Effect		Indicator	Threshold, dB	Reference (chapter, section)
	Change in cardiovascular activity	*	*	3.1.5
	EEG awakening	$L_{Amax, inside}$	35	4.10
Biological effects	Motility, onset of motility	$L_{Amax, inside}$	32	3.1.8, dose–effect relation for aircraft
	Changes in duration of various stages of sleep, in sleep structure and fragmentation of sleep	$L_{Amax, inside}$	35	3.1
	Waking up in the night and/or too early in the morning	$L_{Amax, inside}$	42	3.1.7, dose–effect relation for aircraft
Sleep quality	Prolongation of the sleep inception period, difficulty getting to sleep	*	*	3.1
	Sleep fragmentation, reduced sleeping time	*	*	3.1
	Increased average motility when sleeping	$L_{night, outside}$	42	3.2, dose–effect relation for aircraft
Well-being	Self-reported sleep disturbance	$L_{night, outside}$	42	4.2, dose–effect relation for aircraft/road/rail
	Use of somnifacient drugs and sedatives	$L_{night, outside}$	40	4.5.8
Medical conditions	Environmental insomnia**	$L_{night, outside}$	42	3.1; 4.1; 4.2

* *Although the effect has been shown to occur or a plausible biological pathway could be constructed, indicators or threshold levels could not be determined.*

** *Note that "environmental insomnia" is the result of diagnosis by a medical professional whilst "self-reported sleep disturbance" is essentially the same, but reported in the context of a social survey. Number of questions and exact wording may differ.*

Table 5.2
Summary of effects and threshold levels for effects where limited evidence is available+

Effect		Indicator	Estimated, threshold dB	Reference (chapter, section)
Biological effects	Changes in (stress) hormone levels	*	*	2.5
	Drowsiness/tiredness during the day and evening	*	*	2.2.3
	Increased daytime irritability	*	*	2.2.3
Well-being	Impaired social contacts	*	*	2.2.3
	Complaints	$L_{night,outside}$	35	4.3
	Impaired cognitive performance	*	*	2.2.3
	Insomnia	*	*	4.6
	Hypertension	$L_{night,outside}$	50	2.2.3; 4.5.6
	Obesity	*	*	2.2.3
	Depression (in women)	*	*	4.8
	Myocardial infarction	$L_{night,outside}$	50	4.5.15
	Reduction in life expectancy (premature mortality)	*	*	2.2.3; 2.5
	Psychiatric disorders	$L_{night,outside}$	60	4.8.15
	(Occupational) accidents	*	*	2.2.3; 2.4

+ *Note that as the evidence for the effects in this table is limited, the threshold levels also have a limited weight. In general they are based on expert judgement of the evidence.*

* *Although the effect has been shown to occur or a plausible biological pathway could be constructed, indicators or threshold levels could not be determined.*

The calculation for the total number of effects from reaction data on events (arousals, body movements and awakenings) needs a number of assumptions. The first that needs to be made is independence: although there is evidence (Brink, Müller and Schierz, 2006) that the order of events of different loudness strongly influences the reactions, the calculation is nearly impossible to carry out if this is taken into consideration.

Secondly, the reactions per event are known in relation to levels at the ear of the sleeper, so an assumption for an average insulation value must be made. In this report a value of 21 dB (see Chapter 1, sections 1.3.4 and 1.3.5) has been selected. This value is, however, subject to national and cultural differences. One thing that stands out is the desire of a large part of the population to sleep with windows (slightly) open. The relatively low value of 21 dB already takes this into account. If noise levels increase, people do indeed close their windows, but obviously reluctantly, as then complaints about bad air increase and sleep disturbance remains high. This was already pointed out in the WHO guidelines on community noise (WHO, 1999).

