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ВСЕМИРНАЯ ОРГАНИЗАЦИЯ ЗДРАВООХРАНЕНИЯ
ЕВРОПЕЙСКОЕ РЕГИОНАЛЬНОЕ БЮРО

EUROPEAN CENTRE FOR ENVIRONMENT AND HEALTH
BONN OFFICE

NIGHT NOISE GUIDELINES (NNGL) FOR EUROPE

Grant Agreement 2003309
Between the European Commission, DG Sanco
and the World Health Organization, Regional Office for Europe

Final implementation report

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Introduction

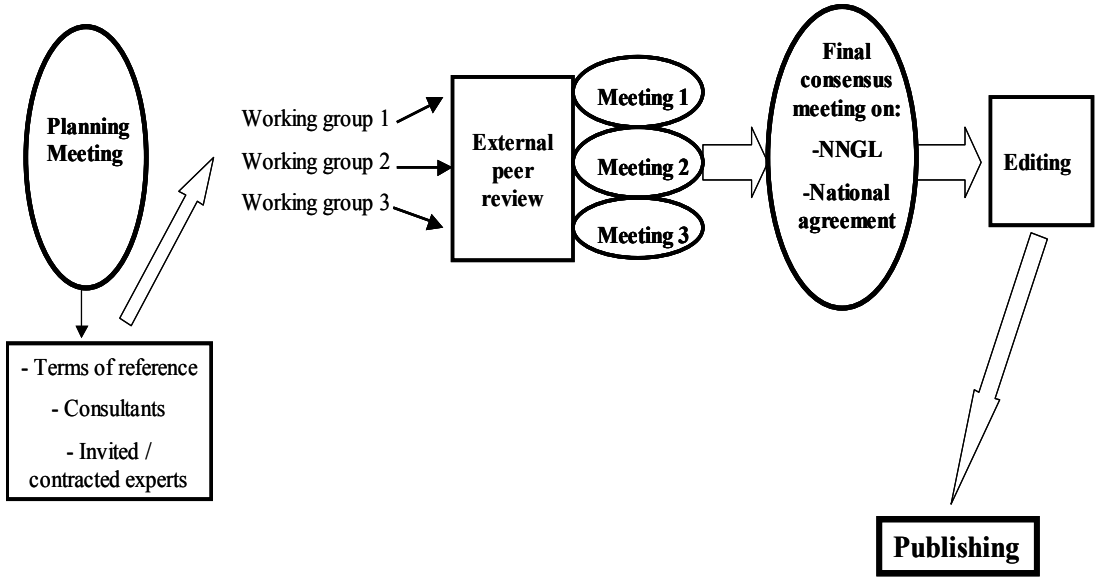
Policies and legislations aiming at night noise control are often based on sleep disturbance in European countries. However, the impacts of noise-induced sleep disturbance on health, either short-term or long-term, have not been investigated comprehensively to support policy-makers. From June 2003 until December 2006, WHO Regional Office for Europe European Centre for Environment and Health (Bonn office) implemented the Night Noise Guideline (NNGL) project co-sponsored by the European Commission.

The goal of the NNGL project was to provide expertise and scientific advice to the European Commission and its Member States in developing future legislations in the area of night noise exposure control and surveillance. The key objectives of the project was to reach a consensus of experts and stakeholders on the following subjects: (a) guideline values for night noise to protect the public from adverse health effects, (b) an agreement on the night penalty factor to be allocated to night time noise in the calculation of L_{den}.

The methodology of developing night noise guidelines was based on the WHO publication EUR/00/5020369 "Evaluation and use of epidemiological evidence for environmental risk assessment" that can be accessible at http://www.euro.who.int/document/e68940.pdf.

Project activities conducted

The work was performed according to the following process.



WHO coordinated overall project activities, providing terms of reference, organising meetings, ensuring timely production of working documents. The partner institutions agreed to participate in one or more working groups and devote a minimum of 6 working days per year for reviewing documents and attending meetings. Experts were contracted with WHO for making scientific reviews of existing literature and for contributing to the contents of the project products.

The project activities were performed according to the following sequence.

1. Identification of the major health end points relevant to sleep disturbance caused by environmental noise (June 2004 – May 2005)

2. Comprehensive identification and systematic review of the existing body of evidence, and expert consensus on guideline values (December 2004 – August 2006)

3. Final consultation for harmonizing the proposed guideline values (October 2005 - December 2006)

4. A final meeting gathering the best available experts and adopting the final guideline values for night noise exposure (December 2006)

5. Reporting and dissemination (December 2006 - March 2007)

The key milestones of project activities were technical meetings of topic-specific experts in the field of acoustics, exposure assessment, sleep pathology, accident epidemiology, immunology, mental health, and health impact assessment. The experts produced the background papers based on their review of scientific evidence on the impacts of noise on sleep and health. These background documents were synthesized into one document proposing health-based guidelines. This document was then distributed among the experts and stakeholders for final peer-review. The final products of the project were discussed at the final meeting of experts and stakeholders where the consensus on the guideline values was reached.

Although some administrative problems arose due to the replacement of two partners at the beginning of the project in 2003, the project was implemented according to the plan outlined in the project proposal. All meeting reports are attached to this final report as Annexes. These meeting reports describe all the activities conducted to achieve the objectives of the project.

The first technical meeting was held in Bonn, at the premises of the WHO/EURO - European Centre for Environment and Health, June 2004. It gathered the project partners, experts and national government officers to define the work plan and discuss organizational issues. This meeting was crucial to define the timetable, allocate responsibilities, organise team coordination, the logistics and finance. A first draft of the table of contents of the guidelines document was discussed. The WHO prepared the background material of the meeting and each partner presented his/her field of knowledge and future role on the project. WHO suggested the partners to cover specialized topics, but they also could decide themselves which issue to contribute to.

The second technical meeting took place in Geneva at the WHO/HQ premises, December 2004. It concentrated on the methodological issues of exposure assessment, metrics, health effects, and formulation of guidelines. The partners presented the first draft papers for the different identified topics and detailed discussion took place for each one of them. The discussions concentrated mainly on central issues like exposure assessment and guideline derivation. The WHO organized and prepared the background material and some partners prepared papers. The discussion was around the papers and on the way forward, especially to address lacks on evidence and what (and how) to consider as health outcome.

The third technical meeting in Lisbon at the premises of the DGS (Direcção Geral da Saúde – Portuguese Directorate General for Health), April 2005, reviewed the final version of the background papers and discussed how to finalize and build consensus until the end of the project. These three meetings contributed to the derivation of guideline values for night noise

both for short and long term exposure, and provided the main contents of the Night Noise Guidelines document.

The fourth technical meeting was convened in Den Haag, September 2005, focusing on the issues on indicators. Through the workshop of acoustics experts, a consensus was made on the use of L_{night} as the single indicator for guideline values as it effectively combines the information on the number of events and the maximum sound levels per event. This meeting contributed to the preparation of the substantiation for an agreement on the night penalty factor to be allocated to night time noise in the calculation of L_{den}.

The final stage of project implementation was delayed due to the departure of initial project coordinator and the technical offier from WHO in 2006. The project was continued by WHO temporary advisor, Martin van den Berg, and WHO scientist on noise burden of disease, Rokho Kim, since August 2006.

At the final meeting for consensus building in Bonn, December 2006, the experts and stakeholders reviewed the final draft of Night Noise Guidelines document. Based on the comments and agreements at the meeting, the final technical report of NNGL project was revised again for finalization.

As of March 2007, the two deliverables of the project are posted on the World Wide Web of the WHO at www.euro.who.int/noise, as the recommendation of the working group for the Nigh Noise Guidelines for Europe. As per agreement between the UN system and the commission, title and industrial property rights in the result of the project and the reports and other documents relating to it vest in WHO. Notwithstanding the above, WHO will grant the commission the right to use freely and as it sees fit all documents deriving from the project, whatever their form.

Manpower for the execution of the activities

A complete list of all the persons who have participated in the execution of the project is presented below together with the man-days of work, the professional level or category and the corresponding unit and total cost.

WHO Personnel

Person	Task
Xavier Bonnefoy, WHO Regional Advisor	Project coordinator,
Celia Rodrigues, WHO Technical officer	Assistant to coordinator
Rokho Kim, WHO Scientist	Coordination since August 2006
Nuria Aznar, WHO Administrative Assistant	Secretary
Martin van den Berg WHO Temporary advisor and Dutch Ministry of Housing	Compilation of background documents and editing of final guidelines document

Partner organisations

Institutions	Person	Tasks (Specific topics)
Università La Sapienza di Roma	Olivero Bruni	Sleep and children
Queen Mary University	Stephen Stansfeld	Mental effects of sleep disturbance
TNO	H.M.E Miedema	Short term effects
CNRS-CEPA	A Muzet	Short term effects
Medical Faculty, Charles University Prague	Sona Nevsimalova	Health effects of sleep disturbance
Instituto Superior Tecnico, Departamento de Engenharia civil, Lisbonne	Joao de Quinhones Levy	General approach of environment related health
Laboratory of applied psychology, Center for built environment, Gävle	Staffan Hygge	Initial review of cognitive impairment Re-review of cognitive impairment
Federal Environmental Agency, Division of Environment and Health, Berlin	Wolfgang Babisch	Cardio-vascular diseases
INRETS LTE	Jacques Beaumont	Indicators of night time noise
Landesgesundheitsamt Baden-Würtemberg, Stuttgart	Snezana Jovanovic	Accidents in children
ARPAT	Gaetano Licitra	Acoustic aspects
RIVM Bilthoven	Danny Houthuijs	Epidemiolo-gical aspects
Institute of Social Medecine, University of Innsbruck	Peter Lercher	Epidemiolo-gical aspects
Katedra i klinika Psychiatryczna, Warsaw	Michal Skalski	Mental effects of sleep disturbance
Institute of Public Health of the Republic of Slovenia, Ljubljana	Leja Dolenc Groselj	Health consequence of sleep disturbance

Partners involved

The project was implemented in collaboration with 17 partners from 12 European Countries (Annex 1). In addition to the formal project partners, WHO received advice and support from a number of international experts, industry associations, non-governmental organizations and other stakeholders regarding specific aspects of the night time noise issues (Annex 2).

- Institute of Hygiene and Social Medicine, University of Innsbruck, Austria
- Univerzita Karlova v Pragze - Medical Faculty Charles University, Czech Republic
- National Institute of Public Health, Denmark
- INRETS / LTE - Laboratoire Transports et Environnement, France
- Centre National de Recherche Scientifique Centre d'études de Psysiologie Appliquée, France
- Umweltbundesamt - Federal Environmental Agency, Germany
- Landesgesundheitsamt Baden-Württemberg, Germany
- ARPAT Dipartimento Provinciale di Pisa, Italy
- University of Rome "La Sapienza" - Center for Pediatric Sleep Disorders, Italy
- EC Joint Research Centre, Institute for Health & Consumer Protection, Italy
- TNO - Netherlands Organisation for Applied Scientific Research, The Netherlands
- RIVM - National Institute of Public Health, The Netherlands
- Katedra i Klinika Psychiatryczna, AM Warszawie, University of Warsaw, Poland
- IST / CESUR - Centro de Estudos Urbanos e Regionais, Portugal
- Institute of Public Health, The Republic of Slovenia
- University of Gävle, Centre for Built Environment, Sweden
- Queen Mary and Westfield College University of London, UK

The themes and work were assigned to the partners on mutual agreement at the first meeting as below.

WORK ASSIGNMENT FOR NNGL DEVELOPMENT

Themes	Responsible expert*
i. Setting the scene Sources, metrics, sensitive areas, number of people exposed, trends, number of events, variations during the night, overview of legislation….	Lercher, Licitra, Beaumont, Levy
ii. Uncertainty in exposure	Kephalopoulos
iii. Instantaneous effects Major sleep disturbances, moderate sleep disturbances, other	Muzet, Miedema

iv. Cardiovascular effects, Hypertension	Babisch
v. Immune system Hormones excretion, decreased glucose assimilation, …	Maschke (Depending on the expert's agreement)
vi. Other health outcomes , i. Physical (fatigue, drowsiness, sleepiness, …) ii. Cognitive impairment (deterioration of performance, attention and motivation and diminishment of mental concentration and intellectual capacity and, increases the chances of accidents at work and during driving,…) iii. Accidents (this point needs special attention although covered in a) and b) iv. Mental health v. Sleep pathologies	Gale Hygge Jovanovic Stansfeld, Skalski Nevsimalova
vii. Animal studies	Ising (Depending on the expert's agreement)
viii. Scoring the evidence	Boegli
ix. Guidelines derivation, methodology aspects	Van den Berg
x. Risk groups	Bistrup, Kahn Passchier Veermer
xi. Meta-analysis	Houthuijs
xii. Neighbourhood noise (night)	Grimwood

*See Annex 2 to find the partner insitutes of responsible experts.

