Health Aspects of Extra-Aural Noise Research

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The WHO definition of "health" is critically discussed in its broad context. Decision making in noise policy has to be made in the evaluation range between social and physical well-being. The term "adverse" is a crucial one in the process of risk characterization. In toxicological terms it refers to the single event itself; in psychosocial terms it refers to the relative number of people affected. The evidence of the association between community noise and cardiovascular outcomes is evaluated. The results of epidemiological studies in this field can be used for decision making when assessing maximum acceptable noise levels in the community. Since dose response relationships were mostly studied with respect to road traffic noise, inferences have to be made with respect to aircraft noise. Issues of statistical inferring are discussed.

Keywords: Risk assessment, Traffic noise, Aircraft noise, Health effects, Cardiovascular risk, Evidence

Health and noise

Health is man's most valuable protective asset. Noise interferes with his activities, evokes annoyance and is emphatically rejected by many people (VDI 3722 Blatt 1, 1988, WHO European Centre for Environment and Health, 1995, Miedema and Vos, 1998). Would it not be reasonable, therefore, to refer to the findings of social surveys as the major basis of action programs for noise policy? According to the WHO definition, health is included as an essential component in man's general contentedness with life (WHO, 1948):

"Health is a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity."

This is a clear statement that health requires a view of the organism integrated with its environment. It is expressed with even more emphasis in the European Charter on Environment and Health (WHO Regional Office for Europe, 1990):

"Good health and well-being require a clean and harmonious environment in which physical, psychological, social and aesthetic factors are all given their due importance. The environment should be regarded as a resource for improving living conditions and increasing well-being."

Without wishing to belittle the quality standards expressed in the WHO statements in their significance for global objectives, experience has shown that these propositions are applied practically for all well-meaning measures, thus losing some of their argumentative strength in the implementation of specific objectives. The practical policy of environment and health need specifications and criteria expressed essentially in more concrete terms for the process of decision-making. They have to be in a position to set priorities within the constraints of limited resources, and deal with cost/benefit and assets considerations suitable for providing the highest measure of environmentally relevant health protection within the framework of possibility (Brown, 1985, Cleland-Hamnett, 1993). In this process they compete with other interests and objectives of society. Rohrmann has drawn attention to this conflict (Rohrmann, 1993):

"Critical limits for environmental stressors cannot be derived from empirical sciences. They are socio-political settings that depend on the system of values of all the groups involved. Limiting values express a normative act as a

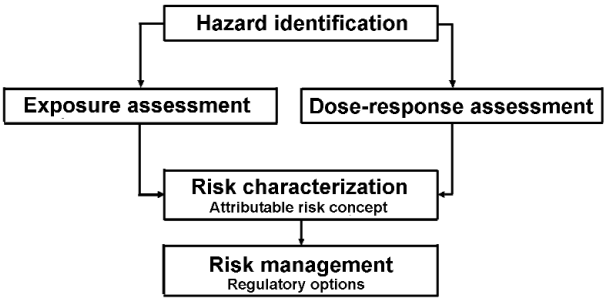


Figure 1. The process of risk assessment

result of complex considerations about benefits, risks and cost." (Translated from German)

Standard values in a community are in principle fluctuating and dependent on its economic possibilities. Individual and group interests, controlled or uncritically inquired about, as well as anxieties about the environment, determine jointly the formation of public opinion in this process.

This clearly outlines the part played by health-orientated noise effects' research. (N.B., that at this point and in the following, the term "health" is strictly used only in the somatic sense of healthiness.) In noise hygiene we move within an assessment spectrum, the anchors of which may be variously described by the key issues of "comfort" (of quiet) and "health" (somatic) or we may say "annoyance" and "disease" (Lindström, 1992, Jansen et al., 1996, Babisch, 2000a). Whereas social science research is more inclined to consider the assessment yardsticks at the lower end of this continuum, it is generally speaking the task of health relevant noise effects research to mark out the upper limits of permissible/acceptable exposure to noise (Fischer, 1999). Formulation of the objectives of protection in the "German Federal Protection From Immissions Act" explicitly distinguishes between warding off (health) hazards and substantial disadvantages or annoyance (BImSchG, 1990, Fischer, 1999).

