

Noise Exposure and Public Health

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Exposure to noise constitutes a health risk. There is sufficient scientific evidence that noise exposure can induce hearing impairment, hypertension and ischemic heart disease, annoyance, sleep disturbance, and decreased school performance. For other effects such as changes in the immune system and birth defects, the evidence is limited. Most public health impacts of noise were already identified in the 1960s and noise abatement is less of a scientific but primarily a policy problem. A subject for further research is the elucidation of the mechanisms underlying noise-induced cardiovascular disorders and the relationship of noise with annoyance and nonacoustical factors modifying health outcomes. A high priority study subject is the effects of noise on children, including cognitive effects and their reversibility. Noise exposure is on the increase, especially in the general living environment, both in industrialized nations and in developing world regions. This implies that in the twenty-first century noise exposure will still be a major public health problem. **Key words:** annoyance, cardiovascular effects, children's health, environmental health, environmental noise, hearing impairment, noise exposure, noise metrics, occupational noise, performance. — *Environ Health Perspect* 108(suppl 1):123–131 (2000).

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Noise Exposure and Health

Assessment of the human health risk associated with the presence of a xenobiotic substance in the environment usually follows the relatively simple scheme depicted in Figure 1. The substance occurs in environmental media at certain concentrations, depending on, among other factors, lifestyle, residence time, and dietary habits, that people may be exposed to this xenobiotic. Any subsequent harm depends on the level of exposure. The modifying impact of exogenous determinants and personal characteristics on the level of exposure and sensitivity with respect to the toxic action usually are not taken into account or are only considered in a standardized way, at least for risk assessment and standard-setting purposes. The data available, often derived from animal experiments and surveys of population behavior, do not allow a more refined analysis.

In the case of the assessment of the health effects of noise exposure, the scheme in Figure 1 is too simple. With the exception of damage to the hearing organ, the exposed organism's reaction to the perception of sound is strongly dependent on the context of the exposure. The effects of noise exposure cannot be understood only by taking mechanisms of toxic action into account. For example, the sounds in a discotheque are music to the dancers but noise to the neighbors. In the first case, the exposure would not be annoying but is expected to contribute to hearing loss; for the neighbors, hearing loss would be improbable, but annoyance would certainly occur. A conceptual model to address the health effects of noise exposure is presented in Figure 2.

The model considers effects on health and quality of life as the outcome of a processing of exogenous determinants or environmental factors—in this case, noise exposure. Exposure, processing, and effect take place within economic and social environments and all are modified by societal factors. Furthermore, lifestyle and concurrent exposure to other factors play a role. An example of the former was given above. An example of the latter is the finding that the perceived presence of the risk of an aircraft crash has been found to augment annoyance (and vice versa) (1). This processing of sounds is influenced by the genetic and acquired characteristics of the organism. For example, some people have a specific sensitivity to noise and will be more susceptible to one or all of its effects than other people. Examples of societal factors that determine the adverse effects associated with noise exposure are insulation of houses, noise level-related depreciation of house prices, and individual and societal appreciation of the activities generating the noise.

These insights, as depicted in the model in Figure 2, are not new. On the contrary, it is striking that in the 1960s most of the effects of sound on health and quality of life were already known or at least hypothesized, including the variety of modifying factors referred to above (2). In the 1970s the research results were sufficiently reviewed to allow science-based recommendations to be made for policy measures to protect public health (3–5). In the last three decades new data have confirmed the earlier insights and, as reviewed here in our present paper, have made more precise assessments of exposure–response relationships and observation thresholds possible. Many of the

newer data stem from epidemiologic studies. If politicians had taken a more protective stance in the 1970s—which would have been legitimate on the basis of the then-available data—this review probably would have been superfluous, as new data would not have been published, but also harm would have been avoided.

In this review we emphasize development of insights into the effects of noise exposure on health and quality of life. In accordance with the relevant literature, we use the term noise to represent sounds generated by sources in the environment (indoors, outdoors, at work, etc.); noise is often appreciated negatively. Because several reviews on this subject have been published recently—some by international groups of scientists (6)—we refer to original research papers only when necessary for our argument. Furthermore, given our background in environmental health sciences, we also discuss policy instruments for health protection.

Characterization of Noise Exposure

Sound Pressure Level and Sound Level

Sound is a physical phenomenon consisting of alternating compression and expansion of air that propagate in all directions from a source. These alternating compressions and expansions can be described as small changes in pressure around atmospheric pressure. The frequency of the alternations determines the pitch of a sound: a high-pitched tone (e.g., 4,000 Hz) has a squeaking sound; a low-pitched tone (e.g., 200 Hz), a humming sound. The environmental noise sources discussed in this review usually generate sounds within a broad frequency range. Sound pressures, relative to the atmospheric pressure, range from < 20 micropascal to > 200 pascal, a range of 1–10 million. Therefore, in acoustics, the logarithm of sound pressure relative to a reference sound pressure is used as a basis for a sound (and noise) exposure measure: the physical quantity sound pressure level expressed in decibel (dB).

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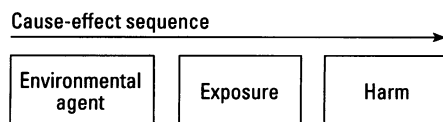


Figure 1. Simple cause-effect chain for the assessment of the health effects of an environmental agent, such as a xenobiotic substance.

The human hearing organ is not equally sensitive to sounds of different frequencies. Therefore, a spectral sensitivity factor is used that rates sound pressure levels at different frequencies in a way comparable to that of the human hearing organ; this is called A-weighting. The biophysical quantity A-weighted sound pressure level (L) is expressed as dB(A) and is referred to as sound level. Examples of sound levels in some common situations are falling leaves (very quiet), 10–20 dB(A); vacuum cleaner, 55–65 dB(A); location close to a main road or highway, 70–80 dB(A); pop music concerts, 100–110 dB(A).