Countries involved

The following countries and their institutes were involved in the project. All institutes of these countries were sent the draft document of night noise guidelines for their review and comments. The final document are sent to all of these institutes for further dissemination.

AUSTRIA
Institute of Hygiene and Social Medicine, University of Innsbruck

CZECH REPUBLIC
Univerzita Karlova v Pragze - Medical Faculty Charles University

DENMARK
National Institute of Public Health

FRANCE
INRETS / LTE - Laboratoire Transports et Environnement
Centre National de Recherche Scientifique Centre d'études de Psysiologie Appliquée

GERMANY
Umweltbundesamt - Federal Environmental Agency
Landesgesundheitsamt Baden-Württemberg

ITALY
ARPAT Dipartimento Provinciale di Pisa
University of Rome "La Sapienza" - Center for Pediatric Sleep Disorders

NETHERLANDS
TNO - Netherlands Organisation for Applied Scientific Research
RIVM - National Institute of Public Health

POLAND
Katedra i Klinika Psychiatryczna, AM Warszawie, University of Warsaw

PORTUGAL
IST / CESUR - Centro de Estudos Urbanos e Regionais

SLOVENIA
Institute of public health of the republic of Slovenia

SWEDEN
University of Gävle, Centre for Built Environment

UNITED KINGDOM
Queen Mary and Westfield College University of London

In addition to the formal project partners, WHO has received advice and support from a number of national experts regarding specific aspects of the night noise issues. The affiliations of these additional expert advisers include:

CANADA - Health Canada
GERMANY - Forschungs- und Beratungsbüro
SWITERLAND - Bundesamt für Umwelt, Wald und Landschaft
SWITZERLAND - Universität St. Gallen, Institut für Wirtschaft und Ökologie
THE NETHERLANDS - Ministry of Housing, Spatial Planning and Environment
UNITED KINGDOM - Casella Stanger Environmental Consultants

Achievement of the objectives

The project achieved all of the objectives as of December 2006.

The first deliverable of the project is a full report proposing health based guideline values for night noise exposure supported with the best available scientific evidence and possible exposure response curves. The executive summary is enclosed in this report as the first part of three project results. The full document is enclosed as the second part.

The second deliverable is a report describing the expert consensus on possible ways to amend the penalty added to night noise levels in the calculations of L_{den}. The technical report on this topic is enclosed as the third part of projects results.

It turned out to be technically impossible to propose the attributable fraction of risk for any health end points due to lack of exposure data. Although the evidence on the exposure-response relationship is often available in the scientific literature, the population´s exposure to night noise in terms of L_{night} are still lacking in many countries. This is because L_{night} is a new indicators atoped by Environmental Noise Directive. Once after the noise directive is enforced for the reporting of noise map from June 2007, night noise exposure data will be available in most of EU member states. WHO is continuing another project to develop methodlogy of estimating burden of disease from environmental noise, Noise EBD project, of which the report will be available at the end of 2007.

In conclusion, the NNGL project produced the expected results listed in the proposal. Guideline values for night noise are based on L_{night} from all sources (either single or combined), integrating air traffic, road traffic, rail traffic and mixed sources into one summary scale. The vulnerability of children to night noise were explicitly addressed in the rationale of guidelines along with the chronically ill and the elderly. A dose-response curve for the levels of exposure above the guideline values are also provided. The extensive list of the references used for deriving the guidelines were provided. This project supported the development of the noise indicators for the EU public health monitoring program in the framework of the ECOEHIS project.

The projects results provide the rationale and the scientific background for drafting a proposal for modifying, if need be, the correction factor proposed for night noise exposure in paragraph 1 of the annex 1 of the directive 2002/49/EC of the 25/06/2002 related to the assessment and management of Environmental noise. The current consensus is to keep the 10dB penalty for night noise until other compelling arguements and evidence emerge in the future.

All of five meeting reports of the NNGL project were prepared and published on WHO website.

Night time air traffic as well as railway freight traffic is likely to increase dramatically between now and 2030 (OECD 2002, Policy Instruments for Achieving EST: Report on Phase 3 of the EST Project). Harmonized legislation based on solid scientific evidence will certainly contribute to improving health of all Europeans, especially, of the citizens of the countries where the public awareness and the legislations are currently rudimentary and scarce around the issue of night noise. The results of the project will provide the commission with elements on which it will be possible to base regulations aiming at enforcing implementation of actions as a consequence of the monitoring of night noise levels performed according to the directive 2002/49/EC.

Project result 1: Summary of Night Noise Guidelines for Europe

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EUROPEAN CENTRE FOR ENVIRONMENT AND HEALTH
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NIGHT NOISE GUIDELINES FOR EUROPE

SUMMARY

Grant Agreement 2003309
Between the European Commission, DG Sanco
and the World Health Organization, Regional Office for Europe

1 Introduction

The aim of this document is to present the conclusions of the World Health Organization (WHO) working group responsible for preparing guidelines for exposure to noise during sleep. This document can be seen as an extension of the *WHO Guidelines for Community Noise* (2000). The need for "health-based" guidelines originated in part from the European Union Directive 2002/49/EC relating to the assessment and management of environmental noise (commonly known as the Environmental Noise Directive and abbreviated as END) which will compel European Union Member States to produce noise maps and data about night exposure from mid-2007. The work was made possible by a grant from the European Commission and contributions from the Swiss and German governments.

Although a number of countries do have legislation directed at controlling night noise exposure, there is little information on actual exposure and its subsequent effects on the population. Estimates made in some countries of the number of people highly disturbed by noise during sleep (see Figure 1 for the Netherlands as an example) indicate that a substantial part of the population could be exposed to levels that might risk their health and well-being

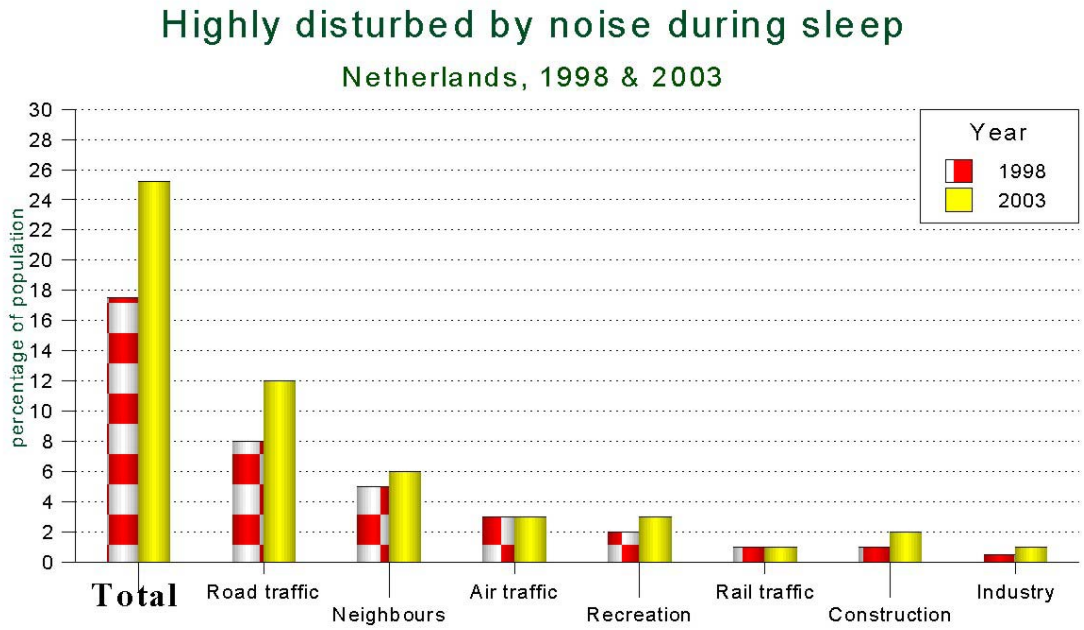

Figure 1. Percentage of population highly disturbed by noise during sleep in the Netherlands. Survey results for 1998 and 2003.

As direct evidence concerning the effects of night noise on health is rarely available, these guidelines also use indirect evidence: the effects of noise on sleep and the relations between sleep and health. The advantage of this approach is that a lot of medical evidence is available on the relation between sleep and health, and detailed information also exists on sleep disturbance by noise.

2 Process of developing guidelines

In 2003, the WHO Regional Office for Europe set up a working group of experts to provide scientific advice to the European Commission and to its Member States for the development of future legislation and policy action in the area of control and surveillance of night noise exposure. The review of available scientific evidence on the health effects of night noise was carried out by an interdisciplinary team who set out to derive health-based guideline values. The contributions from the experts were reviewed by the team and integrated into draft reports following discussion at four technical meetings of the working group. In 2006, all the draft reports were compiled into a draft document on guidelines for exposure to noise at night, which was reviewed and commented on by a number of stakeholders and experts.

At the final conference in Bonn, Germany, on 14 December 2006, representatives from the working group and stakeholders from industry, government and nongovernmental organizations reviewed the contents of the draft document chapter by chapter, discussed several fundamental issues and reached general agreement on the guideline values and related texts to be presented as conclusions of the final document of the *WHO Night Noise Guidelines for Europe.*

3 Noise indicators

From the scientific point of view the best criterion for choosing a noise indicator is its ability to predict an effect. Therefore, for different health end points, different indicators could be chosen. Long-term effects such as cardiovascular disorders are more correlated with indicators summarizing the acoustic situation over a long time period, such as yearly average of night noise level outside at the façade ($L_{night,outside}$[1]), while instantaneous effects such as sleep disturbance are better with the maximum level per event (L_{Amax}), such as passage of a lorry, aeroplane or train.

From a practical point of view, indicators should be easy to explain to the public so that they can be understood intuitively. Indicators should be consistent with existing practices in the legislation to enable quick and easy application and enforcement.

$L_{night,outside}$, adopted by the END, is an indicator of choice for both scientific and practical use. Among currently used indicators for regulatory purposes, L_{Aeq} (A-weighted equivalent sound pressure level) and L_{Amax} are useful to predict short-term or instantaneous health effects.

4 Sleep time

Time use studies, such as that undertaken by the Centre for Time Use Research, 2006 (www.timeuse.org/access/), show that the average time adult people are in bed is around 7.5 hours, so the real average sleeping time is somewhat shorter. Due to personal factors like age and genetic make-up there is considerable variation in sleeping time and in beginning and end times. For these reasons, a fixed interval of 8 hours is a minimal choice for night protection.

Though results vary from one country to another, data show (see Figure 2 as an example) that an 8 hour interval protects around 50% of the population and that it would take a period of 10 hours to protect 80%. On Sundays, sleeping time is consistently 1 hour longer, probably due to people recovering from sleep debt incurred during the week. It should also be borne in mind that (young) children have longer sleeping times.

[1] L_{night} is defined in the END as the outside level. In order to avoid any doubt, the suffix "outside" is added in this document.

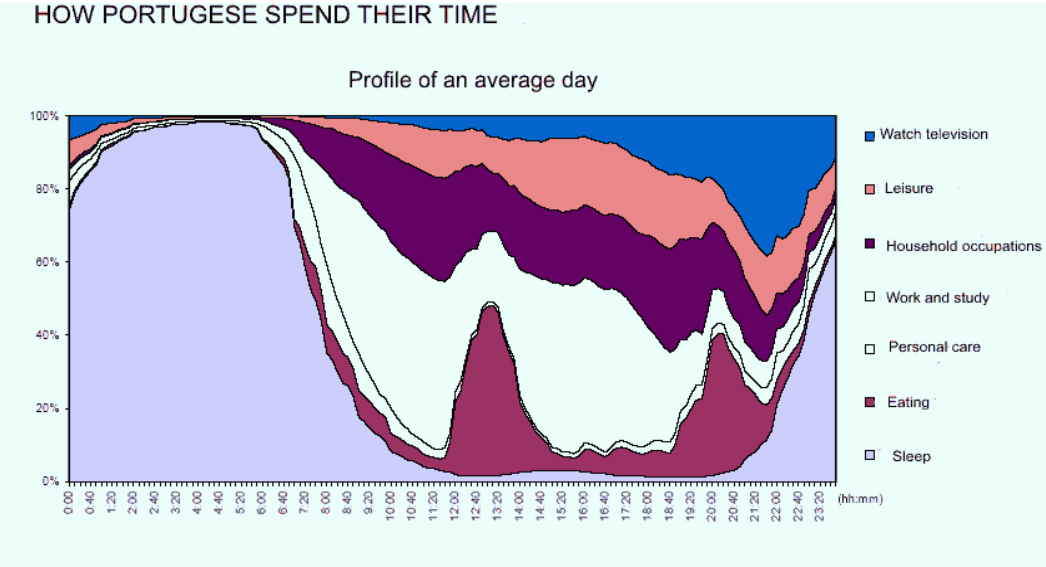

Figure 2. Percentage of time that the Portuguese population spend asleep or in different activities.