From source to source the number of separate events varies considerably. Road traffic noise is characterized by relatively low levels per event and high numbers, while air and rail traffic are characterized by high levels per event and low numbers. For two typical situations estimates are made and presented in graphical form. The first is an average urban road (600 motor vehicles per night, which corresponds roughly to a 24-hour use of 8000 motor vehicles, or 3 million per year, the lower boundary the END sets) and the second case is for an average situation of air traffic exposure (8 flights per night, nearly 3000 per year).

Fig. 5.1 shows how effects increase with an increase of $L_{night,outside}$ values for the typical road traffic situation (urban road). A large number of events lead to high levels of awakening once the threshold of $L_{Amax,inside}$ is exceeded. To illustrate this in practical terms: values over 60 dB $L_{night,outside}$ occur at less then 5 metres from the centre of the road.

In Fig. 5.2 the same graph is presented for the typical airport situation. Due to a lower number of events there are fewer awakenings than in the road traffic case (Fig. 5.1), but the same or more health effects.

In these examples the worst case figures can be factors higher: the maximum number of awakenings for an $L_{night,outside}$ of 60–65 dB is around 300 per year.

A recent study suggests that high background levels (from motorways) with low numbers of separate events can cause high levels of average motility (Passchier-Vermeer, to be published). In Table 5.3 the full details are summarized.

5.4 DEALING WITH SITUATIONS EXCEEDING THE THRESHOLDS

Noise exposure data demonstrate that a large part of the population is over the no-effect levels. It is expected that this will extend into the future for quite some time. This means that circumstances may require that a risk assessment must be made. It is then recommended to apply the method laid out in Chapter 1, using the values given in Tables 5.1 and 5.2 and the dose–effect relations given in Chapter 4.

Typical actions requiring risk assessment are:

- new infrastructure projects (if an environmental impact statement is required)
- improvement programmes

- policy evaluation
- national or international setting of limit values.

In the EU *Position Paper* (European Commission, 2002a) an overview of national night-time noise regulations can be found.

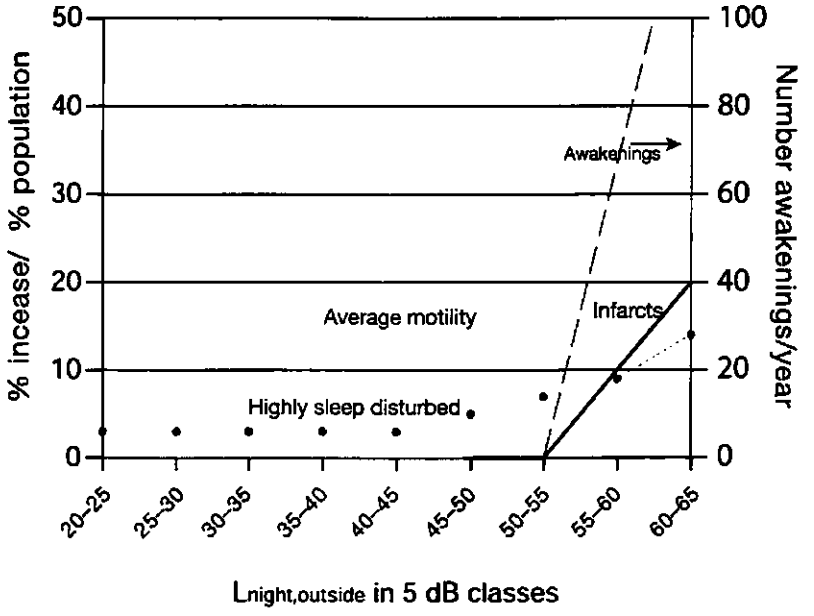


Fig. 5.1 Effects of road traffic noise at night*

Source: European Commission, 2002 a

** Average motility and infarcts are expressed in percent increase (compared to baseline number); the number of highly sleep disturbed people is expressed as a percent of the population; awakenings are expressed in number of additional awakenings per year.*