Equivalent Sound Level and Day-Night Level

Sound level is the basic metric from which other biophysical metrics to specify long-term exposure to noise are derived. Usually a noise metric is assessed on an annual basis. In environmental and occupational situations, sound levels fluctuate with time. From these fluctuating sound levels, the equivalent sound level ($L_{Aeq,T}$) over a period of time, T , is determined from [see (7)]:

$$L_{Aeq,T} = 10 \log \frac{1}{T} \int_0^T 10^{\frac{L(t)}{10}} dt$$

Common exposure periods T are 24 hr (full day) and 8 hr (work day).

For some environmental health assessment purposes, the day-night level (L_{dn}) is used. This metric is the equivalent sound level over 24 hr with the sound levels during the night (11 PM–7 AM) increased by 10 dB(A). Also a day-evening-night level (L_{den}) is used, which is constructed similarly, such that the sound levels during the evening (7 PM–11 PM) are increased by 5 dB(A) and those during the night (11 PM–7 AM) by 10 dB(A). These adjustment factors of 10 or 5 dB(A) take into account that night-time and evening-time noise are more annoying than day-time noise with the same equivalent sound level.

Because of road, railway, and aircraft traffic noise, most of the urban population in industrialized countries are exposed to outdoor L_{dn} levels of > 50 dB(A). Rural populations usually are exposed to outdoor traffic L_{dn} values of < 50 dB(A). Rough estimates of the percentage of people in Europe living in locations with L_{dn} values > 60 dB(A) vary from 2 to 8%, depending on the country in which

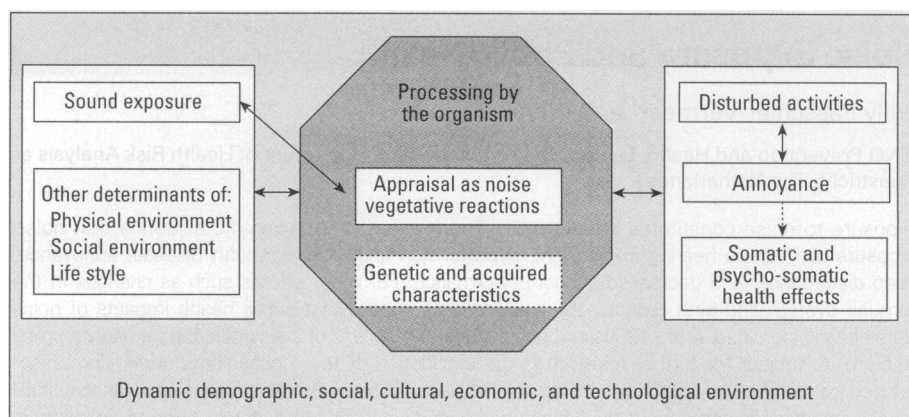


Figure 2. Conceptual model of the interaction of sound with the organism and the occurrence of effects on health and quality of life.

they live. For the Netherlands population this percentage is 4%. It is further estimated that 0.6% of the Netherlands population is exposed to traffic noise with L_{dn} values of > 70 dB(A) (8,9).

Both in research and in policy, L_{dn} or L_{den} is applied in a specific way: the metrics are used as location-specific quantities to be measured in front of the facade of residential buildings.

Sound Exposure Level

A single noise event is characterized by its sound exposure level. The sound exposure level (SEL) of a noise event, such as the overflight of an airplane or the passage of a truck, is the equivalent sound level during the event normalized to a period of 1 sec (10).

Exposure Settings

In this review we discuss the health effects of occupational and environmental noise exposure. Even though noise sources at work are quite divergent, the exposure setting is well defined; i.e., exposure during the execution of occupational tasks. In the living environment not only the sources but also the exposure settings are quite diverse. As mentioned above, a common environmental noise source is traffic. In addition, in industrialized regions industrial noise may affect environmental quality. Another type of noise is neighbor noise, a factor frequently mentioned in surveys on residential satisfaction. Increasingly, people are exposed to noise during recreational activities such as pop music concerts, motor races, and arcade activities; often these types of exposures are undergone consciously or at least taken for granted. In this review emphasis is on chronic environmental noise exposures, particularly those due to traffic and industrial noises. If other sources of noise or exposure settings are meant, this will be mentioned explicitly.

Assessment of Health Effects

The Committee on Noise and Health, an international committee of the Health

Council of the Netherlands, in 1994 assessed the health effects of environmental and occupational noise exposure (6). It rated the evidence in terms of categories used by the International Agency on the Research of Cancer (11) as “sufficient,” “limited,” “inadequate,” or “lacking.” The report also presents observation thresholds for those adverse health effects for which sufficient evidence was considered available. The observation threshold for an effect was defined in the report as the lowest noise exposure value at which on average the effect was observed in well-designed epidemiologic studies (12). This definition implies that in the course of time the observation threshold of an effect may have to be lowered if supported by new information from epidemiologic studies.

In this review, the 1994 Health Council report (6) is considered a starting point. More recent reviews (13–19) and papers presented at the November 1998 meeting in Sydney, Australia, of the International Commission on the Biological Effects of Noise (20) were used to extend the 1994 evaluation. In general, the more recent reviews and papers (13–20) concur well with the conclusions of the Health Council if we take a rating of “inconclusive” (15,16) to be equivalent to the Health Council’s “limited.” With respect to some effects such as ischemic heart disease, hypertension, and congenital defects, there appear to be differences of opinion. This will be further discussed below (“Noise-Induced Stress-Related Health Effects”).

In Table 1 information is presented about the adverse effects related to environmental and occupational noise exposure that have been examined in epidemiologic studies. The table is adapted from Table 1 of the 1994 Health Council report (6). Changes concern the noise metric in which the observation thresholds for hypertension and ischemic heart disease were originally given (21). Also the observation threshold for being awakened

by a single noise event was lowered by 5 dB(A). Finally, we have added that the observation threshold for sleep pattern changes is < 60 dB(A) (expressed in outdoors $L_{Aeq,night}$).

