3. Adverse Health Effects Of Noise

3.1. Introduction

The perception of sounds in day-to-day life is of major importance for human well-being. Communication through speech, sounds from playing children, music, natural sounds in parklands, parks and gardens are all examples of sounds essential for satisfaction in every day life. Conversely, this document is related to the adverse effects of sound (noise). According to the International Programme on Chemical Safety (WHO 1994), an adverse effect of noise is defined as a change in the morphology and physiology of an organism that results in impairment of functional capacity, or an impairment of capacity to compensate for additional stress, or increases the susceptibility of an organism to the harmful effects of other environmental influences. This definition includes any temporary or long-term lowering of the physical, psychological or social functioning of humans or human organs. The health significance of noise pollution is given in this chapter under separate headings, according to the specific effects: noise-induced hearing impairment; interference with speech communication; disturbance of rest and sleep; psychophysiological, mental-health and performance effects; effects on residential behaviour and annoyance; as well as interference with intended activities. This chapter also considers vulnerable groups and the combined effects of sounds from different sources. Conclusions based on the details given in this chapter are given in Chapter 4 as they relate to guideline values.

3.2. Noise-Induced Hearing Impairment

Hearing impairment is typically defined as an increase in the threshold of hearing. It is assessed by threshold audiometry. Hearing handicap is the disadvantage imposed by hearing impairment sufficient to affect one’s personal efficiency in the activities of daily living. It is usually expressed in terms of understanding conventional speech in common levels of background noise (ISO 1990). Worldwide, noise-induced hearing impairment is the most prevalent irreversible occupational hazard. In the developing countries, not only occupational noise, but also environmental noise is an increasing risk factor for hearing impairment. In 1995, at the World Health Assembly, it was estimated that there are 120 million persons with disabling hearing difficulties worldwide (Smith 1998). It has been shown that men and women are equally at risk of noise-induced hearing impairment (ISO 1990; Berglund & Lindvall 1995).

Apart from noise-induced hearing impairment, hearing damage in populations is also caused by certain diseases; some industrial chemicals; ototoxic drugs; blows to the head; accidents; and hereditary origins. Deterioration of hearing capability is also associated with the aging process per se (presbyacusis). Present knowledge of the physiological effects of noise on the auditory system is based primarily on laboratory studies on animals. After noise exposure, the first morphological changes are usually found in the inner and outer hair cells of the cochlea, where the stereocilia become fused and bent. After more prolonged exposure, the outer and inner hair cells related to transmission of high-frequency sounds are missing. See Berglund & Lindvall (1995) for further discussion.

impairment in populations exposed to all types of noise (continuous, intermittent, impulse) during working hours. Noise exposure is characterized by LAeq over 8 hours (LAeq,8h). In the Standard, the relationships between LAeq,8h and noise-induced hearing impairment are given for frequencies of 500–6 000 Hz, and for exposure times of up to 40 years. These relations show that noise-induced hearing impairment occurs predominantly in the high-frequency range of 3 000–6 000 Hz, the effect being largest at 4 000 Hz. With increasing LAeq,8h and increasing exposure time, noise-induced hearing impairment also occurs at 2 000 Hz. But at LAeq,8h levels of 75 dBA and lower, even prolonged occupational noise exposure will not result in noise-induced hearing impairment (ISO 1990). This value is equal to that specified in 1980 by the World Health Organization (WHO 1980a).

The ISO Standard 1999 (ISO 1990) specifies hearing impairment in statistical terms (median values, and percentile fractions between 0.05 and 0.95). The extent of noise-induced hearing impairment in populations exposed to occupational noise depends on the value of LAeq,8h and the number of years of noise exposure. However, for high LAeq,8h values, individual susceptibility seems to have a considerable effect on the rate of progression of hearing impairment. For daily exposures of 8–16 h, noise-induced hearing impairment can be reasonably well estimated from LAeq,8h extrapolated to the longer exposure times (Axelsson et al. 1986). In this adaptation of LAeq,8h for daily exposures other than 8 hours, the equal energy principle is assumed to be applicable. For example, the hearing impairment due to a 16 h daily exposure is equivalent to that at LAeq,8h plus 3 dB (LAeq,16h = LAeq,8h + 10*log\(_{10}\) (16/8) = LAeq,8h + 3 dB). For a 24 h exposure, LAeq,24h = LAeq,8h + 10*log\(_{10}\) (24/8) = LAeq,8h + 5 dB).

Since the calculation method specified in the ISO Standard 1999 (ISO 1990) is the only universally adopted method for estimating occupational noise-induced hearing impairment, attempts have been made to assess whether the method is also applicable to hearing impairment due to environmental noise, including leisure-time noise. There is ample evidence that shooting noise, with LAeq,24h values of up to 80 dB, induces the same hearing impairment as an equivalent occupational noise exposure (Smoorenburg 1998). Moreover, noise-induced hearing impairment studies from motorbikes are also in agreement with results from ISO Standard 1999 (ISO 1990). Hearing impairment in young adults and children 12 years and older has been assessed by LAeq on a 24 h time basis, for a variety of environmental and leisure-time exposure patterns (e.g. Passchier-Vermeer 1993; HCN 1994). These include pop music in discotheques and concerts (Babisch & Ising 1989; ISO 1990); pop music through headphones (Ising et al. 1994; Struwe et al. 1996; Passchier-Vermeer et al. 1998); music played by brass bands and symphony orchestras (van Hees 1992). The results are in agreement with values predicted by the ISO Standard 1999 method on the basis of adjusted time.

