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Community noise burden of disease: an impossible choice of endpoints?ⁱ

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Abstract

In this chapter we discuss a number of approaches to assess the impact of exposure to community noise on public health. Briefly noise characteristics and how they are accounted for in noise metrics are discussed. Successively we assess the current evidence on a range of health endpoints published in the open literature. We apply a conceptual model to make a synthesis of current evidence, linking together the information on individual responses proposing a mechanistic explanation of health effects shown to be associated with noise exposure. Finally, we present a number of different options for crude estimation of burden of disease attributable to noise exposure. This yields an interesting dilemma: the association between noise and 'annoyance' has a high validity, but the public health significance (content validity) is controversial. The other way round, the clinical significance of cardiovascular disease is obvious, but a causal relation with noise exposure is not yet fully supported by available epidemiological data or scientific consensus.

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6.1 An inescapable nuisance

6.1.1 Community noise

Noise is a ubiquitous, persistent and largely inescapable environmental pollutant in most of the (post-)industrial and motorised world (see table 6-1). Transport, comprising road and rail traffic, aviation and shipping, is the most important source of community noise. In specific local situations, stationary sources, such as industry, building sites, sport and leisure facilities may also be a significant source of community noise. In most surveys noise produced in and around the house is reported as a significant cause of annoyance as well, especially in urban environments. This paper will address primarily the first category, as it is typically part of the policy domain of environmental protection.

Table 6-1. The changing 'soundscape'. Based on a comprehensive analysis of historical documents, literature, paintings and poems Schafer estimated the relative contribution of natural, human and machine sound to total noise exposure in different era of human civilisation¹.

Epoch	natural sound (%)	human sound (%)	machine sound (%)
early civilisation	69	26	5
medieval times	34	52	14
industrial age	9	25	66
present	6	26	68

In the Western world engine sound emission levels per vehicle have decreased substantially due to rapid improvements of technology. However, as a result of the application of broader tires the net sound production of private cars has not decreased. New generations of aircraft have become substantially more silent². Furthermore modern urban development often includes spatial separation of transport and residential functions. However, average exposure to noise is still increasing in most countries as a result of tremendous growth in volumes, especially in the developing world. According to the OECD growth will increase even more rapid in the next 20 years. In its Environmental Outlook projections predict an increase in motor vehicle kilometres of 40% in the OECD regions and of 86% in the developing rest of the world. The largest increase is expected in aviation, where the number of passenger kilometres will certainly triple, with the highest growth rates in Asia³. Furthermore, the '24 hours economy' will not only increase the level and area of noise exposure but will also affect the brims of the night⁴.

In European OECD countries around 30% of the population is estimated to be exposed to levels of road traffic noise above 55 dB(A), and around 13% above 65 dB(A) (L_{dn} -values)⁵. Other estimates yield even higher fractions of the population exposed to levels exceeding health-based guidelines⁶. Some 10% of the EU-population is estimated to be seriously disturbed by aircraft noise. Estimates from other regions in the World are scant, but occasional reports indicate the problem of residential noise pollution is not confined to the developed world⁷. Anecdotal surveys report high levels cities in developing countries with scant regulations and little formal urban planning, such as Karachi city, Bombay and Calcutta, where noise levels along busy roads ranged between 80-85 dB(A) (Leq, 8h), respectively 80-92 dB(A) (Leq, 24h), with peaks up to 140 dB(A) during rush hours^{8,9}.

6.1.2 Somewhere between well-being and health

Long-term exposure to noise has been associated with a wide range of effects on human health and well-being. Even though they are not independent, following the argumentation of chapter 3 one might make a

distinction between *social psychological* responses, such as annoyance and disturbance of daily activities on the one hand, and *clinical* effects on the other, such as hearing loss, hypertension and aggravation of cardiovascular symptoms¹⁰.

Noise is a major problem in modern societies. Not many people will argue with that. It is well established that as levels of noise exposure increase a higher percentage of any representative population will report to be severely annoyed. Noise intrudes into our personal privacy, disturbs our daily activities, causing annoyance, stress and affecting the quality of our life. No doubt this presents an important social problem. Whether this is a health problem as well is less obvious. Nonetheless, it is a fact that several studies have demonstrated an association between serious annoyance, and highly correlated social responses, such as perceived stress, anxiety, and risk perception on the one hand and reported (psychological) symptoms, cognitive complaints, (self-)medication and use of health services on the other. The causality of this association however is far from obvious¹¹.

6.1.3 Reader

In this chapter we present and discuss some proposals for simple, spreadsheet assessment of the health impact of exposure to noise at the population level. As promoted by the World Health Organisation comparative assessment of health impacts may facilitate policy makers to properly involve public health interest in infrastructure and urban planning^{12,13}.

We will begin with a short explanation of noise characteristics and how they are accounted for in noise metrics. This is followed by an assessment of the current evidence on a range of health endpoints published in the open literature. We will discuss a conceptual model to make a synthesis of current evidence, linking together the information on individual responses proposing a mechanistic explanation of health effects shown to be associated with noise exposure. Finally we present a number of simple methods for a crude estimation of burden of disease attributable to noise exposure, using the Dutch situation as an example.

To arrive at a quantitative assessment of disease burden associated with population exposure to community noise we will follow an exposure-based approach consisting of the following steps¹⁴:

review of causality, internal and external validity of health outcomes associated with noise exposures, followed by a *quantification of exposure response relations*

assessment of a *population exposure distribution*, using either national noise monitoring data and exposure models if available, or a robust model using data on degree of urbanisation, traffic or vehicle density, vehicle composition and emission data, aircraft noise distribution patterns etc.

estimation of *noise exposure attributable disease burden*, incorporating location specific base-line incidence or prevalence risks for health outcomes¹⁵.

6.2 Characterisation of noise exposure

6.2.1 Characteristics

Sound is a physical phenomenon of alternating compression and expansion of air that propagates from a source in all directions. These can be described as small pressures around the atmospheric pressure. The frequency of these alternations determines the pitch of a sound. A high pitch tone (e.g. 4,000 Hertz) has a squeaking sound; a low pitch tone (e.g. 200 Hertz) has a humming sound. Noise is adequately defined as "unwanted sound" (in physics there are formal definitions as well¹¹).

To assess the impacts on human health and well being various biophysical metrics of noise are relevant. In general these metrics are based on physical quantities to which "corrections" are applied that

take into account certain human noise sensitivity. These corrections depend on the frequency, noise characteristics (impulse, intermittent or continuous noise levels), and the source of noise. within the framework of this chapter the following metrics are important.

Sound pressure level. The sound pressure level (L) is a measure of the air vibrations that make up sound. Because the human ear can detect a wide range of sound pressure levels (from 20 micro-Pascal up to 200 Pascal), sound levels are expressed on a logarithmic scale with units of decibels (dB). An increase of 10 dB is experienced by the human ear as a doubling of the loudness of the sound

Sound level. The human ear is not equally sensitive to sounds at different frequencies. To reflect the spectral sensitivity of the human ear, spectral sensitivity factors are used to weigh the sound pressure level at different frequencies (A-filter). These, so called A-weighted sound pressure levels are expressed in dB(A).