Several health end points are not specific to noise exposure. In fact, in accordance with the conceptual model of Figure 2, factors that apparently modify the effects of noise exposure may also affect health in ways similar to those for noise exposure. Situations exist in which it is difficult to identify primary and modifying factors.

The following sections highlight the main aspects of the data presented in Table 1.

Noise-Induced Hearing Impairment

Hearing impairment is an increase in the hearing threshold level. In the International Standard ISO 1999 (22), a hearing handicap is defined as the disadvantage imposed by hearing impairment sufficiently severe to affect one's personal efficiency in the activities of daily living, usually expressed in terms of understanding conventional speech in low levels of background noise. Hearing impairment is also associated with aging as well as certain diseases, exposure to some industrial chemicals, ototoxic drugs, head injuries, accidents, or factors that are of hereditary origin.

ISO 1999 (6) gives a method to estimate noise-induced hearing impairment in populations exposed to continuous, intermittent, or impulse noises during working hours. Noise exposure is characterized by the equivalent sound level over an 8-hr work day ($L_{Aeq,8h}$). Relations are given (for exposure times up to 40 years) between $L_{Aeq,8h}$ and noise-induced hearing impairment at frequencies between 500 and 6,000 Hz. These relations show that noise-induced hearing impairment occurs predominantly in the higher frequency range of 3,000–6,000 Hz, with largest effects observed at 4,000 Hz. With increasing $L_{Aeq,8h}$ and increasing exposure time, noise-induced hearing impairment can also occur at lower frequencies, more specifically at 2,000 Hz. Even with prolonged occupational noise exposure, however, according to ISO 1999 noise-induced hearing impairment does not occur at $L_{Aeq,8h}$ levels of 75 dB(A) and lower. This value is equal to the value specified in 1980 by the World Health Organization (23).

Since the method specified in ISO 1999 is the only universally adopted method to estimate occupational noise-induced hearing impairment, attempts have been made to assess whether this method also applies to hearing impairment due to environmental noise, including leisure-time noise. The results of various studies strongly suggest that the ISO 1999 procedure can also be accepted for environmental and leisure-time noise exposures of adults and older children

Table 1. Long-term effects related to exposure to noise and classification of the evidence for a causal relationship between noise and effect. The last three columns contain information on the observation threshold of an effect for which the causal relationship with noise exposure (second column) is judged to be sufficient.^a

Effect	Classification of evidence ^b	Exposure situation	Observation threshold		
			Metric	Value (dB(A))	Indoors/ outdoors ^c
Hearing impairment	Sufficient	Occ	$L_{Aeq,8h}$	75	Indoors
		Env	$L_{Aeq,24h}$	70	Indoors
		Occ unb	$L_{Aeq,8h}$	< 85	Indoors
Hypertension	Sufficient	Occ ind	$L_{Aeq,8h}$	< 85	Indoors
		Env	L_{dn}	70	Outdoors
Ischemic heart disease	Sufficient	Env	L_{dn}	70	Outdoors
Biochemical effects	Limited	Occ			
		Env			
Immune effects	Limited	Occ			
		Env			
Birth weight	Limited	Occ			
		Env air			
Congenital effects	Lacking	Occ			
		Env			
Psychiatric disorders	Limited	Env air			
Annoyance	Sufficient	Occ office	$L_{Aeq,8h}$	< 55	Indoors
		Occ ind	$L_{Aeq,8h}$	< 85	Indoors
		Env	L_{dn}	42 ^d	Outdoors
Absentee rate	Limited	Occ ind			
		Occ office			
Psychosocial well-being	Limited	Env			
Performance	Limited	Occ env			
	Sufficient	School	$L_{Aeq,school}$	70	Outdoors
Sleep disturbance, changes in					
Sleep pattern	Sufficient	Sleep	$L_{Aeq,night}$	< 60	Outdoors
Awakening	Sufficient	Sleep	SEL	55	Indoors
Sleep stages	Sufficient	Sleep	SEL	35	Indoors
Subjective sleep quality	Sufficient	Sleep	$L_{Aeq,night}$	40	Outdoors
Heart rate	Sufficient	Sleep	SEL	40	Indoors
Hormone levels	Limited	Sleep			
Immune system	Inadequate	Sleep			
Mood next day	Sufficient	Sleep	$L_{Aeq,night}$	< 60	Outdoors
Performance next day	Limited	Sleep			

Abbreviations: env, living environment; ind, industrial; occ, occupational situation; school, exposure of children at school; unb, unborn; exposure of pregnant mother. ^aThe table is adapted from Table 1 of the 1994 Health Council report (6). ^bClassification of evidence of causal relationship between noise and health. ^cValue relates to indoor or outdoor noise assessment. ^dThe observation threshold for percentage of highly annoyed persons is about 12 dB(A) lower for environmental impulse noise.

provided the exposures are not too extreme and the exposures are expressed in $L_{Aeq,24h}$ (because exposure during the full 24-hr day is important in this case) instead of $L_{Aeq,8h}$ (6,23–31). This implies that exposure to environmental and leisure-time noise with $L_{Aeq,24h}$ values < 70 dB(A) does not cause hearing impairment in the large majority of people (> 95%), even in the case of life-time exposure (32). It should be considered, however, that there are no large-scale epidemiologic studies that investigated noise-induced hearing impairment in the general population that support this proposition. Also, data from animal experiments indicate that young children may be more vulnerable to noise-induced hearing impairments than adults (31). For impulsive (shooting) noises with $L_{Aeq,24h}$ > 80 dB(A) studies on temporary threshold shifts (26) suggest the possibility of an increased risk for impulse noise-induced hearing impairment in adults.