In the publications cited above, exposure to noise with known characteristics, such as duration and level, was related to hearing impairment. In addition to these publications, there is also an extensive literature showing hearing impairment in populations exposed to specific types of non-occupational noise, although these exposures are not well characterized. These noises originate from shooting, motorcycling, snowmobile driving, playing in arcades, listening to music at concerts and through headphones, using noisy toys, and fireworks (e.g. Brookhouser et al. 1992; see also Berglund & Lindvall 1995). Although the characteristics of these exposures are to a certain extent unknown, the details in the publications suggest that LAeq,24h values of these
exposures exceed 70 dB.

In contrast, epidemiological studies failed to show hearing damage in populations exposed to an LAeq,24h of less than 70 dB (Lindemann et al. 1987). The data imply that even a lifetime exposure to environmental and leisure-time noise with an LAeq,24h <70 dBA would not cause hearing impairment in the large majority of people (over 95%). Overall, the results of many studies strongly suggest that the method from ISO Standard 1999 can also be used to estimate hearing impairment due to environmental and leisure-time noise, in addition to estimating the effects of occupational noise exposure.

Although the evidence suggests that the calculation method from ISO Standard 1999 (ISO 1990) should also be accepted for environmental and leisure time noise exposures, large-scale epidemiological studies of the general population do not exist to support this proposition. Taking into account the limitations of the studies, care should be taken with respect to the following aspects:

a. Data from animal experiments indicate that children may be more vulnerable in acquiring noise-induced hearing impairment than adults.

b. At very high instantaneous sound pressure levels, mechanical damage to the ear may occur (Hanner & Axelsson 1988). Occupational limits are set at peak sound pressure levels of 140 dB (EU 1986a). For adults exposed to environmental and leisure-time noise, this same limit is assumed to be valid. In the case of children, however, taking into account their habits while playing with noisy toys, peak sound pressure levels should never exceed 120 dB.

c. For shooting noise with LAeq,24h over 80 dB, studies on temporary threshold shift suggest the possibility of an increased risk for noise-induced hearing impairment (Smoorenburg 1998).

d. Risk for noise-induced hearing impairment may increase when the noise exposure is combined with exposure to vibrations, the use of ototoxic drugs, or some chemicals (Fechter 1999). In these circumstances, long-term exposure to LAeq,24h of 70 dBA may induce small hearing impairments.

e. It is uncertain whether the relationships between hearing impairment and noise exposure given in ISO Standard 1999 (ISO 1990) are applicable for environmental sounds of short rise time. For example, in the case of military low-altitude flying areas (75–300 m above ground) LAmx values of 110–130 dB occur within seconds after the onset of the sound.

Usually noise-induced hearing impairment is accompanied by an abnormal loudness perception which is known as loudness recruitment (cf. Berglund & Lindvall 1995). With a considerable loss of auditory sensitivity, some sounds may be perceived as distorted (paracusis). Another sensory effect that results from noise exposure is tinnitus (ringing in the ears). Commonly, tinnitus is referred to as sounds that are emitted by the inner ear itself (physiological tinnitus).
Tinnitus is a common and often disturbing accompaniment of occupational hearing impairment (Vernon and Moller 1995) and has become a risk for teenagers attending pop concerts and discotheques (Hetu & Fortin 1995; Passchier-Vermeer et al. 1998; Axelsson & Prasher 1999). Noise-induced tinnitus may be temporary, lasting up to 24 hours after exposure, or may have a more permanent character, such as after prolonged occupational noise exposure. Sometimes tinnitus is due to the sound produced by the blood flow through structures in the ear.

The main social consequence of hearing impairment is an inability to understand speech in daily living conditions, which is considered a severe social handicap. Even small values of hearing impairment (10 dB averaged over 2 000 and 4 000 Hz, and over both ears) may have an effect on the understanding of speech. When the hearing impairment exceeds 30 dB (again averaged over 2 000 and 4 000 Hz and both ears) a social hearing handicap is noticeable (cf. Katz 1994; Berglund & Lindvall 1995).

In the past, hearing protection has mainly emphasized occupational noise exposures at high values of LAeq,8h, or situations with high impulsive sounds. The near-universal adoption of an LAeq,8h value of 85 dB (or lower) as the limit for unprotected occupational noise exposure, together with requirements for personal hearing protection, has made cases of severe unprotected exposures more rare. This is particularly true for developed countries. However, monitoring of compliance and enforcement action for sound pressure levels just over the limits may be weak, especially in non-industrial environments in developed countries (Franks 1998), as well as in occupational and urban environments in developing countries (Smith 1998). Nevertheless, regulations for occupational noise exposure exist almost worldwide and exposures to occupational noise are to a certain extent under control.

