# 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.

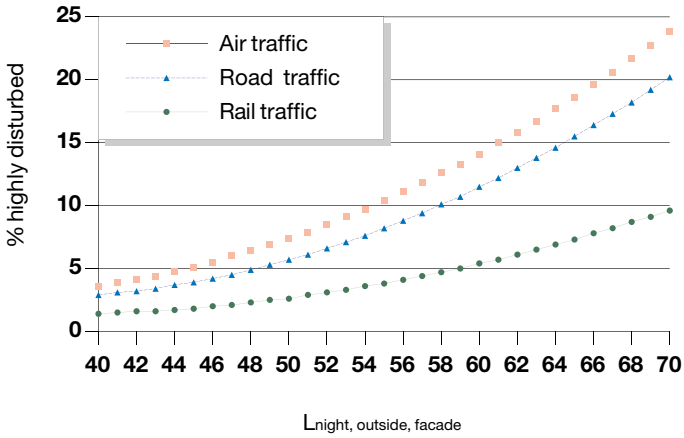



**Fig. 4.1**
**High sleep disturbance by noise at night**

*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 $L_{night}$.

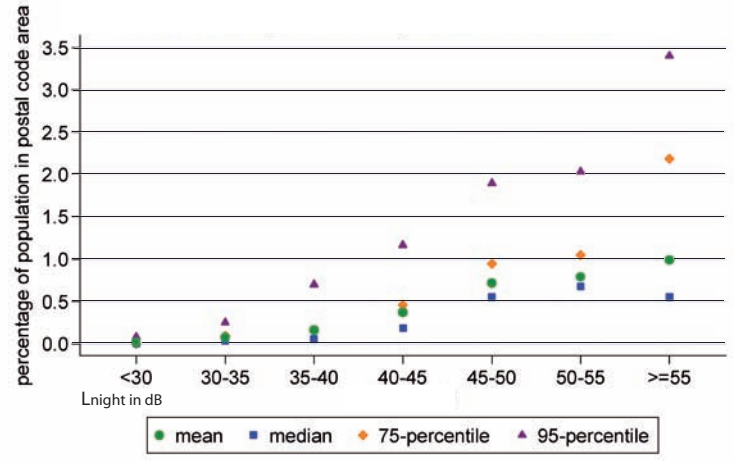



**Fig. 4.2**
**Dose-effect relation for persons having complained at least once during a year between 1994 and 2004**

*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

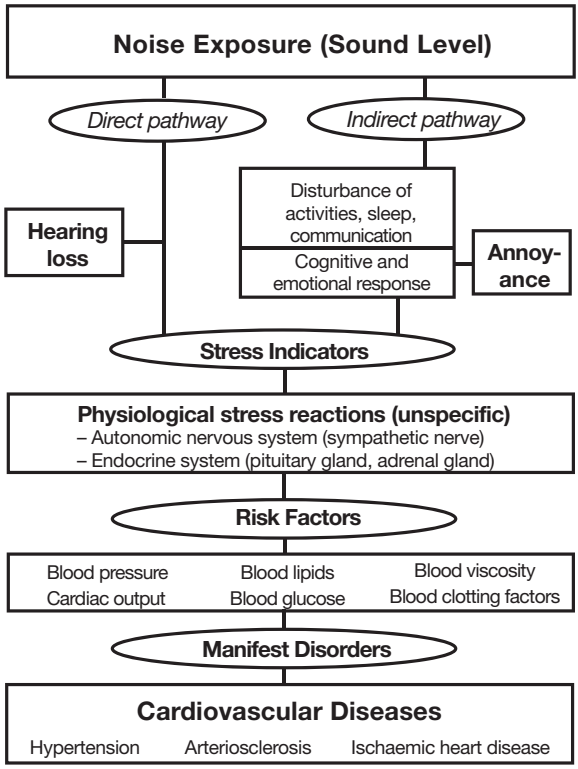



**Fig. 4.3**
**Noise effects reaction scheme**

*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

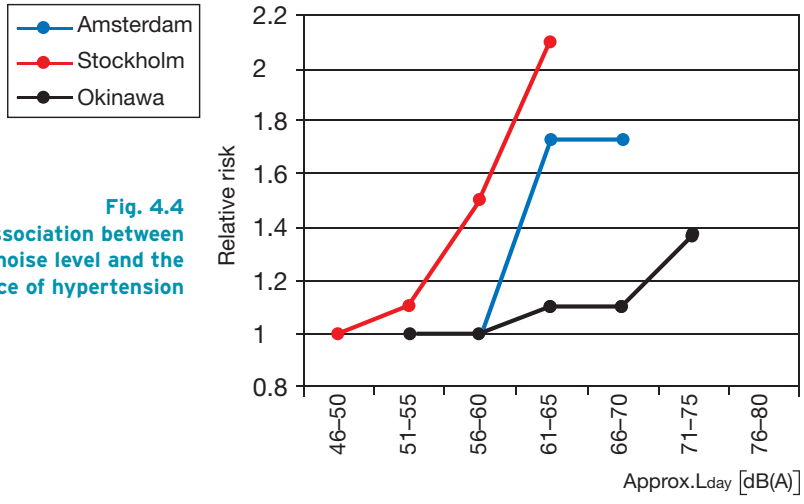



**Fig. 4.4**
**Association between aircraft noise level and the prevalence of hypertension**

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.