5 Noise, sleep and health

There is plenty of evidence that sleep is a biological necessity, and disturbed sleep is associated with a number of health problems. Studies of sleep disturbance in children and in shift workers clearly show the adverse effects.

Noise disturbs sleep by a number of direct and indirect pathways. Even at very low levels physiological reactions (increase in heart rate, body movements and arousals) can be reliably measured. Also, it was shown that awakening reactions are relatively rare, occurring at a much higher level than the physiological reactions.

> ***Sufficient evidence:*** *A causal relation has been established between exposure to night noise and a health effect. In studies where coincidence, bias and distortion could reasonably be excluded, the relation could be observed. The biological plausibility of the noise leading to the health effect is also well established.*
> ***Limited evidence:*** *A relation between the noise and the health effect has not been observed directly, but there is available evidence of good quality supporting the causal association. Indirect evidence is often abundant, linking noise exposure to an intermediate effect of physiological changes which lead to the adverse health effects.*

The working group agreed that there is sufficient evidence that night noise is related to self-reported sleep disturbance, use of pharmaceuticals, self-reported health problems and insomnia-like symptoms. These effects can lead to a considerable burden of disease in the population. For other effects (hypertension, myocardial infarctions, depression and others), limited evidence was

found: although the studies were few or not conclusive, a biologically plausible pathway could be constructed from the evidence.

An example of a health effect with limited evidence is myocardial infarctions. Although evidence for increased risk of myocardial infarctions related to L_{day} is sufficient according to an updated meta-analysis, the evidence in relation to $L_{night,outside}$ was considered limited. This is because $L_{night,outside}$ is a relatively new exposure indicator, and few field studies have focused on night noise when considering cardiovascular outcomes. Nevertheless, there is evidence from animal and human studies supporting a hypothesis that night noise exposure might be more strongly associated with cardiovascular effects than daytime exposure, highlighting the need for future epidemiological studies on this topic.

The review of available evidence leads to the following conclusions.

- Sleep is a biological necessity, and disturbed sleep is associated with a number of adverse impacts on health.
- There is sufficient evidence for biological effects of noise during sleep: increase in heart rate, arousals, sleep stage changes, hormone level changes and awakening.
- There is sufficient evidence that night noise exposure causes self-reported sleep disturbance, increase in medicine use, increase in body movements and (environmental) insomnia.
- While noise-induced sleep disturbance is viewed as a health problem in itself (environmental insomnia) it also leads to further consequences for health and well-being.
- There is limited evidence that disturbed sleep causes fatigue, accidents and reduced performance.
- There is limited evidence that noise at night causes clinical conditions such as cardiovascular illness, depression and other mental illness. It should be stressed that a plausible biological model is available with sufficient evidence for the elements of the causal chain.

6 Vulnerable groups

Children have a higher awakening threshold than adults and therefore are often seen to be less sensitive to night noise. For other effects, however, children seem to be equally or more reactive than adults. As children also spend more time in bed they are exposed more and to higher noise levels. For these reasons children are considered a risk group.

Since with age the sleep structure becomes more fragmented, elderly people are more vulnerable to disturbance. This also happens in pregnant women and people with ill health, so they too are a group at risk.

Finally, shift workers are at risk because their sleep structure is under stress due to the adaptations of their circadian rhythm.

7 Thresholds for observed effects

The (no) observed adverse effect level (NOAEL) is a concept from toxicology, and is defined as the greatest concentration which causes no detectable adverse alteration of morphology, functional capacity, growth, development or lifespan of the target organism. For the topic of

night noise (where the adversity of effects is not always clear) this concept is less useful. Instead, the observed effect thresholds are provided: the level above which an effect starts to occur or shows itself to be dependent on the exposure level. It can also be a serious pathological effect, such as myocardial infarctions, or a changed physiological effect, such as increased body movement.

Threshold levels of noise exposure are important milestones in the process of evaluating the health consequences of environmental exposure. The threshold levels also delimit the study area, which may lead to a better insight into overall consequences. In Table 1, all effects are summarized for which sufficient or *limited evidence* exists. For the effects with *sufficient evidence* the threshold levels are usually well known, and for some the dose-effect relations over a range of exposures could also be established.

Table 1. Summary of effects and threshold levels for effects where **sufficient** evidence is available[1]

	Effect	Indicator	Threshold, dB
Biological effects	Change in cardiovascular activity	*	*
	EEG awakening	$L_{Amax,inside}$	35
	Motility, onset of motility	$L_{Amax,inside}$	32
	Changes in duration of various stages of sleep, in sleep structure and fragmentation of sleep	$L_{Amax,inside}$	35
Sleep quality	Waking up in the night and/or too early in the morning	$L_{Amax,inside}$	42
	Prolongation of the sleep inception period, difficulty getting to sleep	*	*
	Sleep fragmentation, reduced sleeping time	*	*
	Increased average motility when sleeping	$L_{night,outside}$	42
Well-being	Self-reported sleep disturbance	$L_{night,outside}$	42
	Use of somnifacient drugs and sedatives	$L_{night,outside}$	40
Medical conditions	Environmental insomnia[1]	$L_{night,outside}$	42

* Although the effect has been shown to occur or a plausible biological pathway could be constructed, indicators or threshold levels could not be determined.

[1] Please note that "environmental insomnia" is the result of diagnosis by a medical professional whilst "self-reported sleep disturbance" is essentially the same, but reported in the context of a social survey. Number of questions and exact wording may differ.

Table 2. Summary of effects and threshold levels for effects where limited evidence is available[1]

	Effect	Indicator	Estimated threshold, dB
Biological effects	Changes in (stress) hormone levels	*	*
Well-being	Drowsiness/tiredness during the day and evening	*	*
	Increased daytime irritability	*	*
	Impaired social contacts	*	*
	Complaints	$L_{night,outside}$	35
	Impaired cognitive performance	*	*
Medical conditions	Insomnia	*	*
	Hypertension	$L_{night,outside}$ (probably depending on daytime exposure as well)	50
	Obesity	*	*
	Depression (in women)	*	*
	Myocardial infarction	$L_{night,outside}$ (probably depending on daytime exposure as well)	50
	Reduction in life expectancy (premature mortality)	*	*
	Psychic disorders	$L_{night,outside}$	60
	(Occupational) accidents	*	*

* Although the effect has been shown to occur or a plausible biological pathway could be constructed, indicators or threshold levels could not be determined.

[1] Please note that as the evidence for the effects in this table is limited, the threshold levels also have a limited weight. In general they are based on expert judgement of the evidence.

8 Relations with $L_{night,outside}$

Over the next few years, the END will require that night exposures are reported in $L_{night,outside}$. It is, therefore, interesting to look into the relation between $L_{night,outside}$ and adverse health effects. The relation between the effects listed in Tables 1 and 2 and $L_{night,outside}$ is, however, not straightforward. Short-term effects are mainly related to maximum levels per event inside the bedroom: $L_{Amax,inside}$. In order to express the (expected) effects in relation to the single European

Union indicator, some calculation needs to be done. The calculation for the total number of effects from reaction data on events (arousals, body movements and awakenings) needs a number of assumptions. The first that needs to be made is independence: although there is evidence that the order of events of different loudness strongly influences the reactions, the calculation is nearly impossible to carry out if this is taken into consideration. Secondly, the reactions per event are known in relation to levels at the ear of the sleeper, so an assumption for an average insulation value must be made. In the report a value of 21 dB has been selected. This value is, however, subject to national and cultural differences. One thing that stands out is the desire of a large part of the population to sleep with windows (slightly) open. The relatively low value of 21 dB takes this into account already. If noise levels increase, people do indeed close their windows, but obviously reluctantly, as complaints about bad air then increase and sleep disturbance remains high. This was already pointed out in the *WHO Guidelines for Community Noise* (2000).

From source to source the number of separate events varies considerably. Road traffic noise is characterized by relatively low levels per event and high numbers, while air and rail traffic are characterized by high levels per event and low numbers. For two typical situations estimates have been made and presented in graphical form. The first is an average urban road (600 motor vehicles per night, which corresponds roughly to a 24 hour use of 8000 motor vehicles, or 3 million per year, the lower boundary the END sets) and the second case is for an average situation of air traffic exposure (8 flights per night, nearly 3000 per year).

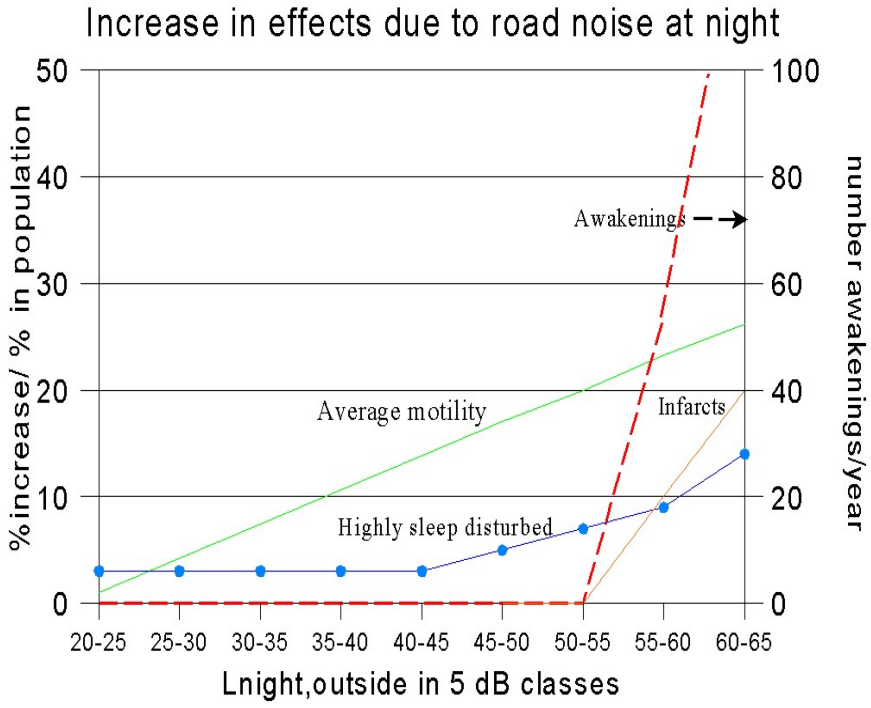

Figure 3. Effects of road traffic noise at night.

Figure 3 shows how effects increase with an increase of $L_{night,outside}$ values for the typical road traffic situation (urban road). A large number of events lead to high levels of awakening once the

threshold of $L_{max,inside}$ is exceeded. To illustrate this in practical terms: values over 60 dB $L_{night,outside}$ occur at less then 5 metres from the centre of the road.