On the other hand, environmental noise exposures due to a number of noisy activities, especially those during leisure-time activities of children and young adults, have scarcely been regulated. Given both the increasing number of noisy activities and the increasing exposure duration, such as loud music in cars and the use of Walkmen and Discmen, regulatory activities in this field are to be encouraged. Dose-response data are lacking for the general population. However, judging from the limited data for study groups (teenagers, young adults and women), and the assumption that time of exposure can be equated with sound energy, the risk for hearing impairment would be negligible for LAeq,24h values of 70 dBA over a lifetime. To avoid hearing impairment, impulse noise exposures should never exceed 140 dB peak sound pressure in adults, and 120 dB peak sound pressure in children.

3.3. Interference with Speech Communication

Noise interference with speech comprehension results in a large number of personal disabilities, handicaps and behavioural changes. Problems with concentration, fatigue, uncertainty and lack of self-confidence, irritation, misunderstandings, decreased working capacity, problems in human relations, and a number of stress reactions have all been identified (Lazarus 1998). Particularly vulnerable to these types of effects are the hearing impaired, the elderly, children in the process of language and reading acquisition, and individuals who are not familiar with the spoken language (e.g., Lazarus 1998). Thus, vulnerable persons constitute a substantial proportion of a country’s population.
Most of the acoustical energy of speech is in the frequency range 100–6,000 Hz, with the most important cue-bearing energy being between 300–3,000 Hz. Speech interference is basically a masking process in which simultaneous, interfering noise renders speech incapable of being understood. The higher the level of the masking noise, and the more energy it contains at the most important speech frequencies, the greater will be the percentage of speech sounds that become indiscernible to the listener. Environmental noise may also mask many other acoustical signals important for daily life, such as door bells, telephone signals, alarm clocks, fire alarms and other warning signals, and music (e.g., Edworthy & Adams 1996). The masking effect of interfering noise in speech discrimination is more pronounced for hearing-impaired persons than for persons with normal hearing, particularly if the interfering noise is composed of speech or babble.

As the sound pressure level of an interfering noise increases, people automatically raise their voice to overcome the masking effect upon speech (increase of vocal effort). This imposes an additional strain on the speaker. For example, in quiet surroundings, the speech level at 1 m distance averages 45–50 dBA, but is 30 dBA higher when shouting. However, even if the interfering noise is moderately loud, most of the sentences during ordinary conversation can still be understood fairly well. Nevertheless, the interpretation required for compensating the masking effect of the interfering sounds, and for comprehending what was said, imposes an additional strain on the listener. One contributing factor could be that speech spoken loudly is more difficult to understand than speech spoken softly, when compared at a constant speech-to-noise ratio (cf. Berglund & Lindvall 1995).

Speech levels vary between individuals because of factors such as gender and vocal effort. Moreover, outdoor speech levels decrease by about 6 dB for a doubling in the distance between talker and listener. Speech intelligibility in everyday living conditions is influenced by speech level, speech pronunciation, talker-to-listener distance, sound pressure levels, and to some extent other characteristics of interfering noise, as well as room characteristics (e.g. reverberation). Individual capabilities of the listener, such as hearing acuity and the level of attention of the listener, are also important for the intelligibility of speech. Speech communication is affected also by the reverberation characteristics of the room. For example, reverberation times greater than 1 s produce loss in speech discrimination. Longer reverberation times, especially when combined with high background interfering noise, make speech perception more difficult. Even in a quiet environment, a reverberation time below 0.6 s is desirable for adequate speech intelligibility by vulnerable groups. For example, for older hearing-handicapped persons, the optimal reverberation time for speech intelligibility is 0.3–0.5 s (Plomp 1986).

For complete sentence intelligibility in listeners with normal hearing, the signal-to-noise ratio (i.e. the difference between the speech level and the sound pressure level of the interfering noise) should be 15–18 dBA (Lazarus 1990). This implies that in smaller rooms, noise levels above 35 dBA interferes with the intelligibility of speech (Bradley 1985). Earlier recommendations suggested that sound pressure levels as high as 45 dBA would be acceptable (US EPA 1974). With raised voice (increased vocal effort) sentences may be 100% intelligible for noise levels of up to 55 dBA; and sentences spoken with straining vocal effort can be 100% intelligible with noise levels of about 65 dBA. For speech to be intelligible when listening to complicated
messages (at school, listening to foreign languages, telephone conversation), it is recommended that the signal-to-noise ratio should be at least 15 dBA. Thus, with a speech level of 50 dBA, (at 1 m distance this level corresponds to a casual speech level of both women and men), the sound pressure level of interfering noise should not exceed 35 dBA. For vulnerable groups even lower background levels are needed. If it is not possible to meet the strictest criteria for vulnerable persons in sensitive situations (e.g. in classrooms), one should strive for as low background levels as possible.

3.4. Sleep Disturbance

Uninterrupted sleep is known to be a prerequisite for good physiological and mental functioning of healthy persons (Hobson 1989); sleep disturbance, on the other hand, is considered to be a major environmental noise effect. It is estimated that 80-90% of the reported cases of sleep disturbance in noisy environments are for reasons other than noise originating outdoors. For example, sanitary needs; indoor noises from other occupants; worries; illness; and climate (e.g. Reyner & Horne 1995). Our understanding of the impact of noise exposure on sleep stems mainly from experimental research in controlled environments. Field studies conducted with people in their normal living situations are scarce. Most of the more recent field research on sleep disturbance has been conducted for aircraft noise (Fidell et al. 1994 1995a,b 1998; Horne et al. 1994 1995; Maschke et al. 1995 1996; Ollerhead et al. 1992; Passchier-Vermeer 1999). Other field studies have examined the effects of road traffic and railway noise (Griefahn et al. 1996 1998).