With strict interpretation one could say that the quality objectives derived by the social sciences, dependent on cost/benefit considerations, are variable and to some extent "negotiable", whereas those derived in terms of health (somatic) ought to be more fixed and "obligatory". Reality shows, nonetheless, that even quality objectives as defined by health are subject to the changing values of a community. In general it can be assumed as a starting point that the environmental standards derived by the social sciences refer to lower noise levels than those derived by health considerations. However, should guideline or limiting values be founded on health risks, the question may be raised as to whether it is ethically correct to make different demands depending on regional utilization. In view of widely developed planning and licensing regulations with regard to traffic and industrial noise, the results of health relevant noise effects research is of particular importance for noise cleanup and the discussion about aircraft flight-noise legislation.

Risk and risk assessment

The term "risk" represents an essential factor in the entire process of evaluating health effects of potential environmental noxae (WHO Regional Office Europe, 2000), comprising the following three levels of data evaluation:

* *Hazard identification*
  (Which health outcome is relevant?),
* *Exposure assessment*
  (How many are how severely affected?),
* *Dose-response assessment*
  (Is there a threshold of effect?),

This is illustrated in Figure 1 (National Research Council, 1983, Neus and Boikat, 2000). From the evaluated results, a risk assessment is carried out ("risk characterization") (Patton, 1993). Examination of the scientific evidence of available data requires a critical discussion of the validity of the studies (influence of chance and systematic errors (bias)), their transparence and the complete literature used (Neus and Boikat, 2000, WHO Regional Office Europe, 2000). A

quantitative effect estimator has to be calculated (e.g. regression coefficient, relative risks) which then – with reference to the population ("attributable proportion") - serves as key information for any follow-up "risk management" (Jasanoff, 1993, Walter, 1998).

In public discussions of environment and health hazards the application of the statistical term "risk" often proves to be problematic. In colloquial language it is understood as a synonym for "danger", thus making competent reasoning rather difficult (Fülgraff, 1992). While the term "danger" is used in defining a qualitative relationship between exposure factor and health, the term "risk" is used in the same relationship for a quantitative assessment (Zeger, 1991). The statistical risk provides the probability for a certain damage occurring at a given point in time, either on the grounds of chronic exposure, or resulting from an acute event. Taking risks is a matter-of-course in everyday life. In this respect, decision making either as an individual or for a community involves risk taking (Moghissi, 1993). This may be seen in reference to the above mentioned system of values and standards prevailing in communities. The WHO-guidelines for drinking water, for instance, tolerate a cancer attributable lifespan and death outcome risk of $1:10^5$ (WHO, 1993), reflecting a socially accepted background risk (Scheuplein, 1993, Dieter and Grohmann, 1995). The German "Federal State Committee for Immission Protection" (Länderauschuss für Immissionsschutz – LAI), however, has established as temporarily adequate a limitation on the total risk of carcinogenic toxic air particles as a lifespan risk of 1:2500 taking into account the realistic background concentrations in densely populated areas (in rural areas: a lifespan risk of 1:5000) (BAGS, 1995, Fischer, 1999).

Considering the diverse effect of outcomes, each combined with diverse severity of grades, and the differing impairments to life quality, it appears to be rather problematic - as seen from the current state of research - to compare or apply any risk evaluations when drawn from various environmental media (Bachmann and Konietzka, 1999, Dieter, 1999, Fischer, 1999, Hapke, 1999, Martignoni and Burkart, 1999, Wichmann and Ihme, 1999). If premature death is determined as an effect outcome, then lost years of life would induce quantitative risk comparisons based on differing induction times and latencies of diseases and their relationship to age. Regarding quantitative "risk assessments", noise-related risk discussions are certainly still in their beginnings.