At high instantaneous sound levels, mechanical damage to the outer and the inner ear may occur. Occupational limits for such types of exposures have been set equal to the observation threshold for this effect at a peak sound pressure level of 140 dB (33). For adults, it is reasonable to assume that a similar threshold applies with respect to exposure to environmental and leisure-time noise. In the case of children, however, taking into account their habits of playing with noisy toys, peak sound pressure levels > 120 dB may cause mechanical damage to the hearing organ (31).

Noise exposure may also result in tinnitus (ringing in the ears). This effect has been observed among teenagers attending pop music concerts and discotheques (34). Noise-induced tinnitus may be temporary, lasting up to 24 hr after exposure, or it may have a more permanent character, such as after prolonged occupational noise exposure. Approximately 25% of workers with both noise-induced

hearing impairment and tinnitus consider tinnitus the more disturbing effect (35).

The main social consequence of hearing impairment is the inability to understand speech in daily living conditions—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.

Psychosocial Effects

Psychosocial effects due to exposure to environmental noise that have been studied in epidemiologic investigations include annoyance, psychosocial well-being, and psychiatric hospitalization. The main psychosocial effect from exposure to occupational noise observed in epidemiologic investigations is annoyance.

Noise annoyance is a feeling of resentment, displeasure, discomfort, dissatisfaction, or offense when noise interferes with someone's thoughts, feelings, or actual activities. It is not yet possible to predict noise annoyance on an individual basis because of the large variety of (partly unknown) endogenous and exogenous characteristics that affect annoyance (Figure 2). However, relationships between noise annoyance and noise exposure have been elucidated on a population level together with several effect-modifying factors. Annoyance in populations is evaluated using questionnaires. Exposure–effect relationships have been derived for exposure to the three main types of traffic noise: road, railway, and aircraft. The most recent and comprehensive relationships are shown in Figure 3 (36). These relationships pertain to populations chronically exposed to noise at specified levels for periods of more than a year. The effect is given as the percentage of the population highly annoyed by a specific environmental noise. “Highly annoyed” persons are those

who respond to a question about the degree of annoyance in the worst 25% range of answer categories (37). The noise exposure is expressed in L_{dn} , assessed in front of dwellings. The relationships depicted in Figure 3 demonstrate that annoyance induced by the different modes of transport—air, road, and rail—differs at higher exposure levels. Taking into account the statistical variations within and between the various studies, Miedema and Vos showed that aircraft noise is statistically significantly more annoying and railway noise is less annoying than road traffic noise (36).

Environmental noise exposure is only one of the factors that contributes to noise annoyance, albeit a significant one. The degree of annoyance experienced by an individual as well as that on a population level in practice can differ considerably from the exposure–response relationships presented in Figure 3 because of the influence of so-called nonacoustical factors. Important nonacoustical effect-modifying factors are anxiety, fear of the noise source, and a feeling that the noise could be avoided. These effect-modifying factors have been identified in multivariate analyses of population data (19,38–41). However, general quantitative multifactorial exposure–response relationships have not yet been published.

Much attention has been paid in laboratory experiments to the effects of uncontrollable noise exposure on such things as task performance and annoyance. No relationships have been assessed between general noise annoyance experienced during working hours and noise exposure. Epidemiologic studies show that annoyance in offices is considerable at equivalent sound levels > 55 dB(A). A few studies show that 35–40% of office workers are highly annoyed at noise levels from 55 to 60 dB(A). If the noise source is more or less constant, such as the noise produced by ventilation systems (e.g., fans in computers), the observation threshold for annoyance in offices is lower than a $L_{Aeq,8h}$ value of 55 dB(A). In industrial situations, similar percentages of highly annoyed workers occur at equivalent sound levels > 85 dB(A).

Also at the workplace, nonacoustical factors have a large effect on the actual noise annoyance on an individual and on a population level. These factors include the meaning and information contents of the noise (telephone conversations and discussions between colleagues score high), predictability, avoidability, controllability, task demands, and attitudes toward the noise source.

Noise-related annoyance is widespread in present-day society. Even though annoyance as such is not directly invalidating, there are indications that for sensitive individuals or in cases of concurrent exposure to other

environmental agents or socially distressing situations, more serious health effects can occur [see Figure 2 and a recent report of another International Health Council committee (42)].

Noise-Induced Stress-Related Health Effects

Reactions to a stressor can be psychological (feelings of fear, depression, sorrow), behavioral (social isolation, aggression, excessive use of alcohol, tobacco, food, drugs), and somatic (cardiovascular, gastrointestinal, respiratory illnesses) in nature. A large number of laboratory experiments [reviewed by Passchier-Vermeer (24)] have shown noise-induced temporal changes in the cardiovascular system. These findings led to several investigations into possible long-term effects associated with noise exposure, e.g., stress-related cardiovascular disorders. In addition, some research has been conducted on the effects of noise exposure on the hormone and immune systems. Effects from occupational or environmental noise on reproduction and development were also studied. High-frequency hearing impairment in babies of mothers exposed to high levels of occupational noise during pregnancy is also considered to be a consequence of a mother's stress induced by exposure to noise during pregnancy (43).

Research into the chronic effects of long-term exposure to noise is complicated because cardiovascular and biochemical changes are nonspecific and a number of other factors may also cause these changes; these factors must be controlled for in research projects. In cross-sectional studies it is difficult to obtain appropriate information about past noise exposure, and longitudinal studies are time-consuming and financially draining. Furthermore there are large individual differences in susceptibility. Also, people intervene in their own situations, e.g., by changing jobs [thus contributing to the “healthy worker effect” (44)] or by moving from noisier surroundings to quieter places. This may result in “noise proof” populations exposed to the higher noise levels (45). Notwithstanding these complications, conclusions on the relationship between noise exposure and cardiovascular disease appear possible from meta-analyses of the available epidemiologic data (6).