The primary sleep disturbance effects are: difficulty in falling asleep (increased sleep latency time); awakenings; and alterations of sleep stages or depth, especially a reduction in the proportion of REM-sleep (REM = rapid eye movement) (Hobson 1989). Other primary physiological effects can also be induced by noise during sleep, including increased blood pressure; increased heart rate; increased finger pulse amplitude; vasoconstriction; changes in respiration; cardiac arrhythmia; and an increase in body movements (cf. Berglund & Lindvall 1995). For each of these physiological effects, both the noise threshold and the noise-response relationships may be different. Different noises may also have different information content and this also could affect physiological threshold and noise-response relationships (Edworthy 1998).

Exposure to night-time noise also induces secondary effects, or so-called after effects. These are effects that can be measured the day following the night-time exposure, while the individual is awake. The secondary effects include reduced perceived sleep quality; increased fatigue; depressed mood or well-being; and decreased performance (Öhrström 1993a; Passchier-Vermeer 1993; Carter 1996; Pearsons et al. 1995; Pearsons 1998).

Long-term effects on psychosocial well-being have also been related to noise exposure during the night (Öhrström 1991). Noise annoyance during the night-time increased the total noise annoyance expressed by people in the following 24 h. Various studies have also shown that people living in areas exposed to night-time noise have an increased use of sedatives or sleeping pills. Other frequently reported behavioural effects of night-time noise include closed bedroom windows and use of personal hearing protection. Sensitive groups include the elderly, shift workers, persons especially vulnerable to physical or mental disorders and other individuals with
sleeping difficulties.

Questionnaire data indicate the importance of night-time noise on the perception of sleep quality. A recent Japanese investigation was conducted for 3,600 women (20–80 years old) living in eight roadside zones with different road traffic noise. The results showed that four measures of perceived sleep quality (difficulty in falling asleep; waking up during sleep; waking up too early; feelings of sleeplessness one or more days a week) correlated significantly with the average traffic volumes during night-time. An in-depth investigation of 19 insomnia cases and their matched controls (age, work) measured outdoor and indoor sound pressure levels during sleep (Kageyama et al. 1997). The study showed that road traffic noise in excess of 30 dB LAeq for nighttime induced sleep disturbance, consistent with the results of Öhrström (1993b).

Meta-analyses of field and laboratory studies have suggested that there is a relationship between the SEL for a single night-time noise event and the percentage of people awakened, or who showed sleep stage changes (e.g., Ollerhead et al. 1992; Passchier-Vermeer 1993; Finegold et al. 1994; Pearsons et al. 1995). All of these studies assumed that the number of awakenings per night for each SEL value is proportional to the number of night-time noise events. However, the results have been criticized for methodological reasons. For example, there were small groups of sleepers; too few original studies; and indoor exposure was estimated from outdoor sound pressure levels (NRC-CNRC 1994; Beersma & Altena 1995; Vallet 1998). The most important result of the meta-analyses is that there is a clear difference in the dose-response curves for laboratory and field studies, and that noise has a lower effect under real-life conditions (Pearsons et al. 1995; Pearsons 1998).

However, this result has been questioned, because the studies were not controlled for such things as the sound insulation of the buildings, and the number of bedrooms with closed windows. Also, only two indicators of sleep disturbance were considered (awakening and sleep stage changes). The meta-analyses thus neglected other important sleep disturbance effects (Öhrström 1993b; Carter et al. 1994a; Carter et al. 1994b; Carter 1996; Kuwano et al. 1998). For example, for road traffic noise, perceived sleep quality is related both to the time needed to fall asleep and the total sleep time (Öhrström & Björkman 1988). Individuals who are more sensitive to noise (as assessed by different questionnaires) report worse sleep quality both in field studies and in laboratory studies.

A further criticism of the meta-analyses is that laboratory experiments have shown that habituation to night-time noise events occurs, and that noise-induced awakening decreases with increasing number of sound exposures per night. This is in contrast to the assumption used in the meta-analyses, that the percentage of awakenings is linearly proportional to the number of night-time noise events. Studies have also shown that the frequency of noise-induced awakenings decreases for at least the first eight consecutive nights. So far, habituation has been shown for awakenings, but not for heart rate and after effects such as perceived sleep quality, mood and performance (Öhrström and Björkman 1988).

Other studies suggest that it is the difference in sound pressure levels between a noise event and background, rather than the absolute sound pressure level of the noise event, that determines the reaction probability. The time interval between two noise events also has an important influence.
of the probability of obtaining a response (Griefahn 1977; cf. Berglund & Lindvall 1995). Another possible factor is the person’s age, with older persons having an increased probability of awakening. However, one field study showed that noise-induced awakenings are independent of age (Reyner & Horne 1995).