For risk assessments of environmental factors and the setting of environmental standards, the severity and prevalence of health outcomes play an essential part. This is outlined in Figure 2 and was taken from the "Handbuch der Umweltmedizin" (Wichmann and Ihme, 1999), and adapted for the issue of noise effects (Babisch, 2002). Since the diagram was originally designed for chemical exposures, the lowest effect grade ("internal exposure") was substituted by "annoyance". Unlike chemical

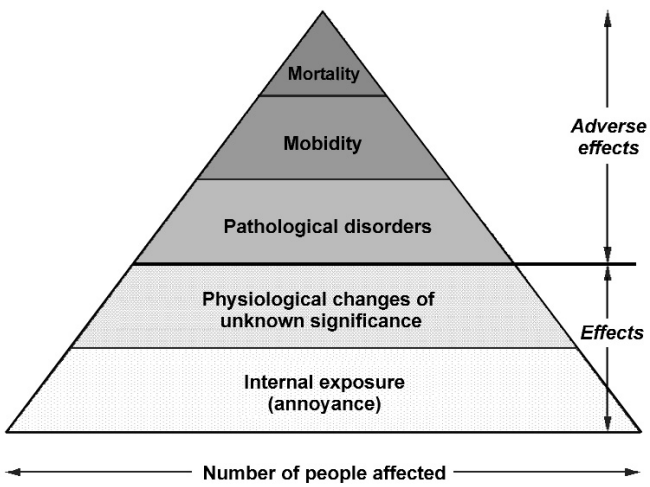


Figure 2. Grading of noise effects on health

noxae, noise as such is not immediately measurable in an organism, only its effects are measurable. This is one reason why toxicological test methods can only partially be applied to noises. The WHO defines adverse health effects as follows (WHO, 1994):
"Change in morphology, physiology, growth, development of lifespan of an organism, which results in impairment of the functional capacity to compensate for additional stress, or increase in susceptibility to the harmful effect of other environmental influences."

Following this definition, the two bottom effect levels in Figure 2, which are "annoyance" and "physiological changes of unclear significance", cannot be considered as 'adverse' effects. The effect levels "pathological changes", "morbidity", and "mortality", however show a higher severity grade and are generally rated as adverse effects.

The fact that an organism responds to a noise stimulation with a natural and physiologically wanted stress reaction, is by itself not a health threat. Acute changes measured under noisy conditions as EEG, finger pulse amplitude, and stress hormone concentrations, all of which represent the orientation reaction (Henry and Stephens, 1977), may be interpreted in terms of NOEL/LOEL (no/lowest observed effect level). It must be noted though, that with the application of improved analytical methods, thresholds of effect may reduce without any physiological background. Because of the lack of health relevance of such effects, it was proposed therefore, in the case of such effect parameters in noise studies, to evaluate them as exceeding standard values. It has to be borne in mind, however, that such excess values are not necessarily coupled with disadvantageous health effects. Normal values are frequently defined on the basis of value distributions of biological variables (percentile), without directly pertaining to any risk of illness. This has to be clarified in each individual case.

If on the other hand clear medical or biological evidence of an effect outcome - as for instance gained from the results of a cohort study - is correlated with either higher or lower values of pathological changes occurring in the organism, then such an outcome is labeled as a "risk factor" per definition. Noise-induced changes associated with such effect outcomes are of actual clinical relevance and may be interpreted in terms of NOAEL/LOAEL (no/lowest observed adverse effect level). Thresholds of effect derived from such outcomes may offer a basis for preventive strategies and standards, with the option of supplementary safety margins to take account for risk groups in a population (WHO, 1994, Arnold et al., 1997, Fischer, 1999, Neus and Boikat, 2000).

Epidemiological noise effects research

Epidemiological research can be used for the derivation of NOAEL/LOAEL levels in the field of noise (Babisch et al., 1992, Samet et al., 1998). It provides the possibility of an integral risk estimation based directly on empirical data gained under genuine conditions of exposure, taking into account any factors which may amplify or attenuate the noise effects. Determination of such effect modifications and identification of the groups at risk is an important assignment of future noise effects research (Thompson, 1996). The dose-response relationships derived from epidemiological data offer a reliable basis for the determination of environmental standards, in this case limiting noise values (Hertz-Picciotto, 1995, Adami and Trichopoulos, 1999, Pearce, 1999, Savitz et al., 1999, Soskolne, 1999).