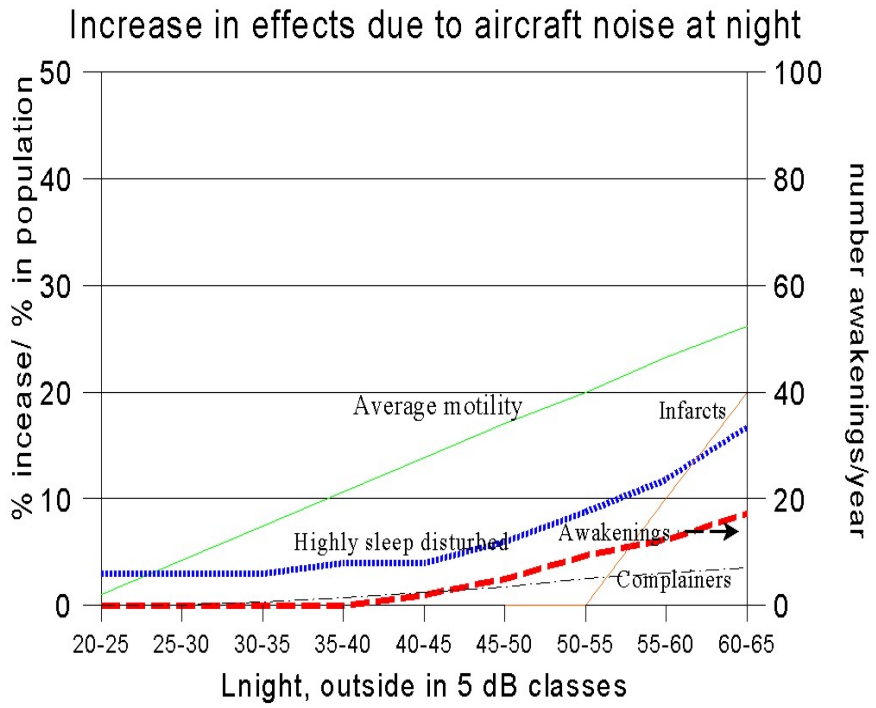

Figure 4. Effects of aircraft noise at night

In Figure 4 the same graph is presented for the typical airport situation. Due to a lower number of events there are fewer awakenings than in the road traffic case (Figure 3), but the same or more health effects. In these examples the worst case figures can be factors higher: the maximum number of awakenings for an $L_{night,outside}$ of 60–65 dB is around 300 per year.

A recent study suggests that high background levels of noise (from motorways) with a low number of separate events can cause high levels of average motility.

Therefore, by using the $L_{night,outside}$ as a single indicator, a relation between effects and indicator can be established. For some effects, however, the relation can be source dependent. Although L_{night} gives a good relation for most effects, there is a difference between sources for some. Train noise gives fewer awakenings, for instance. Once source is accounted for, the relations are reasonably accurate.

9 Recommendations for health protection

Sleep is an essential part of human functioning and is recognized as a fundamental right under the European Convention on Human Rights.[2] Based on the evidence of the health effects of night

[2] Article 8.1: "Everyone has the right to respect for his private and family life, his home and his correspondence." Although, in the case against the United Kingdom, the European Court of Human Rights ruled that the United

noise, an overall summary of the relation between night noise levels and health effects, and stepwise guideline values are presented as shown in Table 3 and 4, respectively.

Table 3. Summary of the relation between night noise and health effects in the population

$L_{night,outside}$ **up to 30 dB**	Although individual sensitivities and circumstances differ, it appears that up to this level no substantial biological effects are observed.
$L_{night,outside}$ of **30 to 40 dB**	A number of effects are observed to increase: body movements, awakening, self-reported sleep disturbance, arousals. With the intensity of the effect depending on the nature of the source and on the number of events, even in the worst cases the effects seem modest. It cannot be ruled out that vulnerable groups (for example children, the chronically ill and the elderly) are affected to some degree.
$L_{night,outside}$ of **40 to 55 dB**	There is a sharp increase in adverse health effects, and many of the exposed population are now affected and have to adapt their lives to cope with the noise. Vulnerable groups are now severely affected.
$L_{night,outside}$ of **above 55 dB**	The situation is considered increasingly dangerous for public health. Adverse health effects occur frequently, a high percentage of the population is highly annoyed and there is some limited evidence that the cardiovascular system is coming under stress.

Especially in the range $L_{night,outside}$ from 30 to 55 dB, a closer look may be needed into the precise impact as this may depend much on the exact circumstances. Above 55 dB the cardiovascular effects become the dominant effect, which is thought to be less dependent on the nature of the noise.

From Table 1, it is clear that a number of instantaneous effects are related to threshold levels expressed in L_{Amax}. The health relevance of these effects cannot be easily established. It can be safely assumed, however, that an increase in the number of such effects over the base line may constitute an subclinical adverse health effect.

For the primary prevention of subclinical adverse health effects in the population related to night noise, it is recommended that the population should not be exposed to night noise levels greater than 30 dB of $L_{night,outside}$ during the night when most people are in bed. Therefore, $L_{night,outside}$ 30 dB is the ultimate target of Night Noise Guideline (NNGL) to protect the public, including the most vulnerabl groups such as children, the chronically ill and the elderly, from the adverse health effects of night noise.

Kingdom government was not guilty of the charges, the right on undisturbed sleep was recognized (the Court's consideration 96).

Two interim targets are proposed for the countries where the NNGL cannot be achieved in a short period for various reasons, and where policy-makers choose to adopt a stepwise approach at the local or national levels (Table 4).

Table 4. Proposed night noise guidelines and interim targets

Interim target I (IT-I)	$L_{night,outside}$ = 55 dB
Interim target II (IT-II)	$L_{night,outside}$ = 40 dB
Night noise guideline (NNGL)	$L_{night,outside}$ = 30 dB

All countries are encouraged to reduce gradually the size of the population exposed to levels over the interim targets, 40 dB of $L_{night,outside}$ (IT-I) and 55 dB of $L_{night,outside}$ (IT-II), as effectively as possible.

It is highly recommended to carry out risk assessment and management activities at national level targeting the exposed population, and aiming at reducing night noise to the level below IT-I and IT-II. IT-II can be used for health impact assessment of new projects (e.g., highways, railways, airports or new residential areas) even before the achievement of IT-I, as well as for the risk assessment of the whole population. In the long run the NNGL would be best achieved by control measures on the sources along with other comprehensive approaches.

10 Relation to the 2000 *WHO Guidelines for Community Noise*

The *WHO Guidelines for Community Noise,* published in 2000, also address night noise. As they are based on studies carried out up to 1995 (and a few meta-analyses some years after), important new studies have become available since then, together with new insights into normal and disturbed sleep.

The currently recommended guideline values of $L_{night,outside}$ = 30 dB, 40dB, 55 dB are not directly comparable with the 2000 guideline value of $L_{Amax,inside}$ = 45 dB(A) because the sound level units are different. However, it is clear that new information since 2000 has made more precise assessment of the risk from night noise. The thresholds for a number of effects are now known, and this is much lower than an $L_{Amax,inside}$ of 45 dB.

One important recommendation still stands: there are good reasons for people to sleep with their windows open, and to prevent sleep disturbances one should consider the equivalent sound pressure level and the number of sound events. The present guidelines allow relevant authorities and stakeholders to do this. Viewed in this way, the present guidelines may be considered as an extension to, as well as an update of, the 2000 WHO *Guidelines for Community Noise*. That also means that the recommendations contained in the sections on noise management and control of 2000 document can be applied to the guideline values of this document.

Project result 2: Unedited final document of Night Noise Guidelines for Europe

NIGHT NOISE GUIDELINES FOR EUROPE

UNEDITED*

*The edited final version can be accessible on the WHO website at http://www.euro.who.int/noise from April 2007.

2 April 2007

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Note from the editor:
The report is composed from contributions from authors who have very different cultural and linguistic backgrounds, and for the most part English is not their native language.
Although the texts are screened for ambiguities, grammatical and spelling errors, no attempt has been made to uniformize the style. This choice of the editor is born from respect for the authors.
MvdB

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CHAPTER I . INTRODUCTION: METHODS AND CRITERIA

With regard to sleep and waking, we must consider what they are: whether they are peculiar to soul or to body, or common to both; and if common, to what part of soul or body they appertain: further, from what cause it arises that they are attributes of animals, and whether all animals share in them both, or some partake of the one only, others of the other only, or some partake of neither and some of both.

Aristotle, On sleep and sleeplessness, 350 BC

1 Introduction

1.1 Existing policy documents for night time noise

The aim of this document is to present guidance for the exposure to noise during sleep. What is already available?
There are 3 related documents at the international level:
- Guidelines for community noise (WHO, 2000)
- EU Directive 2002/49 EC relating to the assessment and management of environmental noise(European Commission, 2002b)
- Position Paper on the dose-effect relationships for night time noise (European Commission, 2004)

In chapter V the relation with the Guidelines for community noise will be fully explained.
The EU directive on the Assessment and Evaluation and management of environmental noise (or, as it usually addressed, the Environmental Noise Directive, END) establishes that Member States should make noise maps (in 2007) and action plans (2008) for part of their territory. The noise maps should present noise levels expressed in the harmonised indicators Lden and L_{night}. Although in the first round only between 20 and 30% of the population will be covered, it is expected that through the use of harmonised methods and indicators a deeper insight will be gained in the exposure of the EU population to noise. The END does not however set any limit values: on basis of the subsidiarity principle this is left to the Member States. The Directive does however force the Member States to report on their limit values and express them in the standard indicators. On the CIRCA website (Communication and Information Resource Centre Administrator,EU-Commission, 2006) an overview of the data reported to the Commission can be found. From the 25 Member States 10 reported on the L_{night} limits. In table I.1 some of this data is summarized.

Table I.1. Reported L_{night} limit values for road traffic noise in new residential areas. EU-Commission, 2006

(EU-Member) State	L_{night}
France	62
Germany	49
Spain	45
Netherlands	40
Austria	50
Sweden	51 (converted from L_{Aeq} limit 30 dB(A) inside bedroom)
Finland	46

Hungary	55
Latvia	40
Estonia	45
Switzerland	50

Due to differences in legal system it is hard to predict what the actual effect of a certain limit value is. It could be a relative high value but rigidly enforced, or a very low value with no legal binding whatsoever.

The Position Paper on night time noise is foreseen in the END (Annex III) and aims to give the competent authorities a tool to evaluate the impact on the population. It neither does provide limit values or guidelines however. The same information that was used in the Position Paper plays also a role in this Guideline.

1.2 General model

There is no doubt a relation between sleep and health and well-being, as most of us know from personal experience. That doesn't mean however that this relation is simple. People who don't sleep well may not feel well the day after, but the reverse is also true: unfit persons may have a disturbed sleep. Untangling the relations between health and disturbed sleep (between the many causes night noise is but one) proved difficult, and figure 10 at the end of chapter II shows why.
The general structure of the report is given in figure 1: evidence for the effects of night-time noise on health (c) is supported by evidence on the indirect route via (a) and (b).in Chapter II the relations between sleep and health are examined (relation b in figure 1), which involves clinical evidence from sleep laboratories, but also the evidence from animal experiments. In Chapter III it is shown how noise disturbs sleep from the basic, autonomous level up to conscious awakenings: relation (a). Chapter IV presents the evidence between night noise and health and well-being: relation (c) in figure 1. The last Chapter, V, then provides guidance on reducing health impacts caused by night noise exposure.

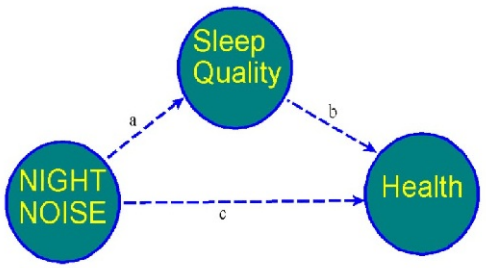

Figure 1. Evidence for the effects of night noise on health (c) is supported by evidence on the indirect route via (a) and (b).

2 Strength of evidence

2.1 Basic concepts.

This document uses well established practices from other disciplines and policy fields. Of main interest here are evidence based medicine, use of epidemiological evidence for environmental risk assessment and the experiences with – principally- air quality guidelines.

The concept of "evidence" is further formalized, as variations in wording and scope are currently in use.

2.2 Risk assessment and risk control

Figure 2 outlines a general approach for risk assessment. This approach consists of the

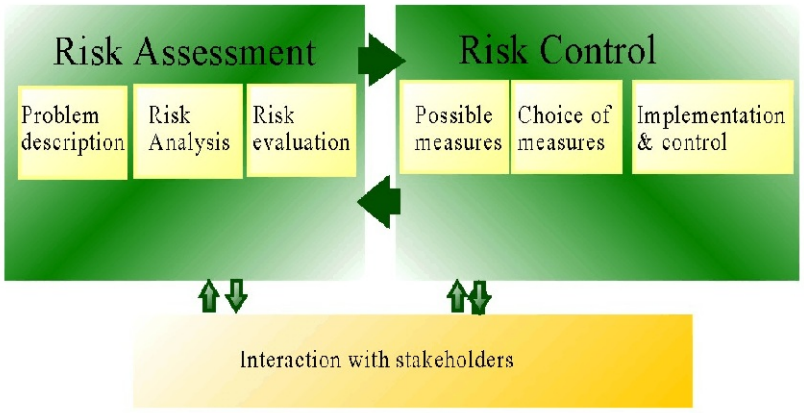

Figure 2. Elements of risk control

following steps:

1. Problem description: assessing the impact on the population
2. Risk analysis: evaluation of impact
3. Risk evaluation: assessing impact considered undesirable
4. Assessment of options to avoid or reduce impact
5. Cost-benefit analysis of the options or of mix of options
6. Assessment of the preferred option
7. Implementation and control

It is important to observe that guideline values can be an input to as well as an output of this process. At lower levels of decisions (a particular infrastructure project for instance) a preset guideline value reduces - intentionally- the degrees of freedom in the process. At the highest national or international level a guideline value is the outcome.