For a good sleep, it is believed that indoor sound pressure levels should not exceed approximately 45 dB LAmax more than 10–15 times per night (Vallet & Vernet 1991), and most studies show an increase in the percentage of awakenings at SEL values of 55–60 dBA (Passchier-Vermeer 1993; Finegold et al. 1994; Pearsons et al. 1995). For intermittent events that approximate aircraft noise, with an effective duration of 10–30 s, SEL values of 55–60 dBA correspond to a LAmax value of 45 dB. Ten to 15 of these events during an eight-hour night-time implies an LAeq,8h of 20–25 dB. This is 5–10 dB below the LAeq,8h of 30 dB for continuous night-time noise exposure, and shows that the intermittent character of noise has to be taken into account when setting night-time limits for noise exposure. For example, this can be achieved by considering the number of noise events and the difference between the maximum sound pressure level and the background level of these events.

Special attention should also be given to the following considerations:

a. Noise sources in an environment with a low background noise level. For example, night-traffic in suburban residential areas.

b. Environments where a combination of noise and vibrations are produced. For example, railway noise, heavy duty vehicles.

c. Sources with low-frequency components. Disturbances may occur even though the sound pressure level during exposure is below 30 dBA.

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

Epidemiological and laboratory studies involving workers exposed to occupational noise, and general populations (including children) living in noisy areas around airports, industries and noisy streets, indicate that noise may have both temporary and permanent impacts on physiological functions in humans. It has been postulated that noise acts as an environmental stressor (for a review see Passchier-Vermeer 1993; Berglund & Lindvall 1995). Acute noise exposures activate the autonomic and hormonal systems, leading to temporary changes such as increased blood pressure, increased heart rate and vasoconstriction. After prolonged exposure, susceptible individuals in the general population may develop permanent effects, such as hypertension and ischaemic heart disease associated with exposures to high sound pressure levels (for a review see Passchier-Vermeer 1993; Berglund & Lindvall 1995). The magnitude and duration of the effects are determined in part by individual characteristics, lifestyle behaviours and environmental conditions. Sounds also evoke reflex responses, particularly when they are unfamiliar and have a sudden onset.

Laboratory experiments and field quasi-experiments show that if noise exposure is temporary, the physiological system usually returns - after the exposure terminates - to a normal (pre-exposure) state within a time in the range of the exposure duration. If the exposure is of sufficient intensity and unpredictability, cardiovascular and hormonal responses may appear, including increases in heart rate and peripheral vascular resistance; changes in blood pressure, blood viscosity and blood lipids; and shifts in electrolyte balance (Mg/Ca) and hormonal levels (epinephrine, norepinephrine, cortisol). The first four effects are of interest because of noise-related coronary heart disease (Ising & Günther 1997). Laboratory and clinical data suggest that noise may significantly elevate gastrointestinal motility in humans.

By far the greatest number of occupational and community noise studies have focused on the possibility that noise may be a risk factor for cardiovascular disease. Many studies in occupational settings have indicated that workers exposed to high levels of industrial noise for 5–30 years have increased blood pressure and statistically significant increases in risk for hypertension, compared to workers in control areas (Passchier-Vermeer 1993). In contrast, only a few studies on environmental noise have shown that populations living in noisy areas around airports and on noisy streets have an increased risk for hypertension. The overall evidence suggests a weak association between long-term environmental noise exposure and hypertension (HCN 1994; Berglund & Lindvall 1995; IEH 1997), and no dose-response relationships could be established.

Recently, an updated summary of available studies for ischaemic heart disease has been presented (Babisch 1998a; Babisch 1998b; Babisch et al. 1999; see also Thompson 1996). The studies reviewed include case-control and cross-sectional designs, as well as three longitudinal studies. However, it has not yet been possible to conduct the most advanced quantitative integrated analysis of the available studies. Relative risks and their confidence intervals could be estimated only for the classes of high noise levels (mostly >65 dBA during daytime) and low levels (mostly <55 dBA during daytime), rather than a range of exposure levels. For methodological reasons identified in the meta-analysis, a cautious interpretation of the results is warranted (Lercher et al. 1998).
Prospective studies that controlled for confounding factors suggest an increase in ischaemic heart disease when the noise levels exceed 65–70 dB for LAeq (6–22). (For road traffic noise, the difference between LAeq (6-22h) and LAeq,24h usually is of the order of 1.5 dB). When orientation of the bedroom, window opening habits and years of exposure are taken into account, the risk of heart disease is slightly higher (Babisch et al. 1998; Babisch et al. 1999). However, disposition, behavioural and environmental factors were not sufficiently accounted for in the analyses carried out to date. In epidemiological studies the lowest level at which traffic noise had an effect on ischaemic heart disease was 70 dB for LAeq,24h (HCN 1994).

The overall conclusion is that cardiovascular effects are associated with long-term exposure to LAeq,24h values in the range of 65–70 dB or more, for both air- and road-traffic noise. However, the associations are weak and the effect is somewhat stronger for ischaemic heart disease than for hypertension. Nevertheless, such small risks are potentially important because a large number of persons are currently exposed to these noise levels, or are likely to be exposed in the future. Furthermore, only the average risk is considered and sensitive subgroups of the populations have not been sufficiently characterized. For example, a 10% increase in risk factors (a relative risk of 1.1) may imply an increase of up to 200 cases per 100 000 people at risk per year. Other observed psychophysiological effects, such as changes in stress hormones, magnesium levels, immunological indicators, and gastrointestinal disturbances are too inconsistent for conclusions to be drawn about the influence of noise pollution.