Laboratory experiments on humans, as far as ethically acceptable, help us understand the effect mechanisms and can reveal individual reaction thresholds as the aftermath of acute noise exposure. However, they only offer provisional information on the long-term effects of chronic exposure and the possible genesis of ill-health. Aspects of adaptation, habituation and physical exhaustion (in the sense of the stress model) remain to a large extent unconsidered. In addition to this, the laboratory scenario influences the results of the study because of a changed acceptance of noise stimulation by the test person. Subjective experience of the noise situation, with all the consequences of

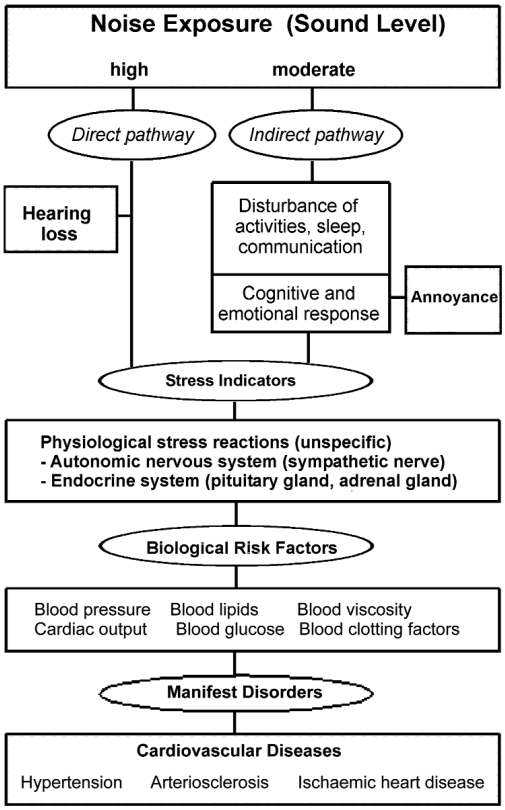


Figure 3. Epidemiological view of noise effects model

annoyance, irritation and the disturbance of activities, plays an essential part in terms of physical reactions to noise.

For the same reasons, animal experiments used to find the answers to toxicological questions offer no effective alternative. This particularly applies to low-dose environmental levels. For the sake of efficiency physiological effects are usually provoked by very high levels of exposure in animal experiments. This may cause general irritations in the organism, played out nonspecifically and independently of the actual exposure factor. Since the noise reactions themselves represent nonspecific stress reactions, the causal effects cannot always be completely separated from each other. In addition to this, the fundamental problem of transferring effect models and thresholds, derived from animal experiments to humans still exists. For this reason, quantitative derivations with reference to humans are only conditionally possible.

Just as uncertain is the extrapolation of noise effects in the environmental range (low dose range) from the results of epidemiological studies with industrial noise (high dose range). Not only the sound intensity influences the noise effects, but also the time structure and the frequency spectrum of the noise, the activity being carried out at the time, the time and place of the noise effects and attributes from the source of the sound itself. For this reason it is hardly surprising if, for example, an average of 85 dB(A) at the workplace induced less bodily reaction than 40 dB(A) during sleep at home.

Cardiovascular disorders have been in the focus of studies on extra-aural noise effects, on the one hand from the results of laboratory experimental research on nonspecific stress reactions to noise, and on the other hand from the considerable significance of these disorders in public health legislation (Doll, 1992, Statistisches Bundesamt, 1998, WHO Regional Office Europe, 1999). Figure 3 reflects the reaction scheme on which epidemiological questions and test hypotheses in noise research is based (Babisch, 2000a, Babisch et al., 2001). Noise directly or indirectly activates the sympathetic and endocrine systems including cortical and sub-cortical structures. Dysregulations in metabolic equilibrium cause chronic changes in values of biological risk

factors, which increase the risk of cardiovascular diseases (Interdisziplinärer Arbeitskreis für Lärmwirkungsfragen beim Umweltbundesamt, 1990). In the causal chain one has to differentiate between stress indicators (for example stress hormones), risk factors (hypertension, blood lipids), and manifest diseases (like myocardial infarction). Their clinical relevance increases in the order mentioned above:

Stress indicators by themselves are not of direct clinical relevance, but serve in assessing effect mechanisms. As short-term reacting parameters they appear first in the reaction-effect chain.

Risk factors are considered as directly relevant to health. Because they act in most cases as continuous variables, even minor non-pathological changes may be included in the evaluation of effect-associated correlations. Nonetheless, for quantitative risk estimation, data from external sources have to be taken into account ("What effect does an x-percent increase in cholesterol have on the risk of myocardial infarction?").