As the scope of this document is to present the health consequences of night time noise exposure (and not so much the economic outcomes of the choice of a certain value) it concentrates on the first 3 elements in the risk-assessment block.
The following questions need to be addressed:

A. What is the strength of the available evidence – what are the uncertainties?
B. What is the health significance for the effects found?
C. How serious is the impact on health?
D. Does every instance of exposure lead to an effect and how are they related?
E. How can the number of affected people be established?

2.3 Cause-effect Chain

Underlying this approach is the notion of a cause-effect chain
between environmental factors and health. Symbolically simplified in figure 3.

Figure 3. Cause effect chain

Important questions are:

1. Is there a causal relation between one link in the chain and the next and
2. What are the intervening factors in that relation?
3. How strong is the evidence for the relations?

The last question is the hardest to answer, as "strength of evidence" is not easy to express in simple numbers or labels. There are two forms of uncertainty because of variability of outcomes and uncertainty due to a lack in knowledge.
For the purpose of this document the following classification will be used (largely based on the IARC (International Agency for Research on Cancer) criteria:

Grade of evidence	Criteria
Sufficient evidence	A causal relation has been established between exposure to night time noise and an effect. In studies where coincidence, bias and distortion could reasonably be excluded the relation could be observed and it is plausible that the effect is (also) caused by the exposure
Limited evidence	A relation was observed between exposure to night time noise and an effect in studies where coincidence, bias and distortion couldn't reasonably be excluded. The relation is however plausible.
	A direct relation between cause and effect has not been

	observed, but there is indirect evidence of good quality and the relation is plausible. Indirect evidence is assumed if exposure leads to an intermediate effect and other studies prove that the intermediate effect leads to the effect.
Insufficient evidence	Available studies are of low quality and lack significance to allow conclusions about causality of the relation between exposure and effect. Plausibility of the relation is limited or absent

2.4 Procedure for deriving guidelines

The following procedure was followed in order to derive an ordering of guideline values:
- collection of relevant data
- evaluation of data in terms of strength of evidence
- evaluation of data in terms of biological effects, health & well-being
- ranking of guideline values (No Observed (Adverse) Effect Levels , Disability Adjusted Life Year-weights)

This procedure is essentially the same as in other guideline documents, be it that steps are more explicitly formalized. A major difference is that sound is a natural environmental quality, which makes defining a no-effect level a futile exercise. Therefore the choice for a series of levels with increasing severeness of effects is made.

3 Considerations with regard to night-time noise indicators

Briefly, the fundamental choices of night time noise indicators with respect to:
- length of night
- use of single event descriptors
- long-term average

are commented on to assist the reader in understanding the relations presented in later chapters.

3.1 Length of night

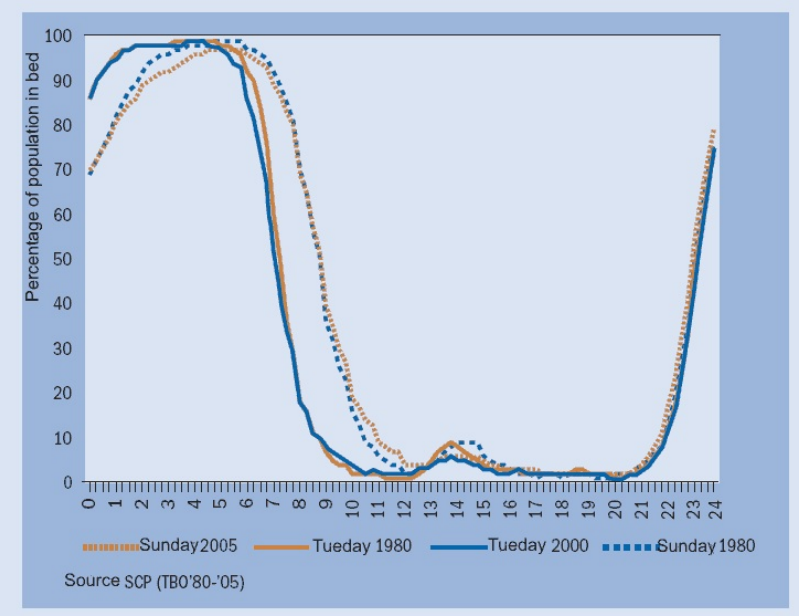

Figure 4. Sleep pattern of Dutch population on weekdays and sundays; 1980-2005

Time use studies (Centre for time use research, 2006) show that the average time adult people are in bed is around 7.5 hours, so the real average sleeping time is somewhat shorter. Due to personal factors like age and genetic factors there is considerable variation in sleeping time and in begin and end times. For these reasons, a fixed interval of 8 hours is a minimal choice for night-time protection. From figure 4 it can be noted that around 50% of the population is protected with an interval of 8 hours and it would take a period of 10 hours to protect 80%. On Sunday sleeping time is consistently 1 hour longer, probably due to people recovering from sleep debt incurred during the week. Data for other countries are readily available but this is the only study covering a long period in a consistent way. A recent result from Portugal shows a an almost identical time pattern as far as sleep is concerned.
It should also be borne in mind that (young) children have longer sleeping times.

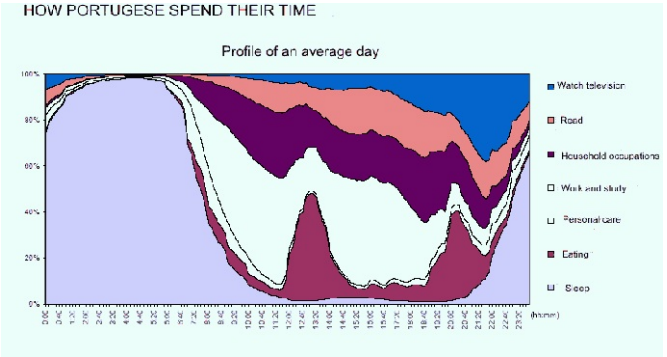

Figure 5. Time use study from Portugal, 2005.

3.2 Event or long-term descriptor

Much attention has been paid to the use of single event descriptors like L_{Amax} (maximum outdoor sound pressure level) and SEL (Sound exposure level). As the Position Paper on Indicators (EC, 2000) points out, this is an important laboratory tool to describe instantaneous reactions to noise. But when it comes to long-term protection, the number of events is equally important. The possibility of predicting after-effects like sleepiness,

reaction time, sleeping pill use and health complaints in particular require a combination of number of events and their level instead of just the average L_{Amax} or average SEL. For events with a similar time pattern there is a relatively simple relation between L_{Amax} and SEL, and therefore between L_{Amax} and L_{night} (night-time noise indicator as defined by the END - see paragraph 3.4 below). Appendix I describes this in detail. For now let it suffice to say that a choice for an L_{night} level ties the L_{Amax} related effects to a maximum and therefore allows for a protective/conservative approach.
This reasoning applies also to the issue of long-term average. A value for an arbitrary single night will, except in extreme cases, bear no relationship to an individual's long term health status, whereas a sustained sufficient high level over a long period may.

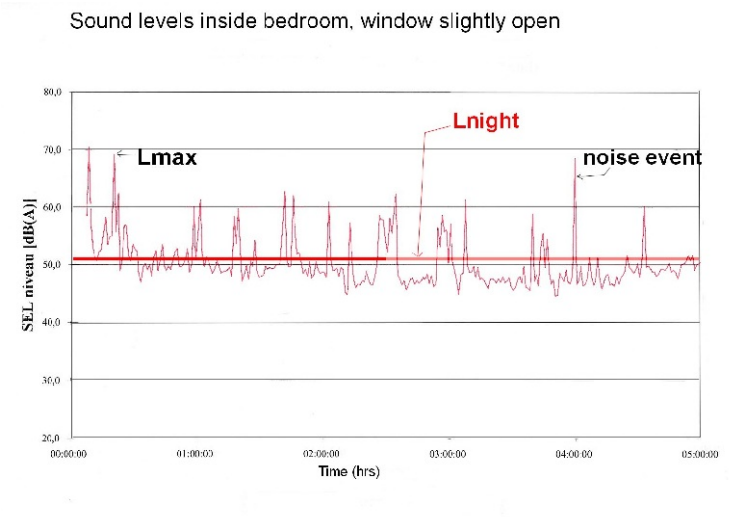

Figure 6. Relation between L_{night}, L_{Amax} and SEL
This is a sound recording in a bedroom for one night. The top of the peaks are the L_{Amax}-levels, the total energy is the L_{night} (thick horizontal line). The sound energy in one event is the SEL (not represented). In reality the L_{night} is the average over all nights in one year.

3.3 Number of events

There is no generally accepted way to count the number of - relevant- noise events. Proposals range from the number of measured L_{Amax} 's, the number of units (vehicles, planes, trains) passing by, to the number exceeding a certain L_{Amax} level (commonly indicated by NAxx; NA70 is the number of events higher than 70 dB(A)) .

3.4 Conversion between indicators

3.4.1 introduction

L_{night} is defined as the 1 year L_{Aeq} over 8 hours outside at the most exposed facade. For the purpose of strategic noise mapping and reporting the height is fixed at 4 meter. As L_{night} is a relatively new definition and because the studies rarely cover such a long period, the research data are expressed rarely in $L_{night.}$ The most frequently used noise descriptor in sleep research is the L_{Amax} or SEL near the sleeper. This means that a considerable amount of conversion work needs to be done if relations are to be expressed in L_{night}.
There are 4 issues:
- conversion between SEL and L_{Amax}
- conversion from instantaneous to long-term
- conversion from inside to outside
- conversion from (outside) bedroom level to most exposed facade

Further background information on these issues is provided in section 6. This section details the conversions that are actually carried out.

3.4.2 SEL to L_{Amax}

SEL is only used for aircraft noise in this report and, according to Ollerhead et. al., (1992) from ground- based measurements, the following relation was found:

$$SEL=23.9+0.81*L_{Amax} . \quad [1]$$

A more general approach can be used to estimate SEL for transportation noise.
If the shape of the time pattern of the sound level can be approximated by a block form, then $SEL \approx {}_{LAmax} + 10lg(t)$, where t (in seconds) is the duration of the noise event. This rule can be used *inter alia* for a long freight train that passes at a short distance. When t is in the range from 3 to 30 seconds, then SEL is 5 to 15 dB(A) higher than L_{Amax}. For most passages of aircraft, road vehicles or trains, the shape of the time pattern of the sound level can be better approximated with a triangle. If the sound level increase with rate (a) (in dB per second), thereafter is at its maximum for a short duration before it decreases with rate (-a), then $SEL \approx L_{Amax} - 10lg(a) + 9.4$. Depending on the distance to the source, for most dwellings near transportation sources the rate of increase is in the order of a few dB per second up to 5 dB per second. When (a) is in the range from 9 to 1 dB per second, then SEL is 0 to 9 dB higher than L_{Amax} .

3.4.3 Events to long term

When the SEL values are known (if necessary after converting from L_{Amax}) they can be converted to L_{night}. In general terms, the relation between L_{night} and SEL is:

$$L_{night} = 10*lg \sum_i 10^{SELi/10} - 10*lg (T).$$

If all (N) events have approximately the same SEL-level, this may be reduced to:

$$L_{night} = SEL + 10*lg(N) – 70.2 \quad [2]$$

in which
N = the number of events occurring in period T
T = time during which the events occur in seconds. For a (night) year 10lg(T) is 70.2

The notation adheres to the END where the L_{night} is defined as a year average at the most exposed facade. Any reference to an inside level is noted as such, eg. $L_{night,inside}$. In order to avoid any doubt the notation $L_{night,outside}$ may be used for instance in tables where both occur.