3.6. Mental Health Effects

Mental health is defined as the absence of identifiable psychiatric disorders according to current norms (Freeman 1984). Environmental noise is not believed to be a direct cause of mental illness, but it is assumed that it accelerates and intensifies the development of latent mental disorder. Studies on the adverse effects of environmental noise on mental health cover a variety of symptoms, including anxiety; emotional stress; nervous complaints; nausea; headaches; instability; argumentativeness; sexual impotency; changes in mood; increase in social conflicts, as well as general psychiatric disorders such as neurosis, psychosis and hysteria. Large-scale population studies have suggested associations between noise exposure and a variety of mental health indicators, such as single rating of well-being; standard psychological symptom profiles; the intake of psychotropic drugs; and consumption of tranquilizers and sleeping pills. Early studies showed a weak association between exposure to aircraft noise and psychiatric hospital admissions in the general population surrounding an airport (see also Berglund & Lindvall 1995). However, the studies have been criticized because of problems in selecting variables and in response bias (Halpern 1995).

Exposure to high levels of occupational noise has been associated with development of neurosis and irritability; and exposure to high levels of environmental noise with deteriorated mental health (Stansfeld 1992). However, the findings on environmental noise and mental health effects are inconclusive (HCN 1994; Berglund & Lindvall 1995; IEH 1997). The only longitudinal study in this field (Stansfeld et al. 1996) showed an association between the initial level of road traffic noise and minor psychiatric disorders, although the association for increased anxiety was weak and non-linear. It turned out that psychiatric disorders are associated with noise sensitivity,
rather than with noise exposure, and the association was found to disappear after adjustment for baseline trait anxiety. These and other results show the importance of taking vulnerable groups into account, because they may not be able to cope sufficiently with unwanted environmental noise (e.g. Stansfeld 1992). This is particularly true of children, the elderly and people with preexisting illnesses, especially depression (IEH 1997). Despite the weaknesses of the various studies, the possibility that community noise has adverse effects on mental health is suggested by studies on the use of medical drugs, such as tranquilizers and sleeping pills, on psychiatric symptoms and on mental hospital admission rates.

3.7. The Effects of Noise on Performance

It has been documented in both laboratory subjects and in workers exposed to occupational noise, that noise adversely affects cognitive task performance. In children, too, environmental noise impairs a number of cognitive and motivational parameters (Cohen et al. 1980; Evans & Lepore 1993; Evans 1998; Hygge et al. 1998; Haines et al. 1998). However, there are no published studies on whether environmental noise at home also impairs cognitive performance in adults. Accidents may also be an indicator of performance deficits. The few field studies on the effects of noise on performance and safety showed that noise may produce some task impairment and increase the number of errors in work, but the effects depend on the type of noise and the task being performed (Smith 1990).

Laboratory and workplace studies showed that noise can act as a distracting stimulus. Also, impulsive noise events (e.g. sonic booms) may produce disruptive effects as a result of startle responses. In the short term, noise-induced arousal may produce better performance of simple tasks, but cognitive performance deteriorates substantially for more complex tasks (i.e. tasks that require sustained attention to details or to multiple cues; or tasks that demand a large capacity of working memory, such as complex analytical processes). Some of the effects are related to loss in auditory comprehension and language acquisition, but others are not (Evans & Maxwell 1997). Among the cognitive effects, reading, attention, problem solving and memory are most strongly affected by noise. The observed effects on motivation, as measured by persistence with a difficult cognitive task, may either be independent or secondary to the aforementioned cognitive impairments.

Two types of memory deficits have been identified under experimental noise exposure: incidental memory and memory for materials that the observer was not explicitly instructed to focus on during a learning phase. For example, when presenting semantic information to subjects in the presence of noise, recall of the information content was unaffected, but the subjects were significantly less able to recall, for example, in which corner of the slide a word had been located. There is also some evidence that the lack of “helping behavior” that was noted under experimental noise exposure may be related to inattention to incidental cues (Berglund & Lindvall 1995). Subjects appear to process information faster in working memory during noisy performance conditions, but at a cost of available memory capacity. For example, in a running memory task, in which subjects were required to recall in sequence letters that they had just heard, subjects recalled recent items better under noisy conditions, but made more errors farther back into the list.
Experimental noise exposure consistently produces negative after-effects on performance (Glass & Singer 1972). Following exposure to aircraft noise, schoolchildren in the vicinity of Los Angeles airport were found to be deficient in proofreading, and in persistence with challenging puzzles (Cohen et al. 1980). The uncontrollability of noise, rather than the intensity of the noise, appears to be the most critical variable. The only prospective study on noise-exposed schoolchildren, designed around the move of the Munich airport (Hygge et al. 1996; Evans et al. 1998), confirmed the results of laboratory and workplace studies in adults, as well the results of the Los Angeles airport study with children (Cohen et al. 1980). An important finding was that some of the adaptation strategies for dealing with aircraft noise, such as tuning out or ignoring the noise, and the effort necessary to maintain task performance, come at a price. There is heightened sympathetic arousal, as indicated by increased levels of stress hormone, and elevation of resting blood pressure (Evans et al. 1995; Evans et al. 1998). Notably, in the airport studies reported above, the adverse effects were larger in children with lower school achievement.