The manifest disease is recognised as an effect outcome of immediate health relevance. It allows a direct risk quantification on the basis of collected data. However, since the available data consist of only discreet and rare incidences, very large sample surveys are needed to present statistical evidence of such effects.

Statistical significance

One of the problems of environmental epidemiology is to statistically ascertain small effects, but small relative risks, on the other hand, may achieve relevance in environmental policy because of the larger number of persons exposed (Neus et al., 1995).

Errors of type-I (α-error, level of significance) are normally used for identifying the statistical uncertainty of effects estimators. Such an error describes the probability with which a test hypothesis (correlation detected) is erroneously accepted instead of a null hypothesis (no correlation). The null hypothesis is conventionally discarded in statistical analyses if the α-error is = 5% (Sachs, 1974). This decision criterion (test of significance), successfully applied in technical fields of quality assurance, is also used in health and environmental research. Errors of type-II (β-error), however, may not be ignored in terms of any possible erroneous decisions in the highly valued asset of health protection (Ortscheid, 1995). This error refers to the probability of zero-hypotheses being erroneously retained. The smaller the á-error chosen for the acceptance of a test hypothesis, the greater is the probability of not determining a true correlation.

Statistical test levels may in principle be variably handled, depending on the question raised (Hartung et al., 1995). In the epidemiological literature, time and again publications are found where α-errors of = 10% are taken as a measure for decisions. This can certainly unleash controversial discussion. However, it has to be borne in mind in the discussion of significance that usually "two-sided" statistical tests are calculated. This means that for distribution parameters in a test sample, deviations in both directions of the expected value are considered and tested. But if there exist justified assumptions of a change in direction of an effect parameter under exposure conditions, e.g. in experimental laboratory tests or for biological plausibility reasons, then the statistical test procedure may also be carried out "one-sided". (Note: The 5% criterion of the one-sided test equals the two-sided test on the 10%-level of significance).

In the specialist literature, the mechanistic application of the significance criterion is rejected, especially in the public health area (Rothman, 1986b, Woolson and Kleinman, 1989, Burton et al., 1998). In environmental epidemiology where large sample tests are needed for statistical effect evidence, non-significant results are often caused by weak test powers. "Non-significance", however, does not mean that no correlation exists (Rothman, 1986b, Morrell et al., 1997). Therefore, the statement of confidence intervals (for instance of the 95% confidence interval) is required for calculating the effect estimator (Rothman, 1986a, Hennekens and Buring, 1987). It is expressly pointed out that the objective of quoting confidence intervals for statistical

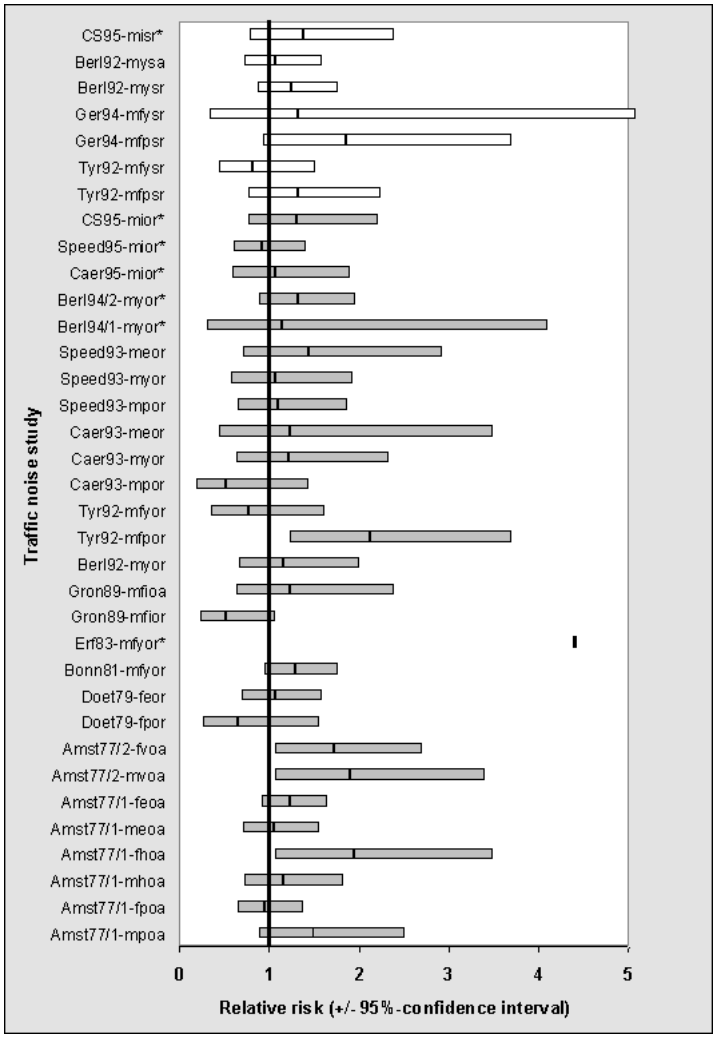
characteristics is not one of reducing the information back down to trivial significance assessment, but to provide a quantitative assessment of the statistical safety of the effect estimator (Rothman et al., 1993). (Note: Inclusion of a relative risk of "1" in the 95% confidence interval is equivalent to an á-error of 5%). An unexpectedly high relative risk, for example, which is significant and comprises a large confidence interval, may yet be of little evidence compared to a low relative risk with a small confidence interval that only just fails statistical significance.