Cardiovascular effects in adults. Epidemiologic environmental noise studies on changes in blood pressure and increased risk for ischemic heart disease in adults are limited mainly to the effects of road traffic noise, with the exception of a Dutch study on the effects of aircraft noise (46,47). In general these studies demonstrate no obvious effects from noise exposure on mean diastolic and mean systolic blood pressure, but some effects

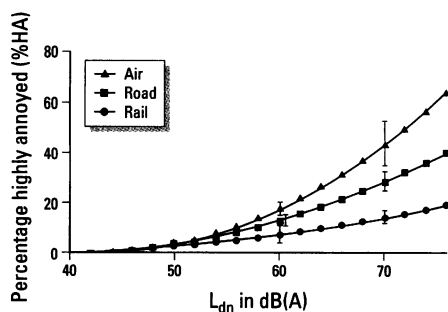


Figure 3. Relationships between the percentage of highly annoyed persons and L_{dn} for air, road, and railway traffic noise. Each curve has been derived by a multilevel analysis of all studies for which original data were available. The vertical bars at 60 and 70 dB(A) represent 95% confidence intervals [bar at 60 dB(A) for road has been displaced by 0.5 dB(A) for clarity].

were observed in terms of an increase in the percentage of people with hypertension (including those who use medication for hypertension). The observation threshold for hypertension is estimated to correspond to an L_{dn} value of 70 dB(A) for environmental noise exposure. The Health Council of the Netherlands in 1994 (6) suggested the same observation threshold for ischemic heart disease (Table 1). The relative risks (compared to populations with low environmental noise exposure) for both hypertension and ischemic heart disease for exposure levels above the observation thresholds are estimated to be about 1.5 (48).

In 1997 a Chinese study (49) was conducted among a large sample of more than 20,000 residents in rural communities. The results show that self-reported exposure to noise (unfortunately, exposure was not assessed objectively) is an important determinant of systolic and diastolic blood pressure. Of special interest is the outcome of the recent, unique longitudinal study (50) on the effect of road traffic noise exposure on the incidence of ischemic heart disease. In this Caerphilly and Speedwell study, two cohorts of about 2,500 middle-aged men in the United Kingdom were recruited for a study of the predictive power of already-known and new risk factors for ischemic heart disease. Noise measurements were performed in each of the streets where subjects lived. Even in the highest noise exposure class, L_{dn} did not exceed 70 dB(A). Statistical analysis on the relationships between incidence of ischemic heart disease (classified in a standardized way) and environmental noise exposure was controlled for potentially confounding factors. The average annual incidence rate of ischemic heart disease appeared to be 1.4% during the second phase of the study (6 year follow-up; mean age of the men, 57 years). If orientation of the living room and the bedroom, window opening habits, and years of residence over 15 years were taken into account, the relative risk for incidence of ischemic heart disease of the highest exposed group relative to the group exposed to levels between 50 and 55 dB(A) was 1.6, which statistically is not significantly different from 1 at the 5% level ($p < 0.10$). This study fits in with the earlier evaluation that above levels of 70 dB(A) there is sufficient evidence for a noise exposure-related effect, and provides no support for lowering the observation level of 70 dB(A) for ischemic heart disease.

Through analysis of twelve studies on the risk of hypertension among occupational noise-exposed workers, the observation threshold for industrial noise exposure was determined to be at most equal to an $L_{Aeq,8h}$ value of 85 dB(A) (24). No data are available for noise exposure in offices. If annoyance

and stress-related health effects are associated, and taking into account that the observation threshold for annoyance in offices is much lower than that for industrial situations, we hypothesize that the observation threshold for noise-induced risk for hypertension in office workers is about 30 dB(A) lower than that for hypertension in blue-collar workers. The data in more recent publications on cardiovascular effects from exposure to noise are not in disagreement with the previous findings (51–53). However, one must be careful interpreting the results of these studies, as either noise measurements or proper control groups are lacking or the size of populations studied is small.

Only few epidemiologic studies considered biochemical and immunologic effects (54,55). More recently, overnight resting levels of epinephrine and norepinephrine levels were assessed in a study of middle-aged women living in Berlin (56). Significantly elevated levels of norepinephrine were found in women whose bedrooms faced busy streets (> 20,000 vehicles a day) and epinephrine levels were also higher in women reporting high disturbances of communication and sleep under closed window conditions. Some smaller studies in industrial settings showed the effects of wearing personal hearing protection on urinary excretion of catecholamines (epinephrine and norepinephrine) and cortisol (57,58). On the days hearing protectors were worn, urinary catecholamine levels were statistically significantly lower than on days protectors were not worn.

Cardiovascular effects in children. Two early studies (59,60) showed an increase in systolic and diastolic blood pressure in children exposed to very high road traffic noise levels or aircraft noise levels. The increases were assumed to be of a transient nature. Recently, Slovakian researchers studied 1,542 children 3–7 years of age in kindergartens (61). The authors observed significantly higher systolic and diastolic blood pressures among children in noisy environments [> 60 dB(A)] compared to those among children in quieter environments. Although the study is carefully designed, the possibility that social class has confounded the results cannot be excluded [see also Lercher et al. (62)]. In the Munich airport study (63,64), schoolchildren were examined during the years Munich airport moved from one location to another. One study location was close to the old airport and another was close to the new airport. The cross-sectional part of the study showed a marginally significant higher systolic blood pressure in children highly exposed at school. Children were matched on socioeconomic characteristics. In the study, neuroendocrine indices of chronic stress (urinary cortisol levels and levels of epinephrine and norepinephrine)

were also examined. Overnight resting levels of epinephrine and norepinephrine levels were significantly higher in children exposed to aircraft noise at the old Munich airport compared to control groups. There were no differences in cortisol levels. After the airport was moved, overnight resting levels of epinephrine and norepinephrine rose significantly among children living under the flight paths of the new airport. Again, no effects were observed on urinary cortisol levels. We propose that this subject be studied further.