For aircraft noise, it has been shown that chronic exposure during early childhood appears to impair reading acquisition and reduces motivational capabilities. Of recent concern are concomitant psychophysiological changes (blood pressure and stress hormone levels). Evidence indicates that the longer the exposure, the greater the damage. It seems clear that daycare centers and schools should not be located near major sources of noise, such as highways, airports and industrial sites.

3.8. Effects of Noise on Residential Behaviour and Annoyance

Noise annoyance is a global phenomenon. A definition of annoyance is “a feeling of displeasure associated with any agent or condition, known or believed by an individual or group to adversely affect them” (Lindvall & Radford 1973; Koelega 1987). However, apart from “annoyance”, people may feel a variety of negative emotions when exposed to community noise, and may report anger, disappointment, dissatisfaction, withdrawal, helplessness, depression, anxiety, distraction, agitation, or exhaustion (Job 1993; Fields et al. 1997 1998). Thus, although the term annoyance does not cover all the negative reactions, it is used for convenience in this document.

Noise can produce a number of social and behavioural effects in residents, besides annoyance (for review see Berglund & Lindvall 1995). The social and behavioural effects are often complex, subtle and indirect. Many of the effects are assumed to be the result of interactions with a number of non-auditory variables. Social and behavioural effects include changes in overt everyday behaviour patterns (e.g. closing windows, not using balconies, turning TV and radio to louder levels, writing petitions, complaining to authorities); adverse changes in social behaviour (e.g. aggression, unfriendliness, disengagement, non-participation); adverse changes in social indicators (e.g. residential mobility, hospital admissions, drug consumption, accident rates); and changes in mood (e.g. less happy, more depressed).

Although changes in social behaviour, such as a reduction in helpfulness and increased aggressiveness, are associated with noise exposure, noise exposure alone is not believed to be sufficient to produce aggression. However, in combination with provocation or pre-existing anger or hostility, it may trigger aggression. It has also been suspected that people are less willing to help, both during exposure and for a period after exposure. Fairly consistent evidence
shows that noise above 80 dBA is associated with reduced helping behaviour and increased aggressive behaviour. Particularly, there is concern that high-level continuous noise exposures may contribute to the susceptibility of schoolchildren to feelings of helplessness (Evans & Lepore 1993).

The effects of community noise can be evaluated by assessing the extent of annoyance (low, moderate, high) among exposed individuals; or by assessing the disturbance of specific activities, such as reading, watching television and communication. The relationship between annoyance and activity disturbances is not necessarily direct and there are examples of situations where the extent of annoyance is low, despite a high level of activity disturbance. For aircraft noise, the most important effects are interference with rest, recreation and watching television. This is in contrast to road traffic noise, where sleep disturbance is the predominant effect (Berglund & Lindvall 1995).

A number of studies have shown that equal levels of traffic and industrial noises result in different magnitudes of annoyance (Hall et al. 1981; Griffiths 1983; Miedema 1993; Bradley 1994a; Miedema & Vos 1998). This has led to criticism (e.g. Kryter 1994; Bradley 1994a) of averaged dose-response curves determined by meta-analysis, which assumed that all traffic noises are the same (Fidell et al. 1991; Fields 1994a; Finegold et al. 1994). Schultz (1978) and Miedema & Vos (1998) have synthesized curves of annoyance associated with three types of traffic noise (road, air, railway). In these curves, the percentage of people highly or moderately annoyed was related to the day and night continuous equivalent sound level, $L_{dn}$. For each of the three types of traffic noise, the percentage of highly annoyed persons in a population started to increase at an $L_{dn}$ value of 42 dBA, and the percentage of moderately annoyed persons at an $L_{dn}$ value of 37 dBA (Miedema & Vos 1998). Aircraft noise produced a stronger annoyance response than road traffic, for the same $L_{dn}$ exposure, consistent with earlier analyses (Kryter 1994; Bradley 1994a). However, caution should be exercised when interpreting synthesized data from different studies, since five major parameters should be randomly distributed for the analyses to be valid: personal, demographic, and lifestyle factors, as well as the duration of noise exposure and the population experience with noise (Kryter 1994).

Annoyance in populations exposed to environmental noise varies not only with the acoustical characteristics of the noise (source, exposure), but also with many non-acoustical factors of social, psychological, or economic nature (Fields 1993). These factors include fear associated with the noise source, conviction that the noise could be reduced by third parties, individual noise sensitivity, the degree to which an individual feels able to control the noise (coping strategies), and whether the noise originates from an important economic activity. Demographic variables such as age, sex and socioeconomic status, are less strongly associated with annoyance. The correlation between noise exposure and general annoyance is much higher at the group level than at the individual level, as might be expected. Data from 42 surveys showed that at the group level about 70% of the variance in annoyance is explained by noise exposure characteristics, whereas at the individual level it is typically about 20% (Job 1988).

When the type and amount of noise exposure is kept constant in the meta-analyses, differences between communities, regions and countries still exist (Fields 1990; Bradley 1996). This is well demonstrated by a comparison of the dose-response curve determined for road-traffic noise.
Stronger reactions have been observed when noise is accompanied by vibrations and contains low frequency components (Paulsen & Kastka 1995; Öhrström 1997; for review see Berglund et al. 1996), or when the noise contains impulses, such as shooting noise (Buchta 1996; Vos 1996; Smoorenburg 1998). Stronger, but temporary, reactions also occur when noise exposure is increased over time, in comparison to situations with constant noise exposure (e.g. HCN 1997; Klæboe et al. 1998). Conversely, for road traffic noise, the introduction of noise protection barriers in residential areas resulted in smaller reductions in annoyance than expected for a stationary situation (Kastka et al. 1995).