Considerations of the evidence on correlations between exposure factors and effects are generally not undertaken on the basis of individual study findings anyway, but on the basis of the entire available literature on the issue in question (qualitatively by graphical or quantitatively by mathematical methods) (Blettner et al., 1999). The reason for conducting a meta-analysis, for instance, may be that a number of studies methodically appraised as good with common design features, which when taken individually would perhaps not produce significant results. When the studies are pooled an overall effects estimate with a smaller confidence interval will be determined, which may in fact be significant in the conventional sense. On the other hand, any grounds for heterogeneity between studies should also be determined within the framework of a meta-analysis (Blair et al., 1995, Olkin, 1995, Takkouche et al., 1999). Meta-analysis provides a more formal statistical approach to the criterion of consistency than the "rule of thumb" summarizing techniques. However, meta-analysis alone is not sufficient for making causal claims (Weed, 2000).

Traffic noise studies

In comparison to other environmental disciplines, there are relatively few epidemiological noise studies from the sphere of the environment. The data available in terms of industrial noise looks somewhat better although many studies here have had doubts cast on their methods (Babisch, 1998). Misclassification of exposure, no or incomplete control of confounding factors and lack of dose-response relationships are problems in some of the traffic noise studies (Babisch, 2000b). This does not necessarily mean of course that provisional conclusions cannot be drawn from the available data (Rose, 1992, Scheuplein, 1993). Fairly consistent results of investigations can be recognized, particularly where ischaemic heart disease is involved as the final health outcome, independently of considerations of significance. The findings have been summarized in graphs and tables as shown in Figure 4 (Babisch, 2000a). The entries are relative risks with 95% confidence intervals. Relative risks were calculated as risk ratios or odds ratios. The dark-shaded beams in the diagram refer to studies where the noise exposure was determined objectively (noise levels), the light-shaded beams where it was determined subjectively (annoyance). Road traffic and aircraft noise studies are viewed here together. No corresponding results are available for rail traffic studies. (Note: If different subgroups of the population were taken into account (males/females) or different final health outcomes observed, specific studies would appear several times in the illustration. When a series of studies from a particular area under investigation were published in the same year, it is indicated by a serial number behind the year. For example, Amst77/1-mpoa means Amsterdam, 1977, Study 1, males, angina pectoris, objective exposure = sound level, aircraft noise).

Most of the investigations are related to road traffic noise, only a few to aircraft noise. The few flight-noise studies offer no information on dose-response correlation. When comparing extreme groups in traffic noise exposure, a shift of the relative risk estimators to values above 1 can be mainly discerned in strongly exposed groups of people (range approx. 1.1 to 1.5). Using careful and critical evaluation of the findings, the results were so interpreted that the relative risk of ischaemic heart disease to persons living in areas subject to high traffic noise exposure may be slightly higher (Babisch, 2000a). Our own work, enabling assessment of the effects in terms of graded exposure categories (5 dB(A) classes)