Equivalent sound levels. When sound levels fluctuate in time, as is usual the case with community noise, the equivalent sound level is determined over a specific period of time. For this purpose the A-weighted sound level is averaged over a period of time (T), using a prescribed procedure (symbol $L_{Aeq,T}$). A common exposure period T in community studies/regulation is from 7 to 23 hours ($L_{Aeq,7-23hr}$).

Day-night level (L_{dn}). This level is used in environmental impact assessment as it correlates much better with community annoyance than the equivalent sound level. L_{dn} is the equivalent sound level over 24 hours, increasing the sound levels during the night (23-07 hours) by 10 dB(A). Also a "day-evening-night level" (L_{den}) is constructed in a similar way, increasing the sound levels in the evening (19-23 hours) by 5 dB(A) and those during the night (23-07 hours) by 10 dB(A).

Sound exposure level of a noise event. The sound exposure level (SEL) of a noise event, such as the noisy passage of an aircraft is the equivalent sound level during the event normalised to a period of one second.

Usually, the values of these metrics are assessed outdoors. In some sleep disturbance studies indoor noise levels have been measured.

In European countries, A-equivalent indices (L_{Aeq} -type) are more common than statistical indices (L_{10} -, L_{50} -type). Unfortunately, noise indices differ per country and within a country even per transport mode. Especially the indices used to describe noise exposure by aircrafts vary considerably between the different countries^{16,17}.

6.2.2 Calculation methods

Noise exposure (distribution) is usually assessed by calculations according to national noise calculation methods. In the Netherlands, for road traffic 'RMV2002' is the method, prescribed by law. Like all national European noise calculation methods, RMV2002 first calculates the noise emission by the source, taking into account the characteristics of the source (type of car, speed, type of pavement, height of the source etc.). The next step is the calculation of noise loads at the receiver.

To do so, characteristics of the noise propagation path have to be taken into account (e.g. distance, type of ground, presence and type of buildings or other objects etc.). Furthermore, air absorption, determined by the air temperature and humidity, plays a role. Corrections can be made for the meteorological conditions (temperature, wind). To estimate the number of people exposed, noise propagation models are combined with demographic data e.g. using a geographic information system¹⁸.

6.3 Reported effects on human health and well being

In this section a brief review is presented of all health and well-being impacts that have been reported to

be associated with the exposure of populations to noise. We will distinguish two types of end-points: *social-psychological* effects and *clinical* end-points that can be linked to diseases in a strict sense (e.g. by an ICD-code, see chapter 2). This review is based on a fair number of recent reviews by national and international scientific advisory committees or established scientists^{19,20,21,22,23,24,11}.

6.3.1 Social-psychological endpoints

6.3.1.1 Annoyance

Annoyance is probably the best-studied response to noise exposure, while at the same time it is probably the least well defined. Several definitions have been proposed: "a feeling of displeasure associated with any agent or condition, known or believed by an individual or group to adversely affect them"²² or "a feeling of resentment, displeasure, discomfort, dissatisfaction or offence which occurs when noise interferes with someone's thoughts, feelings or daily activities"²⁵. The degree of annoyance provoked by noise exposure depends on several characteristics, such as sound level, spectral characteristics and variations with time of the day or season; evening noise is more annoying. However, annoyance also depends on non-acoustical factors, cognitive factors. Noise exposure explains only 25-30% of observed variance in annoyance reports. These may be endogenous, such as age, psychological status, individual noise sensitivity, fear with respect to the source, perceived control over the situation, perceived economic or societal advantages of noise generating activity, or exogenous, such as poor housing, or other environmental exposures^{26,27,28}.

Noise annoyance is always assessed on the level of populations using questionnaires. Based on the results of that type of investigation exposure response relations have been derived for the three main types of transportation noise: road, rail and air traffic (see box 1). These relations pertain to populations exposed to noise at specific levels for more than a year. The effect is given as the percentage of the people highly annoyed by a specific community noise, involving those individuals in the studied population who report a degree of annoyance in the worst quarter range of answer categories^{23,29}. These results are fairly consistent on the level of populations; of course they have very little predictive value on the individual level.

People start reporting severe annoyance at L_{dn} values of around 42 dB(A), annoyance at 37 dB(A), measured at the front of dwellings. At increasing levels annoyance increases most with aircraft noise, followed by highway traffic noise, normal road traffic and railway noise. Studies have all been done in Western-European, Australia, Japan or the United States⁷. No studies from other regions are available.

6.3.1.2 Psychosocial well-being and psychiatric disorders

Several cross-sectional studies indeed revealed a higher prevalence of symptoms such as "headaches", "being tense and edgy", "social orientation", "acute symptoms of depression irritability", "burns, cuts and minor accidents" in high noise areas compared to low noise areas. A number of studies applying psychometric questionnaires to assess psychological morbidity yielded inconsistent results, including specific research of school children^{21,11,22,30}. Early studies have found associations between the level of aircraft noise and admissions to psychiatric hospitals in London and Los Angeles. Other studies, and reanalyses of old studies failed to confirm these findings. Noise exposure does not appear to be a decisive factor in hospital admission, among many other psychosocial variables^{11,20,21}.

Due to inconsistent results and inherent methodological shortcomings no definite conclusion with respect to the causal role of community noise in psychological morbidity can be drawn.

6.3.1.3 Performance

Laboratory studies reveal that noise exposure may have a significant effect on performance. While performing tasks, noise may contribute to arousal, alter (mental) task strategy, distract attention to task aspects. Noise may also affect social performance, mask speech, impair communication, and distract attention from relevant social cues. Results of several studies indicate noise may affect verbal memory tasks, and influence the process of selectivity in memory and attention.

Epidemiological studies demonstrate that equivalent sound levels during school time over 65-70 dB(A) may impair the performance of schoolchildren in cognitive tasks. Children are vulnerable to noise effects because noise may interfere with learning during a critical developmental period, and children have less capacity than adults to anticipate, understand or cope with stressors. Tasks that involve central processing and language comprehension, such as reading, attention, problem solving and memory appear most influenced by noise exposure. Prolonged exposure to noise is found to be associated with deficits in sustained attention, visual attention and concentration, with poorer auditory discrimination and speech perception, memory impairment and poor reading ability and school performance^{30,31,32}. Whether community noise is actually affecting the mental health (hyperactivity, depression, anxiety) of school children is still subject of debate, partly due to disagreement on definitions of mental health outcomes^{33,33}.

Human society depends strongly on speech, which is subject to masking by noise. Community noise, especially varying and intermittent noise, can interfere with many activities involving speech. The extent to which any particular degree of speech interference can be overcome or contribute to stress in various situations is not well understood²².

6.3.1.4 Sleep disturbance

Night-time noise may affect sleep in various ways, resulting in degradation of sleep quality (primary effects), disturbance of functioning, performance or mood the next day (secondary effects). Noise of sufficient intensity may affect the sleep *pattern*, increasing the number of people reporting difficulties falling asleep. There may also be a chronic effect, such as medication use, of prolonged stress. Sleep disturbance as a result of night-time noise is measured by various indicators, such as sleep-pattern, (self-perceived) sleep quality, attention tests (performance) or mood-questionnaires the next day. Motility as measured by wrist-actimetry is an indication for the number of awakenings during the night.