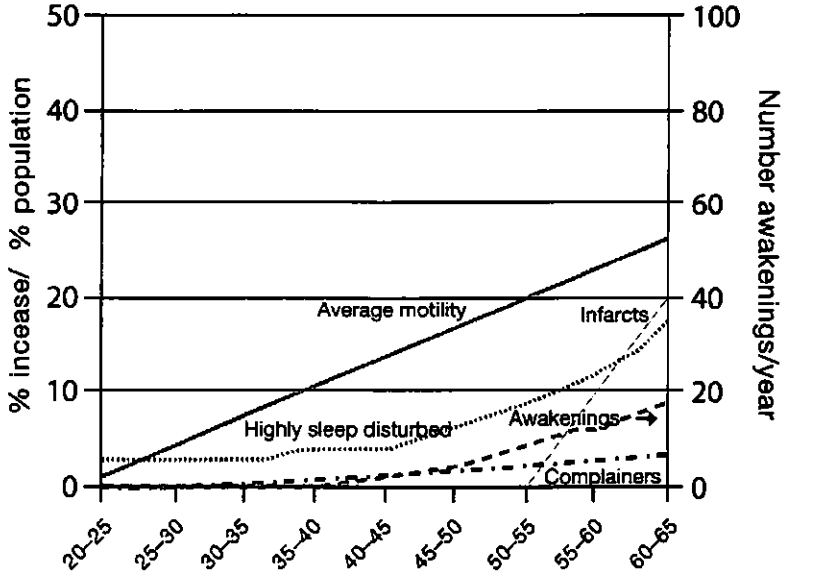


Fig. 5.2 Effects of aircraft noise at night*

Source: European Commission, 2002 a

** Average motility and infarcts are expressed in percent increase (compared to baseline number); the number of highly sleep disturbed people is expressed as percent of the population; complainers are expressed as a percent of the neighbourhood population; awakenings are expressed in number of additional awakenings per year.*

Table 5.3
Effects (yearly, additional with respect to the normal except odd ratio) 0=below threshold, +=increase)

$L_{night, outside}$	Arousals		Body movements related to single exposures (15 sec intervals)		Average body movements (without single exposures)	Awakenings		% sleep disturbed (% highly sleep disturbed)			Myocardial infarcts
UNIT	number	number	number average	number average urban	number	number average	number average urban	% of exposed			odd ratio
		air	traffic	road		air traffic	road	air	road	rail	
NORMAL	Children 2 555	Adults 3 650	21 000		21 000	600		0			1
20–25	0	0	0	0	200	0	0	0	0	0	1
25–30	0	0	7	0	875	0	0	<3	<3	<2	1
30–35	+	+	22	0	1 547	0	0	<3	<3	<2	1
35–40	+	+	37	0	2 220	0	0	4	3	2	1
40–45	+	+	58	243	2 900	2	0	4	3	2	1
45–50	+	+	85	635	3 600	5	0	6	5	2	1
50–55	+	+	111	1 145	4 200	9	0	9	7	3	1
55–60	+	+	145	1 770	4 900	12	54	12	9	4	1.1
60–65	+	+	180	2 520	5 500	17	155	17	14	6	1.2

Source: European Commission, 2002 a

5.5 PROTECTION MEASURES AND CONTROL

What is the best strategy to reduce sleep disturbance? The first thought should always be to reduce the impact, either by reducing the number of events or by reducing the sound levels, or both. For some effects reducing the number of events may seem to be more effective (although that depends on the exact composition). Other effects are reduced by lowering overall noise level by either the number of events, the levels per event or by any combination.

In combination with other measures, sound insulation of bedroom windows is an option, but care must be taken to avoid negative impact on inside air quality. Even then, many people may want to sleep with their windows open, thereby making the insulation ineffective. Although good instruction may go some way to helping to overcome this, it is still a matter well worth taking into account. In warmer climates, in particular, insulation is not a serious option for residential purposes and excessive exposure must be avoided either by removing the people exposed or removing the source if source-related measures fail.

Although air conditioning of houses (or just bedrooms) is not commonplace in the EU, there are indications that its use is increasing, especially in the warmer parts of the Region. Although this still leaves the possibility that people may sleep with their windows open outside the summer season, it is something to consider when discussing measures.