Captions

sex:

f female

m male

noise measurement:

o objective (sound level), dark-shaded beam

s subjective (annoyance), light-shaded beam

type of noise:

a aircraft noise

r road traffic noise

ischemic heart disease:

e ECG-ischemic symbols

h heart complaints

i ischemic heart disease

p Angina pectoris

v cardiovascular complaints in general

y heart attack

type of study: prevalence studies; ***** = cohort or case-control studies

Figure 4. Results of epidemiological studies on the association between traffic noise and ischaemic heart disease

indicate an averaged daytime level of 65–70 dB(A) as a possible threshold for noise effects where traffic noise affects health (Babisch and Ising, 1992). An increase in relative risk can be recognized towards higher exposures in the studies. The suspicion was expressed that the risk of a heart attack for persons at home living in roads with average levels above 65–70 dB(A) in the daytime, is approx. 20% higher than for people living in quieter areas (Babisch and Ising, 1992, Ising et al., 1998).

With regard to aircraft noise and the night-flight problems in the vicinity of busy airports, at present no other alternative exists than the approximate transfer of the quantitative risk observations derived from road traffic noise studies to aircraft noise. However, since aircraft noise acts on all sides of a building, i.e. different to road traffic noise, the suspicion exists that the effects induced by aircraft noise could be greater than those induced by road traffic. This may be due to of the lack of evasive possibilities within the home, and the greater annoyance reactions to aircraft noise, which are usually expressed in social surveys (Ortscheid and Wende, 2000). Such effects have yet to be examined in future studies.

With reference to the difference between exposure during the day and at night, no specific conclusions can be deduced from the epidemiological data. Since the difference between average levels of road traffic in the daytime and at night is normally somewhat less than 10 dB(A) (Ullrich, 1998), and the statutory directives on day/night differences are based on immission guideline values of 10 dB(A), an approximation assumes that the suspected threshold value would correspond to 65–70 dB(A) during the day and 55–60 dB(A) at

night. However, this requires empirical or experimental confirmation.

Evidence of the findings

Conclusive evidence of causality, in the strictest sense of the word, can never be provided by epidemiological methods (Rothman, 1986a, Schlesselman, 1987, Christoffel and Teret, 1991). On the other hand, rejection of the results of studies based on such general grounds of theoretical insight would be senseless and block any progress. Causality and reasons are more concerned with gradual concepts, evidence of which increases with the increasing number of facts. In actual practice the followings items are widely used as arguments for the presence of a causal association: magnitude of the effects estimator, existence of a dose-response relationship, biological plausibility, consistency of the results of studies, observation of the effects in different populations and using different methods. (Bradford Hill, 1965, Evans, 1976, Morabia, 1991, Weed and Hursting, 1998).

A number of review articles, focused on evidence provided by the results of epidemiological environmental noise studies, have been published in the past years (Passchier-Vermeer, 1993, Health Council of the Netherlands, 1994, Berglund and Lindvall, 1995, IEH, 1997, Porter et al., 1998, Health Council of the Netherlands, 1999, Babisch, 2000b, Neus and Boikat, 2000, Passchier-Vermeer and Passchier, 2000). The main criteria of assessment used were the guidelines of the "International Agency for Research on Cancer" (IARC, 1987):

Sufficient evidence is given if a positive relationship is observed between exposure to the agent and the health outcome (cancer), in studies in which chance, bias and confounding can be ruled out with reasonable confidence.

Limited evidence is given if a positive association is observed between exposure to the agent and the health outcome (cancer), for which a causal interpretation is considered by a Working Group (experts) to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence.

Inadequate evidence is given if the available studies are insufficient in quality, consistency or statistical power to permit a conclusion regarding the presence or absence of a causal association.

The authors and expert groups of the reviews mentioned above, made the following statements from the evidence:

Health Council of the Netherlands, 1994 (Passchier-Vermeer, 1993, Health Council of the Netherlands, 1994) and Passchier-Vermeer & Passchier, 2000 (Passchier-Vermeer and Passchier, 2000):

"Limited" evidence for the relationship between noise (including occupational noise) and biochemical effects .
"Sufficient" evidence for the relationship between noise (including occupational noise) and hypertension.
"Sufficient" evidence for a relationship between noise and ischaemic heart disease.