Noise increases the changes between sleep *stages* (awake, 1, 2, 3, 4, REM) and the number of awakenings during the night, respectively starting from SEL levels of about 35 and 60 dB(A). Reported sleep quality is likely to be affected at nightly noise levels above 40 dB(A). In most studies an effect of night-time noise on performance and mood the next day is only seen at levels above 60 dB(A). In large field studies often motility is measured as an important indicator of noise induced arousal and sleep disturbance. Age, sex, season, medical condition and medication are important factors of influence with regard to the level of sleep disturbance³⁴.

Night-time noise exposure may increase heart rate during the night; habituation to this effect does not seem to occur. The observation threshold is a SEL value of 40 dB(A). There are indications noise-induced increased sleep stage changes and awakenings are associated with elevated hormone levels (ephedrine)²⁵.

6.3.1.5 Absenteeism

Several epidemiological studies suggest the level of noise during working hours has an influence on the rate of absenteeism, even among office workers ($L_{Aeq,8h} > 75-90$ dB(A)). However, most studies are flawed

in several aspects and no firm conclusions can be drawn. There are no studies on community noise and absenteeism²³.

6.3.2 Clinical effects

6.3.2.1 Cardiovascular disease

Recently in a meta-analysis we reviewed studies on the association between exposure to noise and cardiovascular risks both in occupational and environmental settings. Responses included increased blood pressure, hypertension, use of anti-hypertension drugs, consultation of GP or specialist, use of cardiovascular medicines, Angina pectoris, myocardial infarction and prevalence of ischaemic heart disease¹⁰. The analysis revealed some inconsistencies among sometimes contradictory results of individual studies, and summary relative risks were statistical significant only in a limited number of end-points.

As most of the studies were of the cross sectional type, several methodological flaws could be identified, such as poor (retrospective) exposure assessment. In most studies there were limited possibilities of controlling confounding variables and selection bias (self-selection: healthy worker, healthy neighbour effect), both extremely important with respect to cardiovascular disease.

Results of a series of recent cross-sectional studies are consistent with a slight increase in the prevalence of hypertension due to noise exposure^{35,36,37,38,39}. But methodological limitations, especially with respect exposure assessment, and lack of statistical significance still do not allow firm conclusion. In most reviews the statistical evidence for a causal relation between noise exposure and cardiovascular health risk is considered to be on the verge of conclusive. However, in most of them a small effect on cardiovascular risk is deemed highly plausible, especially while the overall results on the full range of endpoints from slight elevation of blood pressure to Angina pectoris are consistent with known cardiovascular disease progression and supported by laboratory studies on blood dynamics. Nevertheless, well-designed cohort studies will be needed to confirm these indications^{21,22,23,20,10,40,41,42}.

6.3.2.2 Noise induced hearing loss

Hearing impairment is typically defined as an increase in the threshold of hearing, as assessed by audiometry. Even small values of hearing damage may affect understanding of speech in conditions of every day life. Since speech is the most common means of communication among people, hearing loss should be considered as a severe social handicap. Prolonged occupational noise exposure below a $L_{Aeq,8h}$ of 75 dB(A) is not likely to induce a permanent shift in threshold of hearing. Scarce epidemiological studies of exposure to community noise fail to demonstrate hearing damage below a $L_{Aeq,24h}$ of 70 dB(A).

Current evidence suggests the risks at typical environmental levels of exposure are low. Exposure of children and young adults to loud music during leisure activities (concerts, headphones) may give rise to concern, especially when peak sound pressure level exceed 120-140 dB(A)²³.

6.3.2.3 Other clinical health effects

There is no convincing evidence for a direct effect of exposure to noise on health outcomes such as congenital abnormalities, birth weight, or disorders related to the immune system (infectious or auto-immune disease). So far plausible mechanisms of action for these disorders are lacking²¹.

6.4 'Noisy' epidemiological data

6.4.1 Noise and signals

Among the strong and consistent health impacts of (potentially confounding) social-economically-determined factors, such as life-style or occupational health risks one will often search in vain for an independent effect of environmental exposures in available health statistics, such as mortality and morbidity rates or medical consumption (see chapter 3)^{43,44}. Furthermore, the attitude towards the source of noise, and sensitivity to noise may account for more variation than the level of noise exposure by itself^{21,26}. People who report themselves seriously annoyed might just tend to report symptoms, use self-medication or even visit a GP, irrespectively of actual noise levels. The strong association between socio-economic and environmental conditions, the expected small increases of health risks attributable to environmental exposures combined with random variation produce a 'signal-to-noise'-ratio that goes beyond the resolution of available epidemiological methods (see chapter 2). This is reflected in the fairly inconclusive nature of the results from epidemiological research on noise and cardiovascular disease.

6.4.2 Health significance

The impact of hazardous environmental exposures on human health can take numerous shapes of various severity and clinical significance. Some effects occur soon after the onset of exposure; others emerge after long-term cumulative exposure, including a latency period. Environmental exposures may induce *biochemical, physiological or (socio)psychological changes* that more or less fall within the normal range of biological variation. Whether these changes are of any significance to health depends above all on the degree to which the *function* of organ systems (or social-psychological functioning) is affected, the *reversibility* and *duration* of the changes and the possibilities for *recovery* or *compensation*, and on the possible loss of *resilience*. Acute, transient elevations of blood pressure and pulse rate, palpitations, increased serum levels of stress hormones due to noise exposure might all fall largely within normal homeostasis. On the other hand sustained haemodynamic effects, such as hypertension or prolonged stress-mediated hypercholesterolemia would surely be significant to public health. Given a certain population distribution of for instance serum cholesterol level or systolic blood pressure, even a small shift due to environmental exposure may yield a substantial increase in the prevalence and mortality of cardiovascular disease^{23,40,45,12}.

6.4.3 Harvesting?

At the level of populations these 'subtle' effects may cause some increase in acute morbidity and mortality. For instance several studies have shown some association between average noise levels and the incidence rates for myocardial infarctions¹⁰.

It is plausible that these infarctions have been initiated by 'subtle' haemodynamic changes due to noise exposure, comparable to precipitation of morbidity or mortality ('harvesting') as described with regard to air pollution episodes^{46,47}. A similar mechanism could probably apply to the association of admissions to psychiatric hospitals with traffic noise levels found in some studies⁴⁸. It is not very likely people are suffering from psychological disorders merely by noise exposure, but it might induce episodes of symptom aggravation (see figure 2-3). On the other hand one has to appreciate the fact that the very same physiological responses, such as transient elevations of stress-hormones, haemodynamic and biochemical variables, are considered beneficial when induced in physical exercise or sports²⁰.