3.4.4 Inside to outside

As the L_{night} is a year value, the insulation value is also to be expressed as such. This means that if the insulation value is 30 dB with windows closed and 15 dB with windows open, the resulting value is 18 dB if the window is open 50% of the time. If these windows are closed only 10% of the time, the result is little more than 15 dB. The issue is complicated by the fact that closing behaviour is, to a certain extent, dependent on noise level.
When results about effects are expressed with indoor (i.e. inside bedrooms) exposure levels, they need to be converted to L_{night}., in accordance with the END definition. The most important assumption is the correction for inside levels to outside levels. An average level

difference of 21 has been chosen, as this takes into account that even in well-insulated houses windows may be open a large part of the year. In general:

$$L_{night} = L_{night,inside} + Y \text{ dB} \qquad [3]$$

Y is the year average insulation value of the (bedroom) facade. In this report a default value of 21 is used (see also section 3.5). It should be stressed that this conversion is thought to be highly dependent on local building habits, climate and window opening behaviour.

3.4.5 Most exposed facade

If an inside level is converted to an outside level with [3], it is assumed that this is equivalent to a L_{night} value on the most exposed facade. No information is available on bedroom position and use, so no explicit conversion factor can be given in this report.
This means that the effect estimated on the basis of L_{night} corresponds to an upper limit, because part of the bedrooms will be on a less exposed facade. If an estimate of the exposed population is based on a relation derived with [3], the actual prevalence will be less. From a practical point of view the most exposed facade safeguards protection in cases where there is a possibility that rooms can be swapped .
It should be pointed out that the above does not apply if a relation is based on L_{night} values which are directly measured or computed . These relations will show a large variation because of a misclassification effect, but they give a "correct" estimate of the prevalence of effects in the population. In other words, in some cases a low effect may be attributed to a high L_{night} because the bedroom is on the quiet side.

3.5 Inside / outside differences

Night-time environmental noise affects residents mainly inside their homes. In order to protect residents inside their homes against noise from the outside sources, attention should be focussed on windows since they are generally the weakest points in the sound propagation path. Roofs must also be considered with regard to aircraft noise.

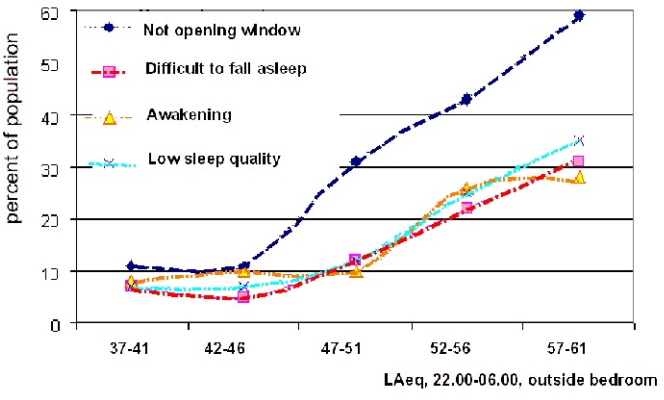

Figure 7. Results from Swedish Soundscape research program, Öhrström, E.(2004).

There are many types of window in the EU, varying from single thin panes within frames without additional insulation, to four-pane windows within insulated frames. The simplest types of facade have a sound reduction (from outside to inside) of usually less than 24 dB, and the most elaborated facades (built to cope with cold climates, for example), have sound reductions of more than 45 dB. In central Europe, most windows are of the double-glazing type, mounted in a rigid and well-insulated frame.

Their range of sound reduction is between 30 and 35 dB when closed.

When night-time environmental noise reaches high levels, residents tend to close their bedroom windows (cf.Diaz e.a.,2004, Langdon, F.J. & Buller, I.B. (1977), Scharnberg,ea (1982) , Schreckenberg, D, ea,(1999)). The two latter studies found that more than 50% of bedroom windows are closed when outside road traffic noise levels exceed 55 dB (L_{Aeq}). Nevertheless, while residents with closed windows reported a reduction of sleep disturbances due to noise, they also reported an increase in sleep disturbances due to poor ventilation. Schreckenberg ea. (1999) report a much steeper increase in the incidence of closed windows when road traffic noise reaches high levels than is the case with increased levels of railway noise. Even when night-time noise levels reach 55 dB, only 35 % of the residents exposed to railway noise reported that they close their windows at night. These finding have been replicated in Sweden, according to recent results from the Swedish soundscape research programme on road traffic noise (figure 7).

When windows are slightly open, outside sound levels are usually reduced by 10 – 15 dB. It should be kept in mind that most European residents want to keep their bedroom windows slightly open at night in order to provide for proper ventilation (Lambert, J (1985), Lambert, J (1994), Scharnberg ea, and the WHO paper on community noise (WHO, 2000) also recommends that people should be able to sleep with their bedroom windows open.
In Passchier-Vermeer (2002) detailed noise measurements were carried out inside and outside the bedroom and at the same time window position was measured with sensors. The results showed that windows are fully closed in only in 25% of the nights.

Table I.3.1 Window positions during research period (April-November) Passchier-Vermeer et. al. (2002)

Window position	% Nights
Closed	25
Slightly open	43
Hand width	23
Half open	5
Fully Opened	4

This results in average inside-outside differences of around 21 dB, with there being only a slight difference between single and double-glazed windows. The survey did not include dwellings which had been specifically insulated against noise. Nevertheless, there was a large variation in insulation values.

Table I.3.2 Average inside-outside differences in dB(A)

	single-glazed window	double-glazed window
average difference at night	21.3	22.2

It should be stressed that this figure only applies to facades that have not been fitted with special appliances to reduce noise impact. To give an extreme example of where this general finding does not apply, rooms may be equipped with air-conditioning so that windows can stay closed, or could even be sealed. Less drastic provisions are sound-attenuated ventilation openings. Little is known however about the experiences (long-term use, appreciation) of these and other solutions by inhabitants. For example sound attenuated ventilation openings are sometimes blocked in order to cut out draughts.

3.6 Background level

A simple definition of background level or "ambient noise" level is the noise not targeted for measurement or calculation. The background noise can interfere with the target noise in a number of ways:
- mask the signal
- interact physically
- interact psychologically

As this report is often dealing with low level target noise, masking is an important issue. The other 2 interactions are more important in the domain of annoyance.
Masking however is a complex process; the human auditory system is uncanny good in separating signals from a "background". Microphones (and the software behind it) are slow to catch up, as can be proved by the unsatisfactory results when it comes to automatically recognize aircraft in long term unmanned measuring stations.
The rule of thumb that a noise can be considered masked if the signal is 10 dB below the background, is only valid is the noises have the same frequency composition and if they actually occur at the same time. This is particularly important to stress where L_{Aeq} levels are compared: even a relatively continuous motorway of 50 dB cannot mask aircraft noise of 30 dB, because this may be composed of 5 aircraft arriving at an L_{Amax} of 57 dB. Birds on the other hand can neither, because the frequency domains do not overlap.
Another factor relevant for this report is that background levels are lower at night time than they are at daytime. This is true for most man made noises, but also for the natural background levels as wind speeds at night slow down.
Most levels mentioned in this report do not take background levels into account - explicitly. Where long term L_{Aeq} levels are related to effects like hypertension and self reported sleep disturbance, background levels are ignored, but they could obscure the effect at the lower end of the scale. This then influences the lowest level where an effect starts to occur.
In sleep laboratory studies the back ground level is kept as low as possible, around 30 dB. The background of the instrumentation is 20 dB.
In semi-field experiments is has been found that background noise levels inside bedrooms are very low, also because people tend to choose their bedrooms on the quiet side of the building. This may have the side effect of exposing children to higher levels.

3.7 Choice of indicators for regulatory purposes

From the scientific point of view the correct choice for a noise indicator is the performance in predicting the effect. There are however a number of additional criteria which may influence the choice. First is that for different health end points different indicators could be suitable. Further considerations are more of political nature, as mentioned in the Position Paper on Indicators (European Commission, 2000). Indicators should also be easy to explain to the public - intuitive understanding, avoid unnecessary breaks with current practice and

enforceable. This probably why in many countries L_{Amax} is a popular indicator: it has undeniable qualities in these areas.
This also the case for LAeq indicators for short periods; like 1 hr or a few hours in the centre of the night. Other fashionable indicators are those looking at the number above a threshold (NAxx).
For these indicators the relation between health end points and their values is either not well established, or the correlation between them and current indicators is high, or the correlation between the indicator and an effect is low.

4 Exposure in the population

4.1 Noise levels

Surprisingly little information is available on the exposure of houses to night time noise. It may be expected that in a few years time the END will allow to create a substantial database of such levels, but up till now only 2 countries have detailed data available.

Table I.4.1 Percentage of dwellings per noise class of L_{night}

	L_{night} in dB				
Country	40-45	46-50	51-55	56-60	61-65+
Switzerland (Müller-Wenk 2002)	--	24%	14%	7%	2%
Netherlands (Nijland and Jabben, 2004)	25%	31%	19%	6%	1%

Notwithstanding the obvious differences between these 2 countries the data show a remarkable similarity.

A first result of the END comes from a study into night regulations for -large - airports (Wubben and Busink, 2004).

Table I.4.2 Number of inhabitants within 45 L_{night} contour

Airport	Number of inhabitants	Number of night operations per year	Night operations as percentage of daytime-operations
Amsterdam	21,863	23,462	5.8%
Frankfurt	134,651	46,662	10.1%
London	477,289	26,465	5.7%
Paris	180,184	51,683	10.3%

4.2 Reported night noise disturbance

Complaints about night-time exposure to noise are wide spread and not exactly new: Roman writers used to complain about racket in the streets at night (Juvenalis, AD160). Surprisingly, little detailed information is available today.
Nevertheless, data collected from a few member states can help to give an impression of the

order of magnitude of effects.

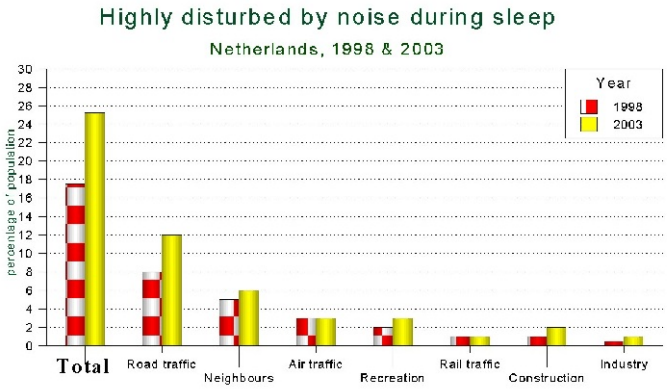

Figure 8. Percent of population stating to be highly disturbed by noise during night time (RIVM survey 2003 (vDongen 2004)).

Figure 8 shows the relative contributions to overall sleep disturbance caused by noise from different sources in the Netherlands. These data were derived from surveys in 1998 and 2003 in which 4000 and 2000 people(van Dongen et al 2004), randomly selected, were asked: "To what extent is your sleep disturbed by noise from (*source mentioned*)...." on a scale from 1 to 10. People recording the 3 highest points in the scale were considered "highly disturbed", according to an international convention . The totals are calculated from the number of people reporting serious sleep disturbance from 1 or more sources.

Unfortunately, comparable research data from other countries or regions is not available, and there is reason to believe that there may be considerable differences in the figures. Since this study is based on a survey conducted in the Netherlands, it is not representative for other Member States in the EU. General (not specific for night time) annoyance data from Germany and the UK give an indication that the same order of magnitude in terms of number of people is affected.

However the fact that other nuisances may contribute significantly to overall sleep disturbance by noise should not be overlooked. Further research on this topic is needed in order to gain an insight into the contribution of various noise sources to sleep disturbance.

5 Conclusions

The methods and criteria for deriving guidelines rest on well established procedures from epidemiology. To relate the effects to the dose, standard metrics will be used where ever available. If possible the values found in literature will be converted to avoid confusion. Most of the conversions are relatively straightforward and depend on physical laws, others, in particular the conversion between outside and inside levels, depend on local factors and should be used only if no other information is available,

Information about night time noise exposure is relatively scarce, even where 10 EU- Member States have limit values for night time noise. The END could substantially increase this information (large scale noise mapping is foreseen in 2007), increasing the demand for guidance.