To obtain an indicator for annoyance, other methods of combining parameters of noise exposure have been extensively tested, in addition to metrics such as $\text{LA}_{eq,24h}$ and $L_{dn}$. When used for a set of community noises, these indicators correlate well both among themselves and with $\text{LA}_{eq,24h}$ or $L_{dn}$ values (e.g. HCN 1997). Although $\text{LA}_{eq,24h}$ and $L_{dn}$ are in most cases acceptable approximations, there is a growing concern that all the component parameters of the noise should be individually assessed in noise exposure investigations, at least in the complex cases (Berglund & Lindvall 1995).

### 3.9. The Effects of Combined Noise Sources

Many acoustical environments consist of sounds from more than one source. For these environments, health effects are associated with the total noise exposure, rather than with the noise from a single source (WHO 1980b). When considering hearing impairment, for example, the total noise exposure can be expressed in terms of $\text{LA}_{eq,24h}$ for the combined sources. For other adverse health effects, however, such a simple model most likely will not apply. It is possible that some disturbances (e.g. speech interference, sleep disturbance) may more easily be attributed to specific noises. In cases where one noise source clearly dominates, the magnitude of an effect may be assessed by taking into account the dominant source only (HCN 1997). Furthermore, at a policy level, there may be little need to identify the adverse effect of each specific noise, unless the responsibility for these effects is to be shared among several polluters (cf. The Polluter Pays Principle in Chapter 5, UNCED 1992).

There is no consensus on a model for assessing the total annoyance due to a combination of environmental noise sources. This is partly due to a lack of research into the temporal patterns of combined noises. The current approach for assessing the effects of “mixed noise sources” is limited to data on “total annoyance” transformed to mathematical principles or rules of thumb (Ronnebaum et al. 1996; Vos 1992; Miedema 1996; Berglund & Nilsson 1997). Models to assess the total annoyance of combinations of environmental noises may not be applicable to those health effects for which the mechanisms of noise interaction are unknown, and for which different cumulative or synergistic effects cannot be ruled out. When noise is combined with different types of environmental agents, such as vibrations, ototoxic chemicals, or chemical odours, again there is insufficient knowledge to accurately assess the combined effects on health.
Therefore, caution should be exercised when trying to predict the adverse health effects of combined factors in residential populations.

The evidence on low-frequency noise is sufficiently strong to warrant immediate concern. Various industrial sources emit continuous low-frequency noise (compressors, pumps, diesel engines, fans, public works); and large aircraft, heavy-duty vehicles and railway traffic produce intermittent low-frequency noise. Low-frequency noise may also produce vibrations and rattles as secondary effects. Health effects due to low-frequency components in noise are estimated to be more severe than for community noises in general (Berglund et al. 1996). Since A-weighting underestimates the sound pressure level of noise with low-frequency components, a better assessment of health effects would be to use C-weighting.

In residential populations heavy noise pollution will most certainly be associated with a combination of health effects. For example, cardiovascular disease, annoyance, speech interference at work and at home, and sleep disturbance. Therefore, it is important that the total adverse health load over 24 hours be considered and that the precautionary principle for sustainable development is applied in the management of health effects (see Chapter 5).

**3.10. Vulnerable Groups**

Protective standards are essentially derived from observations on the health effects of noise on “normal” or “average” populations. The participants of these investigations are selected from the general population and are usually adults. Sometimes, samples of participants are selected because of their easy availability. However, vulnerable groups of people are typically underrepresented. This group includes people with decreased personal abilities (old, ill, or depressed people); people with particular diseases or medical problems; people dealing with complex cognitive tasks, such as reading acquisition; people who are blind or who have hearing impairment; fetuses, babies and young children; and the elderly in general (Jansen 1987; AAP 1997). These people may be less able to cope with the impacts of noise exposure and be at greater risk for harmful effects.

Persons with impaired hearing are the most adversely affected with respect to speech intelligibility. Even slight hearing impairments in the high-frequency range may cause problems with speech perception in a noisy environment. From about 40 years of age, people typically demonstrate an impaired ability to understand difficult, spoken messages with low linguistic redundancy. Therefore, based on interference with speech perception, a majority of the population belongs to the vulnerable group.

Children have also been identified as vulnerable to noise exposure (see Agenda 21: UNCED 1992). The evidence on noise pollution and children’s health is strong enough to warrant monitoring programmes at schools and preschools to protect children from the effects of noise. Follow up programmes to study the main health effects of noise on children, including effects on speech perception and reading acquisition, are also warranted in heavily noise polluted areas (Cohen et al. 1986; Evans et al. 1998).
The issue of vulnerable subgroups in the general population should thus be considered when developing regulations or recommendations for the management of community noise. This consideration should take into account the types of effects (communication, recreation, annoyance, etc.), specific environments (in utero, incubator, home, school, workplace, public institutions, etc.) and specific lifestyles (listening to loud music through headphones, or at discotheques and festivals; motor cycling, etc.).