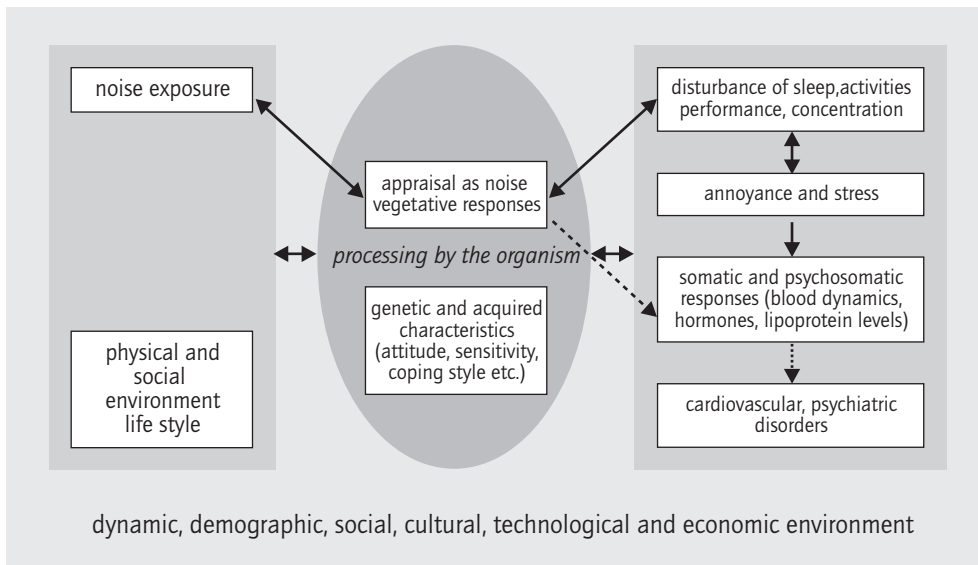


Figure 6-1. Conceptual model representing the relation between noise exposure, health and quality of life^{21,67}.

6.4.4 Well being and health

Social responses like annoyance, anxiety or perceived health risk may or may not fall within the background 'noise' of normal daily hassles. Of course at some point a clear distinction from clinically defined anxiety or depression can hardly be made. Unfortunately good longitudinal data are lacking with respect to onset and development of psychological disorders in this respect. A similar argument can be made for sleep disorders. Sleeping problems and their influence on mood and performance the next day are part of every normal life. However, at some point sleeping problems may become clinically significant as normal physical, mental and social functioning is hampered.

6.4.5 Conceptual model

An appealing conceptual model depicting the possible mechanisms involved in noise-induced disease genesis was proposed by the Health Council of the Netherlands (see figure 6-1). Noise may directly or indirectly influence vegetative, hormonal, cognitive and emotional regulation mechanisms of the organism. Indirect influences may be related to disturbed activities such as communication, recreation or sleep. The negative appraisal of noise may lead to short-term impairment of the organism, for instance through the production of stress hormones or by annoyance and resignation. Continuing noise exposure would result in chronic dysregulation of the organism. This might even include unfavourable life-style aspects, such as taking up smoking or drinking, or other risk seeking activities. On the level of populations this all may lead to an increased prevalence of chronic disease.

The model suggests that exposure to noise in itself is a health risk factor. However, various other factors may modify the way in which the "noise signal" is processed by the organism and will increase the impacts on health and well-being. Examples of such factors are the familiarity, individual or community attitude towards the noise source, and, of course, individual noise sensitivity²¹.

6.5 Approaches to assess disease attributable to noise

6.5.1 Introduction

As we have seen in chapters 2, 3 and 4 disability-adjusted life-years (DALY) may be an appropriate aggregate indicator to represent the multiform health loss due to environmental exposure to noise and thus enable comparative risk assessment. At least three important dimensions of public health are covered in the health adjusted life-years, such as DALYs, viz. loss of life expectancy, loss of quality of life, and number of people affected (social magnitude)⁴⁹. Time is the unit of measurement. Public health loss is defined as time spent with reduced quality of life, aggregated over the population involved, and combining years of life lost and years lived with disability that are standardised by means of severity weights (figure 2-4, in this diagram 'health loss' due to severe noise annoyance is suggestively added)^{ii,50}.

To assess loss of DALYs attributable to environmental exposures, information on population exposure distribution, exposure-response relationships, and incidence and prevalence rates is combined to estimate annual numbers of people affected and the duration of the condition, including premature death (see chapter 4).

From the policy maker's point of view there are several good reasons for this type of aggregation: prioritising and planning preventive actions in health and environment management, assessing performance of environmental protection system, assessing policy efficacy and efficiency, and identifying high risk populations, for instance in situations of geographical accumulation of multiple environmental exposures (see chapter 2)¹⁵.

6.5.2 Exposure assessment

In few countries highly sophisticated population exposure models have been developed for policy analysis, in which detailed source information at street, grid and city level is combined with population and built environment data in Geographic Information Systems. However in most case such models will not be available. In such cases a more crude approach might be used.

An approach for population exposure to road-traffic noise might be based on strong relations between the size of the city and the population exposure distribution. Roovers et al. proposed a simple modelling approach for the European scale that might be used as an example. Residential areas were classified into five categories of noisiness, based on both city size and regional characteristics, ranging from rural to extremely noisy. Regional characteristics involved factors such as latitude (southern cities tend to be noisier), traffic technology (engines, tires) and densities, urban traffic infrastructure, and meteorological factors (ventilation behaviour) etc⁵¹.

Through analysis of existing data for a series of European cities a crude population exposure distribution for these categories was determined. Successively, these distributions were used to assess the traffic noise exposure for the European population (see table 6-2).

ⁱⁱ To arrive at weights for this severity standardisation micro-level HRQL-measurements (e.g. health adjusted life expectancy, HALE) as well as preference-measurements (disability adjusted life-years: DALY's) can be applied. Disability free life expectancy (DFLE) is simply a special case of HALE as a threshold between healthy (1) and disabled or dead is applied (0).

Table 6-2. Traffic noise exposure distribution of EU inhabitant⁵¹.

	noise level inhabitants	<55dB(A) L _{DN} %	L _{DN} mln	55-65 dB(A) L _{DN} %	L _{DN} mln	65-75 dB(A) L _{DN} %	L _{DN} mln	>75 dB(A) L _{DN} %	L _{DN} mln
EU	371.602.000	68	251,3	19	71,2	11	44,4	2	7,7

Data on exposure to aircraft noise are even less available in general. Even in the EU data on population exposure are scant and unreliable. A first approach would be a crude estimate of exposure based on the size of the airport.

For the purpose of this exercise we use population exposure distributions for cumulative noise data modelled in the framework of the Dutch Environmental Balance², expressed in different measures, as shown in table 6-3.

Table 6-3. Examples of cumulative noise distributions for the Netherlands.

Exposure dB(A)	L _{24h} % of population	L _{den} % of pop.	L _{night} % of pop.	L _{Aeq(7-19)} % of population used for cardiovascular end-points assessment	
<40	4	5	43		
41-45	6	7	31		
46-50	14	17	19	51-54	25
51-55	27	29	5	55	5
56-60	30	27	1	56-60	15
61-65	15	11	0	61-65	4
66-70	4	3	0	66-70	1
71-75	1	0	0	71-72	0

Source: EMPARA; Environmental Balance 2004, Environmental Assessment Agency, RIVM.

6.6 Different approaches for disease burden estimation

Given the current quantitative insights in health and well being responses to noise exposure, the strength of the evidence and starting points with respect to definitions of health and health loss, there are basically four manners in which quantitative assessments of noise related disease burden could be achieved. The impact assessment can be based on:

- severe annoyance prevalence,
- sleep disturbances (both as proxies for decreased quality of life),
- noise related population attributive risk for cardiovascular disease
- a noise induced shift of systolic blood pressure distribution or increased hypertension prevalence as risk factors for cardiovascular disease.

The latter two should be considered scenario-wise: what if the indications for an association between noise exposure and cardiovascular disease are true?

6.6.1 Uncertainty assessment

To analyse the uncertainty in the calculations of environmental disease burden we have often applied Monte Carlo techniques (see chapter 4). However in the case of noise health impact assessment uncertainty may just as well be tested by simple well-reasoned worst/best (or what-if) case calculations, using lower, central and high estimates for the model parameters⁵².