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CHAPTER II ON THE RELATION BETWEEN SLEEP AND HEALTH

A night of quiet and repose in the profound silence of Dingley Dell, and an hour's breathing of its fresh and fragrant air on the ensuing morning, completely recovered Mr. Pickwick from the effects of his late fatigue of body and anxiety of mind.- Charles Dickens, the Pickwick Papers

1 Sleep, normal sleep, definitions of sleep disturbance, characteristics mechanisms, the insomnia model *(Groselj)*

1.1 Normal sleep (objective measurements)

Sleep is part of living and with wakefulness forms an inherent biological rhythm (Cooper, 1994). Normal sleep can be defined in objective or subjective manner. The objective criteria are defined with a polysomnographic recording (PSG) of sleep, the method that is measuring different physiological functions during sleep. Minimal polygraphic requirements to score sleep adequately include two channels of EEG-electroencephalography, one channel for the EOG-electrooculogram, and one channel for the submental EMG-electromyography. In routine PSG, additional channels are used to assess respiration, leg movements, oxygenation and cardiac rhythm (Pedley, 2003).
Scoring of sleep stages is usually done on an epoch-by-epoch basis, with 30 seconds epoch length used as a standard. Epochs are scored according to the guidelines of Rechtschaffen and Kales (1968). Each epoch is scored as the stage that occupies more than 50% of that epoch. Sleep can be staged in the following stages:

Arousal is not a uniform concept and has been defined differently by different researchers. Commonly, the occurrence of alpha rhythms is required for EEG arousal. Depending on the additional requirements and on the length of time that the slower cortical rhythms are interrupted, arousals have been called e.g. micro-arousal, minor arousal, EEG awakening or transient activation phases. The American Sleep Disorders Association (1992, 1997) devised a scoring system, taking sequences of 3-15 seconds into account for transient arousals which are not transferred to macroscopic behavioural awakening. 11 further criteria must be met. See also Chapter III.1.2
Vegetative arousals are activations of the sympathic nervous system**.**
Stage W corresponds to the waking stage and is characterized by alpha activity or low-voltage, mixed-frequency EEG activity. REMs, eye blinks, and tonic EMG activity are usually present.
Stage 1 is scored when more than 50% of an epoch is low-voltage, 2-7 Hz activity. Vertex waves may occur in late stage 1. Slow rolling eye movements lasting several seconds are routinely seen early in stage 1, but K complexes and sleep spindles are absent by definition. Tonic EMG activity is usually less than that of relaxed wakefulness.
Stage 2 requires the presence of sleep spindles or K complexes, and less than 20% of the epoch contains delta activity. Sleep spindles bursts must last at least 0,5 seconds before they can be scored. K complexes are defined as biphasic vertex sharp waves with a total duration of greater than 0,5 seconds.
Stage 3 is scored when 20% to 50% of an epoch consists of delta activity that is 2 Hz or slower and is greater than 75 µV in amplitude. Sleep spindles may or may not be present.
Stage 4 is scored when more than 50% of an epoch consists of delta activity that is 2 Hz or slower and is more than 75 µV in amplitude. Reliable differentiation of stages 3 and 4 sleep is difficult by visual inspection, and most laboratories combine stages 3 and 4 into a single

determination of slow-wave sleep.
Stage REM is characterized by relatively low-voltage, mixed-frequency EEG activity with episodic REMs and absent or markedly reduced axial EMG activity. Phasic EMG activity may occur, but tonic activity must be at a level that is as low as, or lower than, that during any other time in the study. Sleep spindles and K complexes are absent. Series of 2- to 5- Hz vertex-negative "saw-tooth waves" occur, particularly just before phasic REM activity. The requirements to score sleep as REM are REMs, low or absent axial EMG, and typical mixed-frequency EEG recording that does not preclude the scoring of REM.
Movement time is scored when more than 50% of an epoch is obscured by movement artifact. Movement time must be preceded or followed by sleep and is thus distinguished from movement occurring during wakefulness.

Additional sleep values are determined from each sleep study and contribute to the clinical interpretation of the study. These additional variables include the following:

Recording time is the time elapsed between "lights out" and "lights on" at the end of the study.
Total sleep time (TST) is the total time occupied by stage 1, stage 2, slow-wave sleep, and REM sleep.
Sleep efficiency (SE) is defined as total sleep time divided by recording time and is expressed as a percentage.
Sleep latency (SL) is the time from "lights off" to the first epoch scored as sleep. Some authors prefer to use the first epoch of stage 2 in order to be more confident about identifying the onset of sustained sleep. However, when sleep is very disrupted, there may be an extended period of time from recognition of stage 1 until an epoch that can be scored as stage 2.
REM latency is the time from sleep onset (as described earlier) to the first epoch scored as REM, minus any intervening epochs scored as wakefulness.
Sleep stage percentages (% in stage 1, stage 2, slow-wave sleep, and REM sleep) are determined by dividing time recorded in each sleep stage by total sleep time.
Wake after sleep onset (WASO) is time spent awake after sleep onset.

The objective criteria defining normal sleep are based on: sleep latency, total sleep time, sleep efficiency and the number of awakenings, including cortical arousals. However all this parameters are age-related, sometimes also gender related, and may vary from one individual to the other.

Normal sleep has a clearly defined architecture that is relatively stable. Predictable changes in sleep architecture occur with age. Beginning in the middle age, slow wave sleep becomes less prominent, the number of awakenings increase, and sleep efficiency decreases. Published information on normal sleep can serve as an outline for normal values in PSG (Williams et al, 2003, Table II.1.1), but each laboratory must study control subjects to identify any significant effects on sleep that result from differences in technique or environment (Current practice of clinical electroencephalography, Ebersole JS, Pedley TA (eds), 3rd ed., Lippincott Willimas &Wilkins, 2003).

Table II.1.1 Average normal values for adults of different ages (Williams RI, Karacan I, Hursch CJ. Electroencephalography (EEG) of human sleep: clinical applications. New York: John Wiley and Sons, 1974.

Sleep parameter(normal values)	20-29 years	40-49 years	60-69 years
TST (min)	419	389	407
Sleep efficiency (TST/TIB)	95%	91%	90%
WASO	1%	6%	8%
Stage 1(% of TST)	4%	8%	10%
Stage 2(% of TST)	46%	55%	57%
SWS(% of TST)	21%	8%	2%
REM(% of TST)	28%	23%	23%
Sleep latency(min)	15	10	8

Passchier-Vermeer (2003) reports that subjects not exposed to loud night noise typically report waking up one and a half to two times during an average sleep period, while the number of EEG awakenings including cortical arousals averages 10-12 per night (Table II.1.2).

Table II.1.2. Parameters of normal sleep (Passchier-Vermeer, 2003).

Subjects not exposed to loud night noise	Subjective report of number of awakenings	Number of EEG awakenings
Normal adult subjects	1.5-2	10-12

Night arousals result in fragmented sleep, which in turn leads to increased daytime sleepiness (EDS). Gold standard for the assessment of EDS is multiple sleep latency test (MSLT), which provides an objective quantification of "sleepiness". The preceding night's sleep requires the PSG to ensure adequate sleep and to exclude sleep disruption. During the day four or five nap times are scheduled every 2 hours. For each scheduled nap time patients lies down and assume a comfortable sleep position with the technician instructions to "close the eyes and attempt to sleep". Each nap is terminated 20 minutes after the nap time started if no sleep occurred, or after 15 minutes of continuous sleep as long as sleep onset criteria are met before the end of 20 minutes, or after 20 minutes if the patient awakens, even if the patients has been asleep less than 15 minutes. The patients are instructed to maintain wakefulness between the nap periods.

Table II.1.3. Mean sleep latency. (Current practice of clinical electroencephalography, Ebersole JS, Pedley TA (eds), 3rd ed., Lippincott Willimas&Wilkins, 2003).				
Group	MSLT(min)	NoneREM SO(% of group)	1REM SO(% of group)	2 or moreREM SO(% of group)
Narcoleptics	2,9 ± 2,7	2	2	96
EDS(nonnarcoleptic, non-sleep apneic)	8,7 ± 4,9	92	8	0
Controls	13,4 ± 4,3	100	0	0

1.2 Definitions of disturbed sleep

Sleep disorders are described and classified in the International classification of sleep disorders (newest reference: American Academy of Sleep Medicine. International classification of sleep disorders, 2nd ed.: Diagnostic and coding manual. Westchester, Illinois: American Academy of Sleep Medicine, 2005).
When sleep is permanently disturbed and becomes a sleep disorder, it is classified in the ICSD-2005 as Environmental sleep disorder. Environmental sleep disorder (of which noise -induced sleep disturbance is an example) is a sleep disturbance due to a disturbing environmental factor that causes a complaint of either insomnia or daytime fatigue and somnolence. Secondary deficits may result, including deficits in concentration, attention, and cognitive performance; reduced vigilance, daytime fatigue, malaise, depressed mood and irritability. The exact prevalence is not known. Fewer than 5% of patients seen at sleep disorders centres receive this diagnosis. The sex ratio is not known. The disorder may occur at any age, although the elderly are at more risk for developing this condition (ICSD-2005).

1.2.1 Insomnia

In the International Classification of Sleep disorders (second edition, 2005) the section Insomnia includes a group of sleep disorders all of which have in common the complaint of insomnia (adjustment insomnia, psycho physiological insomnia, paradoxical insomnia, idiopathic insomnia, . . .). Insomnia is defined by repeated difficulty with sleep initiation, duration, consolidation, or quality that occurs despite adequate time and opportunity for sleep and results in some form of daytime impairment. Insomnia is a symptom that often arises from primary medical illness, mental disorders and other sleep disorders, but may also arise from abuse or exposure. However, the general criteria for insomnia are the same for all sub groups of insomnias.

1.2.2 General Criteria for Insomnia

A. A complaint of difficulty initiation sleep, difficulty maintaining sleep, or waking up too early or sleep that is chronically non-restorative or poor in quality. In children, the sleep difficulty is often reported by the caretaker and may consist of observed bedtime resistance or inability to sleep independently.

B. The above sleep difficulty occurs despite adequate opportunity and circumstances for sleep.

C. At least one of the following forms of daytime impairment related to the nighttime sleep difficulty is reported by the patient:

1. fatigue or malaise
2. attention, concentration, or memory impairment
3. social or vocational dysfunction or poor school performance
4. mood disturbance or irritability
5. daytime sleepiness
6. motivation, energy, or initiative reduction
7. proneness for errors or accidents at work or while driving
8. tension, headaches, or gastrointestinal symptoms in response to sleep loss
9. concerns or worries about sleep

Defining the cause of a sleep-wake disturbance in an insomnia patient is a complex task since it is often multi factorial. In fact, a confluence of factors that support multiple insomnia diagnoses may be judged important in many patients with insomnia. Although selection of a single diagnosis is preferable and this selection may be appropriate, such a selection should not necessarily imply the absence of a subset of factors relevant to an alternate diagnosis. When criteria for multiple insomnia diagnosis are met, all relevant diagnosis should be assigned.

1.2.3 Environmental sleep disorder

In ICSD (2005) the environmental sleep disorder is listed in the category of "Other sleep disorders". Noise induced sleep disturbance is one of the disturbing environmental factors that causes a complaint of either insomnia or daytime fatigue and somnolence.

The diagnostic criteria for Environmental Sleep Disorder are:

A. The patient complains of insomnia, daytime fatigue, or a parasomnia. In case in which daytime fatigue is present, the daytime fatigue may occur as a result of the accompanying insomnia or as a result of poor quality of nocturnal sleep.

B. The complaint is temporally associated with the introduction of a physically measurable stimulus or environmental circumstance that disturbs sleep.

C. It is the physical properties, rather than the psychological meaning of the environmental factor, that accounts for the complaint.

D. The sleep disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.

The prevalence of environmental sleep disorder is not known. Fewer than 5% of patients seen at sleep disorders centres receive this diagnosis.

International standardization and quantification for measurement of the depth of sleep is based on Rechtschaffen and Kales criteria from 1968. Sleep is divided into 30 seconds

epochs, and a phase is only assessed if the specific features are evident for more than 50% of the epoch length. For example: wake is scored when at least 15 seconds of continous awakening is present. Arousal reactions not leading to macroscopic awakening were not included in the definition by Rechtschaffen and Kales. With the arousals as described by the American Sleep Disorders Association (1992) it is possible to display subvigilant sleep fragmentation, caused by intrinsic sensory and autonomic alarm reactions. An arousal index providing the arousal density (events/hour of sleep) was taken as a measure of the degree of severeness. 10-20 arousals/hour are considered as normal in the healthy adults. However, the use of EEG arousals with the ASDA definition provides no sufficient explanation of daytime sleepiness (Pitson and Stradling 1998, Ayas et al 2001) unless they are accompanied by vegetative arousals.
Regarding noise, different vigilance level assessment in various functional systems are important. Koella (1988) proposed investigations of vegetative, motoric, sensoric functions independently of each other. One of the possible factors indicating disturbed sleep is a vegetative arousal index. A vegetative arousal index of more than 30/h is certainly considered as serious, more than 20/h as intermediate and more than 10 as light forms of sleep disorder.
With respect to insomnia (1.2.1), there is the possibility of misclassification if the GP oversees the possible cause of excessive noise for the complaints. There is also the possibility that a insomnia is aggravated by noise.