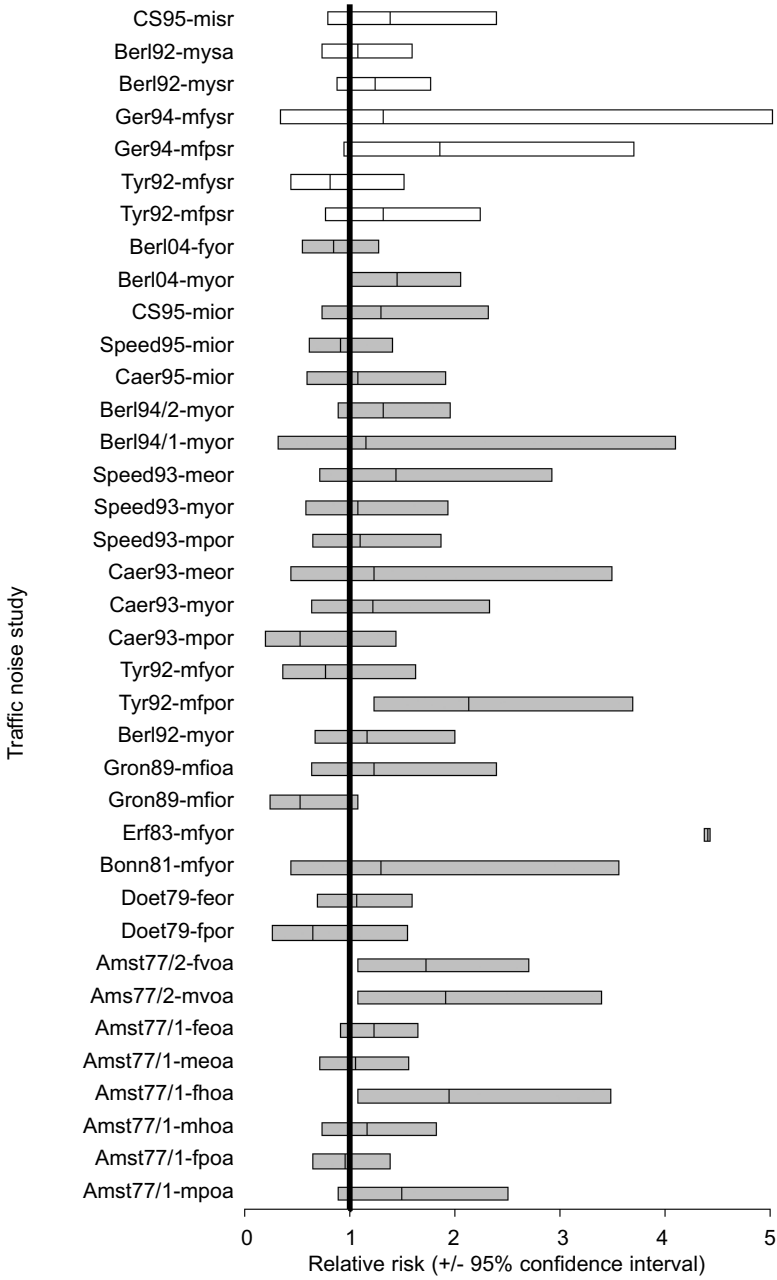



**Fig. 4.5**
**Results of epidemiological studies on the association between traffic noise and ischaemic heart disease**

***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

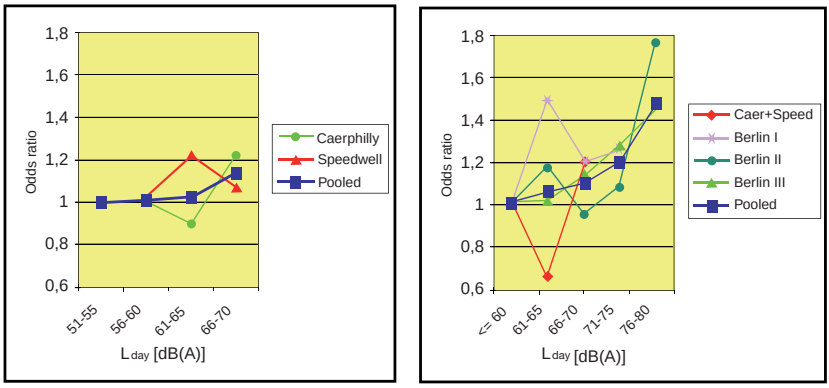
For a quantitative risk assessment and the derivation of guidelines for public health noise policy a common dose–response curve is required. The risk estimates obtained from different noise studies can be summarized using the statistical approach of a meta-analysis. Based on the judgement criteria discussed 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 (0.68-1.68),[18.92] | 1.46 (0.77-2.78),[9.27] | 4035 |