6.6.2 Annoyance-based impact assessment

For noise annoyance ample quantitative exposure response relationships are fairly well established. Based on a pooled analysis of original datasets from noise-annoyance surveys carried out in Europe, Australia, Japan and North-America, exposure response relationships have been derived for road, rail and air traffic noise⁷. These curves have been derived from probably the most elaborate database currently available and can be used to predict the number of annoyed people in an exposed population (in general three degrees of annoyance are defined: 'little', 'moderate' and 'severely' or 'highly annoyed'). They are recommended for use in the EU Directive on the Assessment and Management of Environmental Noise⁵³. It has to be noted that there is still debate on the procedures used for meta-analysis. Furthermore the differences between the three noise sources may be due to methodological differences in the original studies (e.g. poor exposure assessment, differences in adequacy noise insulation^{54,55}).

The fraction of severely annoyed people for sound levels in L_{dn} en L_{den} can be estimated using the analyses of Miedema and Oudshoorn (see Box 1.).

Box 1. Exposure response relations for severe noise annoyance (HA) for L_{DN} en L_{DEN}

Aircraft: $\%HA = -1.395 \cdot 10^{-4} (DNL-42)^3 + 4.081 \cdot 10^{-2} (DNL-42)^2 + 0.342 (DNL-42)$;
 Road traffic: $\%HA = 9.994 \cdot 10^{-4} (DNL-42)^3 - 1.523 \cdot 10^{-2} (DNL-42)^2 + 0.538 (DNL-42)$;
 Railways: $\%HA = 7.158 \cdot 10^{-4} (DNL-42)^3 - 7.774 \cdot 10^{-3} (DNL-42)^2 + 0.163 (DNL-42)$.

Aircraft: $\%HA = 9.199 \cdot 10^{-5} (DENL-42)^3 + 3.932 \cdot 10^{-2} (DENL-42)^2 + 0.2939 (DENL-42)$;
 Road traffic: $\%HA = 9.868 \cdot 10^{-4} (DENL-42)^3 - 1.436 \cdot 10^{-2} (DENL-42)^2 + 0.5118 (DENL-42)$;
 Railways: $\%HA = 7.239 \cdot 10^{-4} (DENL-42)^3 - 7.851 \cdot 10^{-3} (DENL-42)^2 + 0.1695 (DENL-42)$.

If the number of severely annoyed people is estimated, the next step would be to assess the disease burden. Within the concept of health adjusted life-years this can be done by simply multiplying the number of people reporting to be severely annoyed with a severity weight factor discounting for the disease state "severely annoyed", thus yielding an estimate of the annual 'health' loss (duration 1 year). In the framework of an environmental burden of disease assessment a panel of health professionals defined a severity weight for severe noise annoyance in comparison with other diseases or limitations in functioning (see Box 2)^{56,52}. However, it is noted here that not everyone would agree that severe annoyance should be regarded as health loss in the strict sense, but at the same time it could lead to various symptoms or a limitation in functioning regarding daily tasks or activities.

Box 2. A severity weight factors for noise exposure

'Severe annoyance'

In an environmental disease burden study in the Netherlands "severely annoyed" was selected as a health significant endpoint and evaluated following the protocol of the Dutch Burden of Disease Study³⁷. A definition of severe annoyance was included in a severity weigh exercise to arrive at discount factors. The following definition was evaluated by a group of environmental physicians, epidemiologists and public health professionals n=35). "*Regularly (and partly inescapably) persons are disturbed during daily activities, such as having conversation, listening to the radio, watching television, reading a book or a magazine. Furthermore persons may have feelings of resentment, displeasure, discomfort, dissatisfaction or offence, occurring when noise interferes with their thoughts, feelings or daily activities*". This definition was presented together with a Euroqol 5D+ description to assist in structuring the evaluation (see chapter 2 and 4).

- No problems walking about (mobility)
- No problem with washing or dressing oneself
- No problems with usual activities (work, study, housework, family of leisure activities)
- No pain or discomfort
- Not (95%) or mildly (5%) anxious or depressed
- No (95%) to some (5%) cognitive impairment (e.g. problems with memory, concentration, disorganisation, IQ-level).

The respondents evaluated the health states by means of interpolation using indicator conditions ranked on a disability scale (see figure 5.4). The severity factor derived in this exercise was used to discount for the health state "sever annoyance". This procedure yielded a severity weight factor of 0.12 (standard deviation: 0.16, median 0.07).

Severe sleep disturbance

The following definition was evaluated by a group of environmental physicians, epidemiologists and public health professionals (35). *"Regularly persons are disturbed in their sleep, manifest as an increase of awakenings and/or shifts in sleep stages during the night. This affects the perceived quality of sleep and their mood the next day. During the day they might experience sleepiness, slight fatigue, and concentration difficulties"*. This definition was presented together with the following Euroqol 5D+ description to help the respondents structure their evaluation.

No problems walking about (mobility)

No problem with washing or dressing oneself

No problems with usual activities (work, study, housework, family or leisure activities)

No pain or discomfort

Not anxious or depressed

No (90%) to some (10%) cognitive impairment (e.g. problems with memory, concentration, disorganisation, IQ-level).

The respondents evaluated the health states by means of interpolation using indicator conditions ranked on a disability scale (see figure 5.4). The severity factor derived in this exercise was used to discount for the health state "highly disturbed". This procedure yielded a severity factor of 0,1 (standard deviation: 0.1, median: 0.08)⁵⁶.

Calculations of the burden of noise annoyance are fairly simple applying the above mentioned exposure response relations on a given population noise exposure distribution (see table 6-4)⁵⁸. Based on the exposure-response curves from box 1 it can be calculated that the current noise exposure distribution in the Netherlands produces around 64,000 severely annoyed persons per million (around 6.4% of the population). Applying a severity weight of 0.07 (median) this would mean an annual disease burden of around 4,500 disability (severe annoyance) adjusted life years.

In this example we used the exposure response curve for the percentage of *highly* annoyed persons due to road traffic^{iii,iv}. Doing the calculations with the range of reported severity weights would be another way to investigate the uncertainty of this type of calculations, clearly showing a high sensitivity of the calculations to attributed severity weights (ranging from 0.00 to 0.33 among 34 professional respondents). In a preliminary Swiss study among 41 physicians disability weights of 0.033 and 0.055 were assigned to communication and sleep disturbance respectively⁵⁹; in an earlier disease burden study we applied 0.005 (0.00-0.02), being the least severe category of the first GBD-study (see chapter 4)^{60,2}.

ⁱⁱⁱ In case no specific noise exposure distributions are available one could consider to use curves for railroad noise (lowest annoyance potency) and aircraft noise (more potent) to calculate a range between lower and higher estimates of the fraction severely annoyed: here 2.5-10% of the population.

^{iv} These estimation are in reasonable accordance with the results of a recent survey (3% for private cars – 10% for truck traffic); however 19% of the respondents reported themselves to be severely annoyed by the noise of mopeds⁵⁸.