Exposed areas could be a good choice for uses such as offices, where there will be no people at night, or where it is a physical impossibility to sleep with the windows open (fully air-conditioned buildings, for example hotels and sometimes hospitals).

A simple measure is the orientation of noise-sensitive rooms on the quiet side of the dwelling (this applies to road and rail traffic noise).

Zoning is an instrument that may assist planning authorities in keeping noise-sensitive land uses away from noisy areas. In the densely populated areas of the EU this solution must often compete, however, with other planning requirements or a simple lack of suitable space.

5.6 RECOMMENDATIONS FOR HEALTH PROTECTION

Sleep is an essential part of healthy life and is recognized as a fundamental right under the European Convention on Human Rights[1](European Court of Human Rights, 2003). Based on the systematic review of evidence produced by epidemiological and experimental studies, the relationship between night noise exposure and health effects can be summarized as below. (Table 5.4)

Table 5.4
Effects of different levels of night noise on the population's health[2]

Average night noise level over a year $L_{night,outside}$	Health effects observed in the population
Up to 30 dB	Although individual sensitivities and circumstances may differ, it appears that up to this level no substantial biological effects are observed. $L_{night,outside}$ of 30 dB is equivalent to the NOEL for night noise.
30 to 40 dB	A number of effects on sleep are observed from this range: body movements, awakening, self-reported sleep disturbance, arousals. The intensity of the effect depends on the nature of the source and the number of events. Vulnerable groups (for example children, the chronically ill and the elderly) are more susceptible. However, even in the worst cases the effects seem modest. $L_{night,outside}$ of 40 dB is equivalent to the LOAEL for night noise.
40 to 55 dB	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.
Above 55 dB	The situation is considered increasingly dangerous for public health. Adverse health effects occur frequently, a sizeable proportion of the population is highly annoyed and sleep-disturbed. There is evidence that the risk of cardiovascular disease increases.

[1] "Article 8:1. Everyone has the right to respect for his private and family life, his home and his correspondence." Although in the case against the United Kingdom the Court ruled that the United Kingdom Government was not guilty of the charges, the right to undisturbed sleep was recognized (the Court's consideration 96).

[2] $L_{night,outside}$ in Table 5.4 and 5.5 is the night-time noise indicator (L_{night}) of Directive 2002/49/EC of 25 June 2002: the A-weighted long-term average sound level as defined in ISO 1996-2: 1987, determined over all the night periods of a year; in which: the night is eight hours (usually 23.00 – 07.00 local time), a year is a relevant year as regards the emission of sound and an average year as regards the meteorological circumstances, the incident sound is considered, the assessment point is the same as for L_{den}. See *Official Journal of the European Communities*, 18.7.2002, for more details.

Below the level of 30 dB $L_{night,outside}$, no effects on sleep are observed except for a slight increase in the frequency of body movements during sleep due to night noise. There is no sufficient evidence that the biological effects observed at the level below 40 dB $L_{night,outside}$ are harmful to health. However, adverse health effects are observed at the level above 40 dB$L_{night,outside}$, such as self-reported sleep disturbance, environmental insomnia, and increased use of somnifacient drugs and sedatives. Therefore, 40 dB $L_{night,outside}$ is equivalent to the LOAEL for night noise. Above 55 dB the cardiovascular effects become the major public health concern, which are likely to be less dependent on the nature of the noise. Closer examination of the precise impact will be necessary in the range between 30 dB and 55 dB as much will depend on the detailed circumstances of each case.

A number of instantaneous effects are connected to threshold levels expressed in L_{Amax} (Table 5.1). The health relevance of these effects cannot be easily established. It can be safely assumed, however, that an increase in the number of such events over the baseline may constitute a subclinical adverse health effect by itself leading to significant clinical health outcomes.