| Berlin III | 1.00 | 1.01 (0.77-1.32),[54.42] | 1.13 (0.86-1.49),[50.87] | 1.27 (0.88-1.84),[28.24] | --- | 4115 |
| Pooled | 1.00 | 1.05 (0.86-1.29) | 1.09 (0.90-1.34) | 1.19 (0.90-1.57) | 1.47 (0.79-2.76) | |
| Q-Test | | p=0.57 | p=0.87 | p=0.84 | p=0.90 | |

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).**

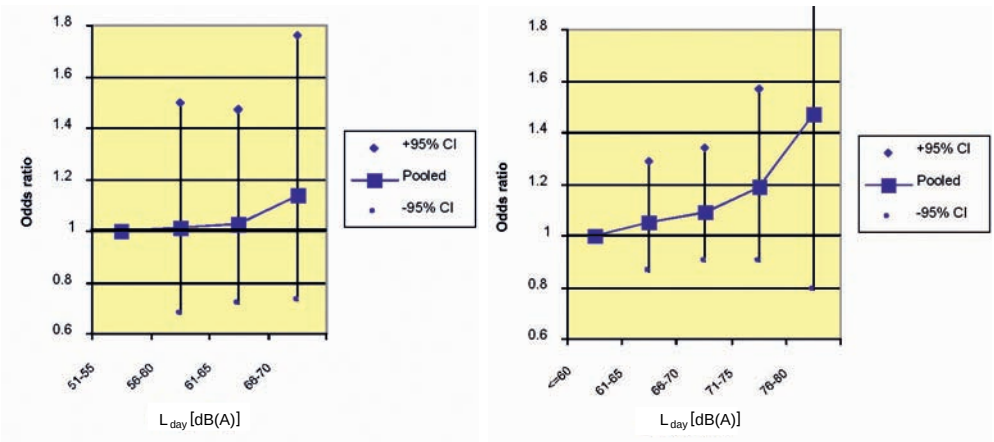



Fig. 4.8 and Fig. 4.9 show the two risk curves for descriptive and analytic studies (Hennekens and Buring, 1987). The graphs show the pooled effect estimates (odds ratios) and the 95% confidence intervals for each noise category. Whereas the cross-sectional studies (Fig. 4.9) cover the sound level range 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.