Table 6-4. Example of spreadsheet calculation of number of severely annoyed people per million, based on exposure distribution in the Netherlands (cut-off 'severe': 28% most annoyed, Miedema et al., 2002)

exposure category dB(A)	average L _{DEN} dB(A)	exposure distribution: % of population	% severely annoyed	number/ 1000.000
<40	40	5	0,0	0
41-45	43	7	0,5	364
45-50	48	17	2,7	4,535
51-55	53	28	5,4	14,983
56-60	58	26	21,3	22,922
61-65	63	11	13,8	15,098
66-70	68	2	21,3	5,279
>71	73	0,3	31,8	1,011
total		100		64,193

6.6.3 Sleep disturbance based impact assessment

In a similar way the fraction of people experiencing severe sleep disturbance can be assessed. However, there is much less consistency in and consensus on appropriate exposure response functions. Based on an analysis of original data from 15 datasets (12 field studies, 12000 observations), relationships have been proposed (box 2) that give the percentage highly sleep disturbed (*%HSD*), sleep disturbed (*%SD*), and (at least) a little sleep disturbed (*%LSD*) by road traffic and railway noise as a function of the outdoor L_{night} at the most exposed façade. Sleep disturbance questions vary a lot between surveys, in wording and in the number of response categories.

Box 3. Exposure response relations for sleep disturbance for L_{night}

Road traffic:	$\%HSD = 20.8 - 1.05L_{night} + 0.01486L_{night}^2$ $\%SD = 13.8 - 0.85L_{night} + 0.01670L_{night}^2$ $\%LSD = -8.4 + 0.16L_{night} + 0.01081L_{night}^2$,
Railways:	$\%HSD = 11.3 - 0.55L_{night} + 0.00759L_{night}^2$ $\%SD = 12.5 - 0.66L_{night} + 0.01121L_{night}^2$ $\%LSD = 4.7 - 0.31L_{night} + 0.01125L_{night}^2$,

In order to obtain comparable disturbance measures the sets in the selected studies were translated into a scale from 0 to 100. Cut-off points to assess the percentage of highly sleep-disturbed persons were used analogue to the definitions of the percentage (highly) annoyed persons. No relationships for aircraft noise were proposed because of the large variance in results.

Box 3 presents the curves Miedema et al. proposed for the percentage people reporting to be highly sleep disturbed (*%HSD*), sleep disturbed (*%SD*), and (at least) a little sleep disturbed (*%LSD*) as a function of the outdoor L_{night} at the most exposed façade for the source concerned³⁴.

The exposure-response relationships presented in box 3 can be used to calculate the prevalence of highly disturbed in a given population. To estimate the burden of disease from sleep disturbance, a severity weight has to be defined (see box 2).

Calculation of sleep disturbance burden is similar to the calculation of annoyance burden, applying the exposure response relations on a given population noise exposure distribution (L_{night}-distribution). Using the exposure response curve for the percentage of *highly* sleep disturbed persons due to road traffic (cut-off: ≥35 dB(A)) a fraction of 0.7-1.4% can be calculated to be highly sleep disturbed as a result of cumulative community noise exposure. Using a severity factor of 0.8 (median) this number yields an

annual burden of 550 - 1,100 disability ('severe sleep disturbance') adjusted life-years'/million. Again, these calculations are a highly sensitivity to attributed severity weights (ranging from 0,0 to 0.40 among 35 professional respondents).

6.7 Impact assessment based on cardiovascular endpoints

6.7.1 Epidemiological data

Meta-analysis of the epidemiological research on the effects of community noise on cardiovascular health yields few estimates for the calculation of noise attributable fractions of cardiovascular disease (see table 6-5). They are defined as 'day-time' levels $L_{Aeq, 7-19h}$, $L_{Aeq, 6-22h}$, $L_{Aeq, 8h}$ in dB(A) for aircraft, road traffic and occupational exposure, respectively. For aircraft noise the analysis produced a statistically significant RR-estimate for hypertension (defined as systolic BP > 160 mmHg, diastolic BP > 95 mmHg), and a borderline significant estimate for the use of cardiovascular drugs. For road traffic the results of three studies are contradictory (see table 6-5). The RR-estimate for IHD prevalence is clearly statistical significant (but based on two cross-sectional studies) and the RR-estimate for past myocardial infarction is borderline significant. The results of several recent studies are consistent with a small effect on hypertension (see above)^{35,36,37,38,39}. We emphasise that the following health impact assessments should be regarded as a scenario-like exercise: *what* is the public health significance of current community noise exposures *if* the cardiovascular connection were to be true?

Table 6-5. Summary estimates for the association between noise exposure, hypertension, and ischemic heart diseases, adjusted for sex and age, expressed as $RR_{5\text{ dB(A)}}$ (increase per 5 dB(A))¹⁰.

Noise exposure ^a	Outcome	$RR_{5\text{ dB(A)}}$	95% CI ^d	number of estimates ¹⁰	Measurement range (dB(A))
Occupation	Systolic blood pressure mmHg/5dB(A)	0.51	0.01 - 1.00	14	55 - 116
Occupation	Hypertension ^b	1.14	1.01 - 1.29 *	9	55 - 116
Road traffic	Hypertension	0.95	0.84 - 1.08	2	<55 - 80
	Use of antihypertensives	0.96	0.76 - 1.22	2	>50 - 73
	Consultation of GP/specialist	0.91	0.73 - 1.12	1	55 - 70
	Angina Pectoris	0.99	0.84 - 1.16	2	51 - 70
	Myocardial Infarction ^c	1.03	0.99 - 1.09	3	51 - 80
	IHD-total ^c	1.09	1.05 - 1.13 *	2	51 - 70
Air traffic	Hypertension	1.26	1.14 - 1.39 *	1	55 - 72
	Use of anti-hypertensives	0.99	0.87 - 1.14	1	55 - 72
	Consultation of GP/specialist	1.10	0.95 - 1.27	2	55 - 77
	Use of cardiovascular drugs	1.05	0.99 - 1.11	2	38 - 77
	Angina Pectoris	1.03	0.90 - 1.18	1	55 - 72

^a The noise exposure measures differed between the noise exposure sources: occupational noise exposure expressed in $L_{Aeq, 8hr}$ in dB(A), road traffic noise exposure expressed in $L_{Aeq, 6-22h}$ in dB(A) and air traffic noise exposure expressed in $L_{Aeq, 7-19h}$ in dB(A).

^b Adjusted for age, sex and worktype.

^c Ischaemic heart disease (ICD-9:410-414), only prevalence estimates.

^d CI = Confidence Interval * Significant, p<0.05.

6.7.2 Calculation of cardiovascular disease attributable to noise

For estimating the burden of cardiovascular disease due to noise we make no qualitative distinction between aircraft and road traffic noise, although annoyance studies show they may be processed in different ways. We simply use the combined noise levels from the Dutch national noise calculation system (EMPARA) in $L_{Aeq, 7-19}$ dB(A) as exposure measure (which comprises mainly road traffic) and is in fair accordance with the other two definitions.

To estimate the number of people affected we calculated population attributable risks (PAR's) by combining population exposure distributions (see table 6-3) with quantitative exposure-response information, applying the following equations:

$$RR_i = e^{\frac{(L_i - L_{cut-off}) * \beta}{5}}$$

$$PAR = \sum \frac{p_i * (RR_i - 1)}{p_i * (RR_i - 1) + 1}$$

in which RR_i = relative risk in exposure class i and p_i = exposure probability in class i , L_i = exposure level in class i , expressed in dB(A), $L_{cut-off}$ = cut-off (e.g. 54 dB(A)), and β is the risk function estimate from the meta-analysis (see table 6-5).