Effects on the unborn child. Data from older studies suggest that it is possible that when pregnant women are exposed to high levels of aircraft noise [$L_{dn} > 62$ dB(A)], small reductions in birthweight occur. In a more recent study of 200 Taiwanese women, noise exposure was measured by personal noise dosimeters on three occasions during pregnancy (65). Noise exposure was not related to birthweight after adjustment for social class, smoking and alcohol use, maternal weight gain in pregnancy, gender of the child, and duration of pregnancy. Older and more recent investigations do not show statistically significant effects of occupational or environmental exposure of pregnant women to noise in the course of pregnancy and congenital defects of babies, with the exception of high-frequency hearing damage mentioned at the beginning of this section.

Absences due to accident and sickness. Epidemiologic studies suggest that the absentee rate of industrial workers increases when they are exposed to equivalent sound levels during working hours of over 75 dB(A) [CORDIS study, (66)] or over 90 dB(A) (67). The CORDIS study also showed that the number of accidents increases with rises in equivalent sound levels during working hours. Mortality from injury was studied in more than 20,000 steelworkers (68). On the basis of job and workplace information, industrial hygienists estimated noise exposure as high [$L_{Aeq,8h} > 95$ dB(A)], medium [$L_{Aeq,8h}$ 90–95 dB(A)], low [$L_{Aeq,8h}$ 85–90 dB(A)], and minor. Hearing damage and noise exposure in the high and medium noise classes appeared to be factors that contributed statistically significantly to mortality. The study did not identify the mechanisms behind these findings. Moreover the impact of using personal hearing protectors is unclear. Wearing of hearing protection by workers with substantial noise-induced hearing impairment reduces the possibility of hearing moving sound sources, warning signals, or colleagues shouting and hampers localization of moving sound sources because of reduced capacities to determine the direction of a sound source. Therefore, we hesitate to consider stress as the underlying mechanism for the increase in mortality.

Sleep Disturbance

Sleep is a recovery process essential for humans to function properly. In addition, people like to sleep and usually consider a good night's sleep to be an important aspect of an individual's quality of life. Deleterious health effects are expected from chronic noise-induced interference with sleep, as it impairs the functions of sleep such as brain restoration and provision of a period of respite for the cardiovascular system (69,70). In addition to the physiologic aspects of a noise-induced reduction of sleep quality, night-time noise exposure of sufficient intensity (Table 1) is also related to subjectively experienced sleep quality (71). Reduced sleep quality also interferes with daytime functioning and can have adverse effects on mood next day and possibly on vigilance and cognitive performance.

Sleep quality can be quantified by subjective and objective methods. The most commonly applied subjective methods are self-reporting using sleep logs or diaries and, to a lesser extent, behavioral observations. The most commonly used objective methods are electroencephalograph (EEG) recordings and actimetry. In field studies on noise-induced sleep disturbance subjects usually wear watchlike actimeters for movement detection at their wrist. Sleep quality may be adversely affected by

- changes in the cardiovascular system;
- changes in sleep pattern such as increased sleep latency time and reduced sleep time because of premature awakening;
- changes in sleep stages from deeper to less-deep sleep;
- increases in motility during the sleep period;
- increases in number of awakenings during the sleep period;
- changes in subjectively experienced sleep quality; and
- changes in the hormonal and immune systems.

Present knowledge about the relationships between awakening and exposure to single noise events indicates that habituation or adaptation occurs. This insight is not new. Cohen stated in 1968 [in Ward and Fricke (2)] that

Aspects of adaptation to noise with regard to sleep disturbance also need to be evaluated. Common experience has found that the city dweller, frequently encountering significant levels of outdoor and indoor noise, becomes accustomed to such exposures and can sleep in their presence. The same individual vacationing in the quiet atmosphere of the country finds it difficult to sleep because of the background of cricket noise. The degree of familiarity or meaningfulness of the noise has a considerable effect on its disturbing quality.

From the epidemiologic studies there appears to be sufficient evidence for a causal relationship between exposure to night-time noise and changes in sleep pattern, sleep stages, awakenings, subjective sleep quality, heart rate, and mood the next day (6). Observation thresholds for these effects are given in Table 1. Evidence for other effects is limited (hormone levels and performance the next day) or inadequate (immune system). Exposure-response functions have been derived from field studies for only some of these effects, among others or reduction of subjective sleep quality and increase in number of awakenings during sleep period time. The relationship between the risk of awakening and exposure to night-time environmental noise is established only for single noise events, with exposure specified by the indoor SEL values of the events.

An international group of experts who were convened in 1997 by the Health Council of the Netherlands assessed the observation threshold for awakening due to single noise events at the lower indoor SEL value of 55 dB(A) (Table 1) instead of 60 dB(A) (8). This change reflected improved knowledge of the transfer functions of SEL values measured outdoors compared to those measured indoors in some of the underlying studies. Using the relationship between indoor SEL value and the risk of awakening due to single noise events, the expected maximal number of awakenings per year in an adult habituated to night-time noise exposure was estimated as a function of the equivalent sound level during the night. The latter quantity was calculated from the number of single noise events during the night and their indoor SEL values (8). The result, which represents a worst-case situation, is depicted in Figure 4.

Apart from the direct effects of night-time noise on sleep, various authors point to the importance of the impact of sleep disturbance on quality of life, including such factors as somatic health and annoyance. Such observations had already been made in the 1950s. Cohen, referring to a paper of Borsky from 1958, states

Field studies have shown that much greater annoyance results when sleep and rest are disturbed than when only talking or listening activities are interrupted. This finding plus the health significance attributed to rest and sleep suggest that criteria for annoyance be based on noise-induced disturbances to sleep. [in Ward and Fricke (2)]

Babisch et al. reported larger overnight changes in epinephrine levels in subjects reporting high disturbance of sleep than in those without severe complaints (56). Another study showed that psychosocial

well-being of subjects exposed to high levels of road traffic noise was not related to daytime noise exposure but to night-time equivalent sound level in the bedroom and to subjectively experienced sleep quality (71).