Institute for Environment and Health, 1997 (IEH, 1997) and Porter et al., 1998 (Porter et al., 1998):

"Inconclusive" evidence for a causal link between noise exposure and hypertension.
"Sufficient" evidence for a causal association between noise exposure and ischaemic heart disease.

Health Council of the Netherlands, 1999 (Health Council of the Netherlands, 1999)

"Limited" evidence for the relationship between noise (including occupational noise) and biochemical effects.
"Sufficient" evidence for an association between ambient noise and hypertension.
"Sufficient" evidence for an association between ambient noise and ischaemic heart disease (observation threshold: $L_{eq,6\text{-}22\ h}$: 70 dB(A)).

Babisch, 2000 (Babisch, 2000b):

"No" scientific evidence for association between transportation noise and mean blood pressure readings (exception: in children consistently higher readings were found in the exposed groups).
"Little" evidence regarding the association between transportation noise and hypertension.
"Some" evidence regarding the association between transportation noise and ischaemic heart disease. The latter was viewed as being "sufficient" for action.

Neus and Boikat, 2000 (Neus and Boikat, 2000):

"Limited" evidence regarding the association between traffic noise and ischaemic heart disease.

The various evidence ratings of a correlation between traffic noise and cardiovascular disease can be summarized as follows:

Biochemical changes of risk factors: *"Limited" evidence*,
Hypertension: *"Inadequate/limited" evidence*,
Ischemic heart diseases: *"Limited/sufficient" evidence*.

Recently, a systematic review was published which used the meta-analytic approach to assess quantitative estimation of the relationship between community noise and occupational noise on blood pressure, hypertension and ischaemic heart disease (IHD) (Kempen et al., 2002). The authors concluded that the epidemiological evidence on noise exposure, blood pressure and ischaemic heart disease is still limited. With respect to blood pressure and hypertension, results were contradictory and for IHD only a few studies were available. Inconclusive findings may be due to limitations in exposure characterization, adjustment for important confounders, and the occurrence of publication bias.

Conclusions

Health-related noise effect research has, like any other discipline of environmental protection, to be prepared to meet objective and quantitative risk discussions. The concept of "evidence-based medicine" (Heller and Page, 2002) should not be overburdened in this process. This concept addresses the viewpoints of definite and faultless diagnosis as well as the resulting specific and well-targeted treatment of the patient (Hernández-Aguado, 2002). It is going to be difficult to reach a point in noise effects research where the medical profession in established practices is in a position to interpret noise without doubt as responsible for pathological disorders of their patients – with the exception of the aural effects of noise. This, however, is not what ought to be expected of noise effect research. The principle objective, as in other environmental contaminants in the low dose range, is assignment of the statistical probability of health hazards, based on the observation of exposed and non-exposed groups of the population. Extra-aural noise effect research, being a part of general environmental hygiene, is not aimed at treating those afflicted (curative medicine) but rather at regulating exposure from the preventive viewpoint. This means that assessment yardsticks applied to the results of investigations are different to those of clinical research. In place of the deterministic approach to observation in individual medicine, a probabilistic approach is in the foreground of environmental hygiene (group medicine).

On the other hand, noise-induced changes in physiological processes alone, can hardly be used as an argument for establishing environmental standards if no health relevance is presented. Health-related considerations as such should not be set up independently of quantitative risk evaluation. This applies to noise as it does to other environment disciplines. It is to be doubted in this context whether the requirement of a "zero-risk" is at all reasonable or justified. It is the duty of public health policy to formulate and enforce, on the basis of reliable scientific results and competent weighing of public interests, the upper limits of tolerable noise exposure / permitted noise levels.

Above and beyond this, the claims, necessities and wishes directed towards the quality of life by human beings living in a community require endeavor. This has nothing to do with health in the narrowest sense. Most specifically, the attitude towards considerable annoyance plays an important part (BImSchG, 1990). The advantages and disadvantages of sources of noise with regard to all aspects of the quality of life, bearing in mind the relevance of health, have to be weighed against each other if a consensus is to be formed. Individual mobility certainly also creates a wide range of life's qualities. Individual and group interests cannot always be brought into harmony. Quietness is one asset where individuals are prepared to dispense with the consumption of others if circumstances allow.

Noise protection is preventive!

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