Subsequently we estimated the noise attributable prevalence of cardiovascular endpoints by multiplying the PAR with annual prevalence data, obtained from Dutch health statistics⁶¹.

Table 6-6. Estimation of community noise attributable cardiovascular endpoints for the Dutch situation, per 1000,000 inhabitants per year; exposures ≥ 55 dB(A), exposure distribution in L_{Aeq} (7-19 h)

Cardiovascular endpoint (P_0)	RR-estimate (95%-CI) from meta-analysis ¹⁰	noise attributable number / 1000,000	results using lower and upper RR-estimate	
Hypertension prevalence (0.196)	air traffic: 1.26 (1.14-1.39)	11,500	6,000	18,000
Hypertension prevalence (0.196)	occupation: 1.14 (1.01-1.29)	6,200	440	13,000
Ischaemic Heart Disease prevalence (0.035)	road traffic: 1.09 (1.05-1.13)	700	390	1,020
Past myocardial infarct prevalence (0.017)	road traffic: 1.03 (0.99-1.09)	110	0	340

In table 6-6 results are presented of simple calculations of the noise exposure attributable numbers of attributable prevalent cases of hypertension, ischaemic heart disease and past myocardial infarction per 1000,000 inhabitants, assuming that the exposed population is similar to the total population. Attributable fractions were calculated based on the L_{Aeq} (7-19h)-exposure distribution for the Netherlands and RR-estimates from air traffic, road traffic and occupational studies, respectively (cut-off at ≥ 55 dB(A), including around 25% of the Dutch population, see table 6-3).

These calculations are highly sensitive to the choice of cut-off point. Applying a cut-off point of 51 dB(A) instead of 55 dB(A) increases the number of attributable prevalent IHD or past myocardial infarction cases with a factor of 2.6, especially due to the substantial increase of the fraction of the population exposed to 'effective' levels (see table 6-3, 25% in class 51-54 dB(A)).

6.7.3 Impact assessment of cardiovascular disease burden

6.7.3.1 *Ischaemic heart disease burden*

The most straightforward approach to estimate disease burden attributable to community noise is calculating the disability adjusted years (YLD) associated with noise attributable *ischaemic heart disease* prevalence (see table 6-6), over one year, using the aggregate severity weight from the Dutch Burden of Disease study (0.288)⁶². This yields an annual number of 200 DALYs (110-295) per million. If we assume these cases will eventually die, having the same loss of life expectancy as general cases, we can also approach noise associated disease burden with the attributable fraction of coronary heart disease burden per million in the Netherlands (around 22,000 DALYs/year): 445 DALYs (245-640)/year/million.

6.7.3.2 *Mortality due to noise attributive hypertension*

As discussed in section 6.3.2.1 it is assumed that noise exposure has an effect along the overall process of cardiovascular disease genesis. Positive associations are found for a number of manifestations of cardiovascular disease often in individual studies: elevated blood pressure, hypertension, cardiovascular medication use (including hypertensives), consultation of the general practitioner or cardiologist, prevalence of Angina pectoris and/or ischaemic heart disease¹⁰. Following the reasoning behind the conceptual model (figure 6-1) we assume that the risk elevations associated with noise exposure for these different endpoints are a fair indication of a small contribution to total disease prevalence.

From this perspective, besides calculating noise attributable IHD, one could also approach diseased burden by calculating the noise population attributable fraction of annual hypertension population attributable mortality.

$$PAR_{\text{mortality/noise}} = PAR_{\text{mortality/hypertension}} * PAR_{\text{hypertension/noise}}$$

In this exercise we applied a RR-estimate of 1,4 for total mortality risk in a population with hypertension relative to a population without⁶³. Given the current noise exposure distribution for the Netherlands an attributable fraction of total mortality can be estimated: 0.0059, representing 38 cases per million inhabitants. Assuming a loss of life expectancy of around ten years associated with general cardiovascular cases this yields an annual loss of 380 (200-580) DALYs (YLLs) per million. Again these calculation are sensitive to the choice of cut-off point.

6.7.3.3 *Noise attributable increase in systolic blood pressure*

Another way of crude estimation is applying the adjusted estimate for noise attributable increase of usual systolic blood pressure derived from a slightly more solid epidemiological database: a meta-analysis of 14 occupational studies, yielding an estimate for a noise induced increase of (usual) systolic blood pressure: 0.01-1.0 mmHg for every 5dB(A) increase in noise exposure (see table 6-5). Of course in occupational studies risk estimates are based on 8-hour measures ($L_{Aeq, 8h}$ -exposure measure). However this distribution is fairly similar to the $L_{Aeq (7-19 h)}$ -distribution we have used so far. This exposure-response function allows the calculation of a population attributable fraction for total annual disease burden based on a noise attributable shift in (usual) systolic blood pressure.

Within each relevant exposure category (≥ 55 dB(A)) this shift results in an elevated relative risks for ischaemic heart disease and stroke. Age and gender specific RR-functions for ischaemic heart disease and stroke were taken from the Asia Pacific Cohort Study (a meta-analysis comprising data of over 425,000 individuals and over 3 million observation years)^{64,65,66}. Similar to the global burden of disease study that

was done for lead exposure, we applied a relative risk of 1.057 per mmHg increase in systolic blood pressure (SBP) for ischaemic heart disease and 1.066/mmHg for stroke (adjusted Dutch population characteristics). Using 0.51 (0.1-1.0) mmHg as a crude estimate of population SBP-shift per 5 dB(A) noise attributable disease burden in the Dutch population for ischaemic heart disease is 140 (3-270) and for stroke^v 88 (2-169) DALYs. However, no clear indications for an association between noise exposure and stroke have been reported so far.

Table 6-7. Summary of results of crude estimations of annual noise attributable disease burden: DALYs/1000,000 inhabitants

Type of estimate	central (DALYs/million)	lower (DALYs/million)	upper (DALYs/million)
'annoyance adjusted life years'		640 *	4,500
'sleep disturbance adjusted life-years'		60 *	1,100
IHD based on prevalence years (YLD)	215	125	305
IHD based (YLD+YLL)	390	225	550
Noise attributable hypertension mortality	380	200	580
IHD and CVA based on BP-shift (DALY)	230	5	450

* applying 0.01 as disability weight factor, being the least severe category from the first Global Burden of Disease Study

The results of the proposed exercises of tentative disease burden assessment are presented in table 6-7. Noise attributable loss of 'disability' adjusted life-years is potentially largest for the social psychological endpoints 'severe annoyance' and 'severe sleep disturbance'. Estimates for noise attributable cardiovascular disease are all in the same order of magnitude (200-600 DALYs/year/million), somewhere between 0.1 and 0.2% of total disease burden.

6.8 Discussion

6.8.1 In case of a causal association: a small, but significant disease burden

Assuming the indications for an association between community noise exposure and cardiovascular disease are true, a small but significant disease burden can be attributed to current exposure levels in the Netherlands (no more than 2% of total ischaemic heart disease burden). Given the definition of the exposure distribution, obviously this assessment does not include a possible effect of night-time noise. There are some indications that night-time noise is more effective with respect to cardiovascular endpoints, such as hypertension³⁷. At the same time these explorative calculations show that the social-psychological impacts of community noise exposure may constitute a substantially greater burden to health and well-being.