Based on the exposure–effects relationship summarized in Table 5.4, the night noise guideline values are recommended for the protection of public health from night noise as below (Table 5.5).

Table 5.5
Recommended night noise guidelines for Europe

Night noise guideline (NNG)	$L_{night,outside}$ = 40 dB
Interim target (IT)	$L_{night,outside}$ = 55 dB

For the primary prevention of subclinical adverse health effects related to night noise in the population, it is recommended that the population should not be exposed to night noise levels greater than 40 dB of $L_{night,outside}$ during the part of the night when most people are in bed. The LOAEL of night noise, 40 dB $L_{night,outside}$, can be considered a health-based limit value of the night noise guidelines (NNG) necessary to protect the public, including most of the vulnerable groups such as children, the chronically ill and the elderly, from the adverse health effects of night noise.

An interim target (IT) of 55 dB $L_{night,outside}$ is recommended in the situations where the achievement of NNG is not feasible in the short run for various reasons. It should be emphasized that IT is not a health-based limit value by itself. Vulnerable groups cannot be protected at this level. Therefore, IT should be considered only as a feasibility-based intermediate target which can be temporarily considered by policy-makers for exceptional local situations.

All Member States are encouraged to gradually reduce the proportion of the population exposed to levels over the IT within the context of meeting wider sustainable development objectives. It is highly recommended to carry out risk assessment and management activities at local and national levels targeting the exposed population, and aiming at reducing night noise to the level below IT or NNG. IT and NNG can be used for health impact assessment of new projects (for example construction of roads, railways, airports or new residential areas) even before the achievement of IT, as well as for the risk assessment of the whole population. In the long run the NNG would be best achieved by control measures aimed at the sources along with other comprehensive approaches.

5.7 RELATION WITH THE GUIDELINES FOR COMMUNITY NOISE (1999)

The *Guidelines for community noise* (WHO, 1999) have been quoted a number of times in this paper, so one could rightfully ask what the relation is between the 1999 guidelines and the present NNG.

Impact of night-time exposure to noise and sleep disturbance is indeed covered in the 1999 guidelines, and this is the full statement (WHO, 1999):

> "If negative effects on sleep are to be avoided the equivalent sound pressure level should not exceed 30 dBA indoors for continuous noise. If the noise is not continuous, sleep disturbance correlates best with L_{Amax} and effects have been observed at 45 dB or less. This is particularly true if the background level is low. Noise events exceeding 45 dBA should therefore be limited if possible. For sensitive people an even lower limit would be preferred. It should be noted that it should be possible to sleep with a bedroom window slightly open (a reduction from outside to inside of 15 dB). To prevent sleep disturbances, one should thus consider the equivalent sound pressure level and the number and level of sound events. Mitigation targeted to the first part of the night is believed to be effective for the ability to fall asleep."

It should be noted that the noise indicators of the 1999 guidelines are L_{Aeq} and L_{Amax}, measured inside for continuous and non-continuous noise, respectively. The present night noise guidelines adopt an harmonized noise indicator as defined by Environmental Noise Directive (2002/49/EC): L_{night} measured outside, averaged over a year.

It should also be borne in mind that the 1999 guidelines are based on studies carried out up to 1995 (and a few meta-analyses some years later). Important new studies (Passchier-Vermeer et al., 2002; Basner et al., 2004) have become available since then, together with new insights into normal and disturbed sleep.

Comparing the above statement with the recommendations, it is clear that new information has made more precise statements possible. The thresholds are now known to be lower than L_{Amax} of 45 dB for a number of effects. The last three sentences still stand: there are good reasons for people to sleep with their windows open, and to prevent sleep disturbances one should consider the equivalent sound pressure level and the number of sound events. The present NNG allow responsible authorities and stakeholders to do this. Viewed in this way, the *Night noise guidelines for Europe* complements the 1999 guidelines. This means that the recommendations on government policy framework on noise management elaborated in the 1999 guidelines should be considered valid and relevant for the Member States to achieve the guideline values of this document.

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