Although in the 1990s several field studies were started (72) or completed (73–76), there still is an urgent need for a tested model on sleep disturbance, environmental noise exposure, and secondary effects, in which causal and modifying factors and their mutual relations are assessed.

Effects on Performance

There is overwhelming evidence from laboratory experiments that the presence of uncontrollable noise can significantly impair cognitive performance. Noise can induce learned helplessness, increase arousal, alter the choice of task strategy, and decrease attention to the task. Noise may also affect social performance, mask speech and other sound signals, impair communication, and distract attention from relevant social clues. Adverse acute effects already have been assessed at low levels. Performance of a task involving motor and monotonous activities is sometimes not only is not decreased, but on the contrary, is enhanced.

Two older epidemiologic studies have shown that schoolchildren when exposed to high levels of traffic noise show impairments in performing cognitive tasks (59,60). The observation threshold derived from these data is 70 dB(A) (expressed in $L_{Aeq, schoolhours}$). More recently this subject has received renewed attention (64,77–78). In the Munich airport study mentioned previously, reading comprehension and long-term memory were impaired in children attending schools located around the old Munich airport and reading comprehension improved after the closing of the airport. However, reading comprehension deteriorated in children subjected to aircraft noise exposure near the new Munich airport. Recently, in the United Kingdom a field study with tests repeated annually was conducted to assess whether the association between aircraft noise exposure and reading comprehension was mediated

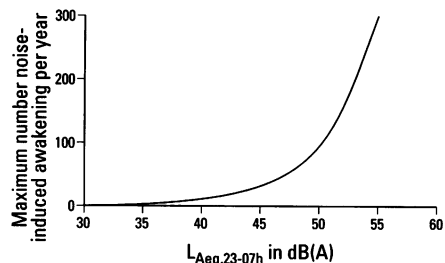


Figure 4. The maximum number of awakenings per year as a function of the outdoors night-time equivalent sound level (8).

through sustained attention and whether it was confounded by social deprivation and language spoken at home. The 340 children who participated were about 9 to 10 years of age. They attended a school classified either as a high-noise school [$L_{Aeq,16h} > 66$ dB(A)] or as a low-noise school [$L_{Aeq,16h} < 57$ dB(A)]. There appeared to be a high correlation between the noise at school and the aircraft noise exposure at home. Results show that the average reading comprehension of children attending the high-noise schools was poorer at both measuring times compared with that of children from the low-noise schools. Sustained attention, measured only at follow-up, was poorer in children at the high-noise schools than in children at the low-noise schools. Sustained attention did not play a significant role in explaining the relation between reading comprehension and aircraft noise exposure. However, if adjustments are made for age, main language spoken at home, and social deprivation, the differences in reading comprehension failed to be significant. These results are not in disagreement with the 1994 evaluation (6) leading to an observation threshold of 70 dB(A) (expressed in $L_{Aeq,schoolhours}$), but setting the threshold at a lower level does not appear to be warranted. Given the possible long-term consequences of cognitive effects in children, we feel that further research into mechanisms and contributing factors is urgently needed.

Noise Metrics and Noise Limits for Health Protection

Several biophysical quantities to represent noise exposure were introduced in the section "Characterization of Noise Exposure." Exposure quantities are not only of scientific interest, i.e., for recording data and communicating research results; policymakers and risk managers need exposure quantities to judge the necessity of taking protection or mitigation measures and to evaluate the effectiveness of such measures. Criteria for noise exposure metrics to be used in health and environmental policy (8) are that they should be *a*) relatively simple to determine or measure; *b*) transparent with respect to exposure-response relationships; *c*) correlated with health effects on a population level; *d*) applicable to all outdoors noise sources; *e*) universal; and *f*) communicative. In practice it appears almost impossible to derive a single metric and at the same time fulfill all these criteria because noise sources, noise characteristics, and exposure situations differ extensively.

One application of noise metrics is to set exposure limits. Because such limits are intended for health protection it is essential that the science policy decisions made to derive the metrics be known to the policymaker.

Noise-Induced Hearing Impairment

Sound exposure measures were already being proposed in the 1960s and 1970s that would apply to a variety of settings and, if an exposure-response relationship were known, would be a good predictor of effects to be expected for any case at the population level. A good example is the equivalent sound level over an 8-hr work period ($L_{Aeq,8h}$), which correlates well on a population level with noise-induced hearing impairment. Confidence in the exposure-response relationships is such that they have been standardized by ISO (22). This is reflected in policy debates; such debates focus on the measures to be taken if certain exposure levels are exceeded, and not on the validity of the exposure-response relationships.

A value of $L_{Aeq,8h}$ of 85 dB(A) (sometimes lower) has been almost universally adopted as a limit for unprotected occupational noise exposure, with additional requirements for personal hearing protection above this value (79). However, among workers exposed to an $L_{Aeq,8h}$ value of 85 dB(A), some noise-induced hearing impairment will occur. Given such exposure over a lifetime in a job, a hearing impairment at 4,000 Hz of about 5–10 dB is estimated for most workers, although for those persons highly sensitive to noise, noise-induced impairment is considerably greater. This implies that a lifetime of exposure to 85 dB(A) of occupational noise will slightly increase the risk for a hearing handicap in a small proportion of exposed persons.

We concur with the suggestion to use the equivalent noise level over a period of 24 hr, to set targets for the exposure during the full 24-hr day for protecting the hearing of the general population (80). Setting such targets appears to be warranted, given the increasing number of noisy activities and exposure duration, such as loud music in cars, the use of portable music cassette and CD players, and the playing of loud computer games at home and in arcades.