These explorative health impact assessment exercises are to some extent acceptable if we take into account that the epidemiological database on a range of observed end-points is fairly consistent with known cardiovascular disease progression. Small transient, stress related haemodynamic responses to noise exposure may in the end result in a slight increase of cardiovascular risk at level of populations^{10,41}. The proposed approaches at least provide some sense of the order of magnitude of possible noise attributable health loss, in case the associations are true: clearly a 'what if scenario approach. This may help giving community noise exposure its proper place on the environmental agenda. Furthermore, it cannot be ruled out that the next well-performed cohort study may reveal a greater effect of community noise on cardiovascular disease incidence.

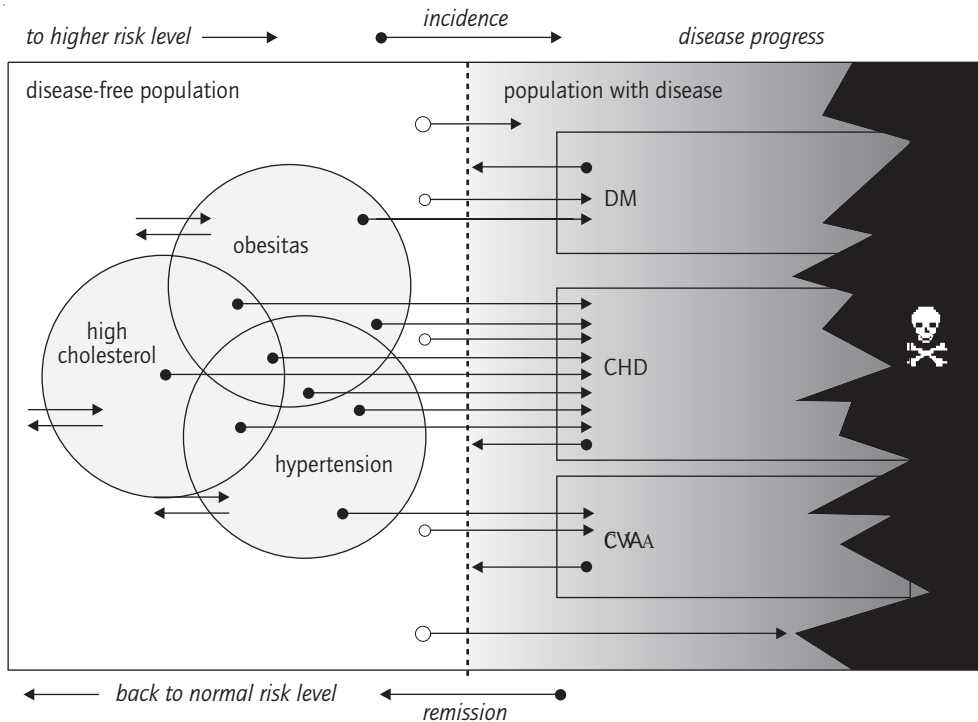


Figure 6-2. Diagram representing disease burden calculations with respect to noise induced blood pressure shift. A shift in blood pressure on a population level would result in an increased number of persons with hypertension, increasing the incidence and thus prevalence of patients with coronary heart (CHD) disease or stroke (CVA), increasing disease specific mortality (and loss of life years).

6.8.2 Different sources of noise

Due to lack of good data in the examples of health impact calculations we could not properly take differences between noise from different environmental sources into account. Meta-analysis of studies of noise related annoyance suggests there is a difference in the way different sources of noise are 'processed' by exposed individuals. At the same level of exposure aircraft noise appears to provoke more annoyance than road traffic noise; railway noise causes the least effect. And of course their phenomenology can be quite different. On the other hand, there are some indications that differences in exposure response relations for different transport sources may be due to differences in methodology of the original studies, such as exposure assessment (e.g. taking noise insulation of dwellings into account)^{54,55}.

6.8.3 The severity of annoyance

From comfortable hindsight one might question the appropriateness of the definition of 'severe annoyance' (box 2) applied in the weight-attributing exercise. This definition that we took from Health Council reports on noise^{25,67}, may be slightly overstating the condition people report in the daily practice of large-scale noise-surveys: annoyed by noise on a scale from zero to ten. Of course a fraction of the population, in particular typically noise-sensitive people, will experience to be *'regularly (and partly inescapably) disturbed during daily activities, such as having conversation, listening to the radio, watching television, reading a book or a magazine and thus have feelings of resentment, displeasure, discomfort, dissatisfaction or offence, occurring when noise interferes with their thoughts, feelings or daily activities'*. However it is hard

to conceive people being disturbed in conversation, listening to the radio due to exposures as low as of 42 dB(A) at the facade of their homes, resulting in substantially lower levels inside. Other, non-acoustical factors may play a significant role as well in the reported annoyance.

Moreover we assumed people to be in a state of 'severe annoyance' throughout the whole year. It is likely that the reported severe annoyance may concern much more restricted periods, such as 'sunny' weekends in which people tend to pass time outside the house or go out for a walk and are thus much more bothered by community noise.

It is reasonable to assume that the application of the severity weigh factors from the official weighing exercises tends to produce relatively high noise attributable 'disease burden' associated with annoyance. The same applies to a lesser extend for sleep disturbance. Therefore, it might be a good suggestion to implement some form of severity weight exercise into noise annoyance surveys to produce more realistic estimates for disability weight factors.

6.8.4 Uncertainties

In these types of integrated assessments many substantial uncertainties are accumulated. This may pertain to available data for modelling (criterion validity) as well as to the impact assessment models themselves (construct validity). With respect to criterion validity 'what-if' or Monte Carlo analysis is a simple and adequate way of providing probability distributions for the outcome variables. However, in this case many of the uncertainties may concern the construct validity, the validity of the exposure-response relations themselves. This means much effort will have to be put into conveying the right message to policy makers and the public. As is often the case, the association between noise and 'annoyance' has a high criterion and construct validity, but the public health significance (content validity) is more controversial. The other way round, the clinical significance of cardiovascular disease is obvious, but a causal relation with noise exposure is not yet fully supported by data or scientific consensus.

6.8.5 An impossible choice of endpoints?

So, which of the proposed endpoints should be applied in comparative risk assessment to support environmental and spatial policy? *Numbers* per million for a conceptually consistent range of social-psychological and cardiovascular endpoints (table 6-5 and 6-6) clearly indicate that public health may be at stake, but is less appropriate in a framework of comparative risk assessment. The calculations of attributable burden of cardiovascular disease (*DALYs*) indicate a small, but clinically significant health loss of less than 2% of total ischaemic heart disease burden and less than 0.1% of total national disease burden⁶⁸. At the same time these values are the result of a 'what if the epidemiological data are valid' scenario based on the best available data. Besides that, we have calculated a burden expressed in 'severe annoyance' adjusted life-years, which –when allowed in the same league as pure clinical health endpoints-, may amount to 2% of total disease burden, especially depending on attributed severity weights.

In chapter 3 we argued in favour of stakeholder involvement in risk management procedures when risk analysis is highly uncertain and fundamentally normative choices have to be made. The community noise problem may just be a good example for such an approach. There is still much uncertainty, but awaiting better science, we could facilitate the decision process by clearly framing the problem and calculating the results of different options. The various exercises proposed here, could have a meaningful place in such a procedure.

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6.9 References

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