Annoyance and Stress-Related Disorders

Metrics such as $L_{Aeq,24h}$, L_{dn} , and L_{den} came into use several decades ago (3,4) to regulate general annoyance. Recently, an international group of experts convened by the Health Council of the Netherlands again studied specification of a biophysical metric to express noise-induced general annoyance for public health purposes (8). The Health Council committee agreed on adjustment factors to be applied to the metrics to account for differences in annoyance related to the tonal and impulse characteristics of noise. The committee extensively debated the choice between L_{den} and L_{dn} . Analysis of available data indicated that for road traffic noise, general annoyance is estimated from L_{den} with smaller

confidence intervals than those from the other metrics. However, a decision based on statistical grounds could not be made (81). Because from a policy viewpoint, using L_{den} would result in somewhat more plausible protection and mitigation measures, the committee finally expressed its preference for this metric. In Europe L_{den} may become the future noise metric to represent general noise-related annoyance (82).

When using the adjusted L_{den} levels, the exposure-response relationships are statistically significantly different for different modes of transport. One might envisage performing a further adjustment that would result in a single relationship for all types of transportation noise and possibly industrial noise; in fact recommendations for such a further integration were made in the Health Council report (8). We strongly support such a development that would require standardization of the noise exposure-general annoyance relationship; basic data for reaching that goal are presently available (36). The final step to representing general annoyance with a universal noise metric would be a procedure to combine concurrent noise exposures from different sources. This step requires further research, as limited data have been published on exposure to two or more sources at the same time. However, in most practical situations exposure from one source will dominate.

A question arises about whether noise abatement policies based on the adjusted L_{den} are also effective in reducing the prevalence of other noise-induced health effects such as hypertension, ischemic heart disease, and cognitive performance in schoolchildren. Although this appears to be plausible, further study is needed.

A cautionary remark is in order here. As previously indicated, many other factors in addition to noise exposure influence noise-related health effects on a population level. The exposure-response relationships between, for example, the percentage of highly annoyed persons and L_{den} should be used for policy guidance rather than to obtain accurate predictions of effects expected in specific situations. However, the quantitative relationships presented here are the best science has to offer today and appear to be rather robust.

Sleep Disturbance

There appears to be consensus that for protection against sleep disturbance a separate night-time noise exposure metric is required, even though limiting exposure using L_{den} or a similar 24-hr metric would also provide some limitation of night-time noise exposure. The Health Council committee report (8) mentioned previously proposed the night-time equivalent sound level (see Figure 4). A

science policy question arises about whether the worst-case approach in converting SEL to $L_{Aeq,23-07h}$ is acceptable in policy situations. In specific situations, for example, populations in the vicinity of large airports, economic consequences of the noise abatement measures may be severe, although from a health protection viewpoint using the equivalent sound level could be defended.

The Way Ahead

In addition to natural sounds, speech, and music, noise is widespread and becoming more so in our present day society. Major factors are the increase in motorized traffic, apparent preferences for noisy leisure and recreational activities among large groups in industrialized societies, and increasing urbanization, particularly in the Third World, resulting in megacities where high noise levels 24 hr a day have become commonplace. Although at the beginning of the 1960s the major effects of noise exposure were already known, at the beginning of the 21st century noise exposure still is a major public health problem. Given our view that knowledge about effective noise abatement measures for public health protection has been available for decades, solving the problem appears now to be primarily in the hands of policymakers. In addition to regulatory measures, activities in the realm of health education with respect to the impact of noise exposure in everyday life appear to be warranted. Concerns are increasing that noise-induced hearing loss is not only an occupational risk but may become an environmental risk as well.

Science still has important contributions to make. We mentioned in the previous section the study of policy-relevant metrics, for which more insight in the exposure-response relationships for different types of noise and exposure settings is required. The modifying influence of nonacoustical factors is of utmost importance, as these influences may to a large degree determine the effectiveness and efficiency of noise abatement measures directed at reducing effects other than noise-induced hearing impairment. This is obvious in the case of environmental exposures, but it may also be relevant for the workplace, for example, the possible differences between observation thresholds for cardiovascular disorders in white and blue collar workers mentioned previously.

We believe that two subjects should be priorities in research: the study of cardiovascular effects and the underlying mechanisms and the study of the effects of noise on children. Attention to effects on children is particularly urgent, as such effects in terms of years of life with a reduced quality of life or a handicap are greater for children than for adults.

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- In the equivalent sound level over a period T , the highest sound levels occurring during this period are counted more heavily than those in the 'normal' average sound level over period T . This is demonstrated by the following example. Suppose that during half of period T the sound level is 80 dB(A) and during the other half it is 40 dB(A). Then the equivalent sound level over period T is 77 dB(A), whereas the arithmetic average sound level is 60 dB(A).
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- If during a period T (in s) n noise events occur with the same SEL value, the following formula applies: $L_{Aeq,T} = SEL + 10 \lg n - 10 \lg T$.
- Preamble to the IARC Monographs. <http://193.51.monoeval/preamble.html>, 12-6-1997. Consulted: 3-4-1998.
- An observation threshold concerns an average population of adults or adult workers or an average population otherwise specified, such as babies of women exposed to noise during pregnancy.
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- The conversions are between the day-time equivalent sound level and L_{dn} . For most situations with higher environmental noise exposures, daytime and nighttime exposures are highly correlated. For road traffic noise, the average difference between daytime and nighttime equivalent sound levels is approximately 8 dB(A). This implies that the difference $L_{dn} - L_{Aeq,24h}$ is about 2 dB(A), and the difference between the day-time equivalent sound level and L_{dn} about -1 dB(A). In Table 1, also taking into account the accuracy with which the observation thresholds for hypertension and ischemic heart disease could be established, for those noise-induced effects L_{dn} is taken to be equal to the daytime equivalent sound level.
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81. On average the differences between L_{den} and L_{dn} are only marginal. By analyzing over 60,000 situations with road, railway, or aircraft noise, the average difference between L_{den} and L_{dn} appeared to be less than 0.5 dB(A). In specific situations, however, the difference may be substantially larger.
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