1.3 Conclusions

Published information on normal sleep can serve as an outline for normal values in PSG, however these values are only informative, because each sleep laboratory must study control subjects to identify any significant effects on sleep that result from differences in technique or environment. Excessive daytime sleepiness is a consequence of disturbed night sleep and can be objectively assessed by MSLT, which provides an objective quantification of "sleepiness".

2 Long term health risk mediated by sleep disturbances *(Nevismalova)*

2.1 Stressors, neuro behavioural data and functional neuro imaging

It is generally accepted that insufficient sleep and particularly sleep loss has a great influence on metabolic and endocrine functions (Spiegel et al. 1999) as well as on inflammatory markers and contributes to cardiovascular risk. C-reactive protein (CRP) as a major marker of the acute phase response to inflammatory reaction promotes secretion of inflammatory mediators by vascular endothelium and may be therefore directly involved in the development of atherosclerotic lesions. CRP as a risk predictor of strokes and heart attacks linearly increases with total and/or partial sleep loss (Meier-Ewert et al. 2004).
An additional factor, closely linked to cardiovascular health, glucose regulation and weight control, is leptin. Leptin is one of the major regulators of energy homeostasis and its circadian profile interacts closely with sleep.
Leptin secretion increases at night and decreases during the day.
Decreased leptin level, that is connected with sleep loss, increases appetite, predisposes to weight gain, impaired glucose tolerance and impaired host response.
Other studies have been focussed on how sleep loss affects neuro behavioural functions, especially neuro cognitive performance. Functional brain imaging and EEG brain mapping studies show that the patterns of functional connectivity between brain regions evident in performing specific cognitive tasks are altered by sleep loss (NCSDR, 2003). To go by this

finding, the maintenance of sustained performance during sleep loss may depend upon regional functional plasticity. Cumulative waking neuro cognitive deficits and state instability that develop from chronic sleep loss have a basis in a neurobiological process that can integrate homoeostatic pressure for sleep across days. Increased efforts have helped to determine the roles of REM and NREM sleep in memory.
Functional brain imaging techniques such as positron emission tomography (PET), functional magnetic resonance imaging (fMRI), magnetic resonance spectroscopy (MRS), single photon emission computed tomography (SPECT), magneto electroencephalography (MEG) have recently been analysed in the study of sleep and waking (NCSDR, 2003). These techniques allow to measure metabolic and neurochemical activity throughout the brain, and can reveal dynamic patterns of regional cerebral activity during various brain states including stages of sleep and levels of alertness during wakefulness or during functional challenge. These techniques can also help identify both normal and abnormal sleep/wake processes.
In the last five years, functional neuro imaging techniques (particularly PET) have revealed that NREM sleep is associated with deactivation of centre encephalic regions (brainstem, thalamus, basal ganglia) and multi modal association cortices (e.g. prefrontal and superior temporal/inferior parietal regions). REM sleep is characterized by reactivation of all centre encephalic regions deactivated during NREM sleep except the multimodal association areas. PET studies during sleep-deprived wakefulness have revealed regional cerebral deactivations that are especially prominent in prefrontal and inferior parietal/superior temporal cortices, and in thalamus. This pattern is consistent and helpful in explaining the nature of cognitive performance deficits that occur during sleep loss. As revealed by means of fMRI techniques during cognitive tasks performance, the maintenance of performance following sleep loss may be a function of the extent, to which other cortical brain regions can be recruited for task performance in the sleep-deprived state.
PET, SPECT and fMRI studies have revealed, in depressed patients, initially elevated activation in anterior cingulate and medial orbital cortices (NCSDR, 2003). In these patients, sleep deprivation reduces this regional hyper-activation, and improvements in mood are a function of the extent, to which this activity is reduced. These studies point to possible mechanisms by which antidepressant drugs may exert their effects. Further research should be oriented towards neuroimaging and measurements of changes in the brain´s metabolic activity at the neurotransmitter level.

2.2 Signals mediated by subcortical area (the amygdala) and the role of stress hormones in sleep disturbance and their health consequence

Experimental as well as clinical studies (Waye et al. 2003, Ising and Kruppa, 2004) showed that the first and fastest signal of stressors introduced by noise is detected and mediated by a subcortical area represented by the amygdala while the stress response to noise is mediated primarily by the hypothalamus-pituitary-adrenal (HPA) axis. A major intrinsic marker of the circadian rhythm is in the level of circulating corticosteroids derived from activity within the HPA axis. A protracted stress response with activation of HPA axis is a major physiological response to environmental stressors. The cortisol response to awakening is an index of adrenocortical activity and long-term nocturnal noise exposures may lead, in persons liable to be stressed by noise, to permanently increased cortisol concentration above the normal range. The hypothesis of an increase risk of cardiovascular diseases to be connected with the stress concepts is generally accepted (Ising and Kruppa, 2004, Ekstedt et al. 2004), Stress reactions may lead to derangement of normal neuro-vegetative and hormonal processes and influence vital body functions. Cardiovascular parameters such as blood pressure, cardiac function,

serum cholesterol, triglycerides, free fatty acids, hemostatic factors (fibrinogen) impede the blood flow in terms of increased viscosity and presumably blood sugar concentration as well. Insulin resistance and diabetes mellitus, stress ulcers and immune system deficiency is frequent consequence of stress reaction as well. Disturbed sleep influences immuno-suppression and diminished protein synthesis (Horne 1988).
Besides non-specific stress response on functioning the immune system, there is considerable evidence for a relation between sleep, especially SWS, and the immune system (Brown 1992). This evidence includes surges of certain immune parameters and growth hormone at onset of SWS, correlation of NREM sleep, total sleep time and sleep efficiency with natural killer cell activity, and correlation of SWS with recovery from infections. These data, taken together with information on the effect of intermittent transportation noise on SWS during the first sleep cycles and overnight, suggest that the immune response could be impacted also directly by environmental noise during sleep (Carter 1996).

2.3 Sleep restriction, environmental stressors (noise) and behavioural, medical and social health consequences of insufficient sleep. Risk of morbidity and mortality.

Sleep restriction due to environmental stressors leads to primary sleep disorders but health is influenced also by a consequence of stress response to noise mediated by HPA axis and/or by restriction of specific sleep stages (see above).
Sleep restriction leads approximately in 40% of affected subjects to daytime sleepiness that interferes with work and social functioning. Excessive daytime sleepiness is thus a major public health problem as a consequence of interference with daily activities including cognitive problem, motor vehicle crashes (especially at night), poor job performance and reduced productivity (Lavie et al. 2002). In the last decade, experimentally based data have been collected on chronic restriction of sleep (by one to four hours at night), accumulating daytime sleepiness and cognitive impairment. Most individuals develop cognitive deficits from chronic sleep debt after only a few nights of reduced sleep quality or quantity; new evidence suggests additional important health-related consequences from sleep debt related to common viral illnesses, diabetes, obesity, heart disease, depression and other age-related chronic disorders.
The effect and consequence of sleep deprivation is summarized in Table II.2.1 (according to Lavie et al. 2002).

Table II.2.1 Consequences of sleep deprivation

Type	Short term	Long term
Behavioural	Sleepiness Mood changes Irritability and Nervousness	Depression /mania Violence
Cognitive	Impairment of function	Newly learned skills Short-tem memory Complex task Slow reaction time
Neurological	Mild and quickly reversible	Cerebellar ataxia, nystagmus, tremor, ptosis, slurred speech, increased reflexes, increased sensitivity to pain

Biochemical	Increased metabolic rate Increased thyroid activity Insulin resistance	Decreased weight despite increased caloric intake (in animals) Diabetes, obesity (in human)
Others	Hypothermia Immune function impairment	Susceptibility to viral illness

The relationship between sleep quantity and quality and estimates of morbidity and mortality remains controversial. Epidemiological data (NCSDR, 2003) suggest that habitually short sleep (less than six hours sleep per night) is associated with increased mortality. Epidemiological studies of the recent years elucidated, however, that too much sleep is a problem as well. Kripke et al. (2002) evaluated a questionnaire study of 1.1 million men and women aged 30-102 years and found the lowest mortality risk between respondents sleeping 7 h/night. Mortality risk significantly increased when sleep duration was less than 6 or higher than 8 h/night. Similar results published also other authors (Tamakoshi and Ohno, 2004, Patel et al. 2004). Patel et al. (2004) in a prospective study of sleep duration and mortality risk in 5409 women confirmed previous findings that mortality risk is lowest among those sleeping 6 to 7 hours/night. The mortality risk for death of other causes significantly increased in women with sleep amount less than 5 and more than 9h/night. It is not clear how the length of sleep can increase this risk, although animal evidence points to a direct link between sleep time and life span (see section 5). Up to now, no epidemiological prospective study has been published, examining the relationship between sleep and health outcomes (morbidity and mortality) with subjective and objective estimates. Recent studies, however, show that sleep duration of least eight hours is necessary for optimal performance and for prevention of daytime sleepiness and accumulation of sleep debt.

Environmental stressors including noise mostly cause insomnia (see . Insomnia also involves daytime consequences, such as "tiredness, lack of energy, difficulty concentrating, irritability." A reasonable prevalence estimate for chronic insomnia in the general population is about 10%, when considering insomnia of any duration or severity - between 30% and 50%, and their incidence increases with aging. In the course of perimenopausal time, women are particularly vulnerable to developing this complaint.

The major consequences and co-morbidity of chronic insomnia (see Table II.2.2) comprise behavioural, psychiatric and medical problems; several studies also report a higher mortality risk (Zorick and Walsh 2000).

TableII.2. 2: Consequences of chronic insomnia	
Behavioral	Poor performance at work, fatigue, memory difficulties, concentration problems, automobile accidents
Psychiatric	Depression, anxiety conditions, alcohol and other substance abuse
Medical	Cardiovascular, respiratory, renal, gastrointestinal, musculosceletal disorders.

	Obesity Impaired immune system function
Mortality	Increased risk is reported

- Behavioural consequences: Transient (short lasting) insomnia is usually accompanied by spells of daytime sleepiness and performance impairment the next day. Persistent (long lasting) insomnia tends to be associated with poor performance at work, fatigue, memory difficulties, concentration problems and twice as many fatigue-related automobile accidents as in good sleepers.
- Psychiatric conditions: Epidemiological research indicates that the prevalence of any psychiatric disorder is two or three times higher in insomniacs. The risk of depression as a co-morbid state appears to be particularly strong, being approximately four times more likely in insomnia patients. Futhermore, insomnia may be an early marker for psychiatric disorders such as depression, anxiety conditions and alcohol abuse. Anxiety has been found quite common in insomniacs compared with general population. About 25-40% of insomnia patients are estimated to have significant anxiety, the abuse of alcohol and other substances is increased in insomniacs relative to good sleepers (Ford and Kamerow, 1989). Samples of unselected psychiatric patients have about a three-fold increase in the frequency of insomnia compared with healthy control subjects, and the severity of the condition correlates with the intensity of the psychiatric symptoms. Among samples of outpatients who consulted their general practitioners for insomnia, about 50% presented with psychiatric conditions, and about half of these patients were probably depressed (Zorick and Walsh, 2000).
- Medical consequences: Insomnia has been statistically associated with various medical conditions, including disorders of the cardiovascular, respiratory, gastrointestinal, renal and musculoskeletal systems. A large series of insomniac patients showed that poor sleepers are more than twice as much at risk of ischemic heart disease as good sleepers (Hyyppa, 1989). insomnia patients were also shown (Irwin et al.1995) to have impaired immune system function. Keith ea (2006) hypothesizes a connection between sleep deficit as one of the possible factors to explain the obesity rise. Hormone changes and animal experimets apparently support this.
- Mortality risk: Only a few epidemiological studies deal with mortality in insomniacs. According to Kripke et al. (1991), reduced sleep time is a greater mortality risk than smoking, hypertension, and cardiac disease. Increased death rates are also reported in short sleepers; in this respect, though, further systematic investigation of the link between insomnia, short sleep, and death is desirable.

3 Risk groups

Risk groups are people who may be either sensitive (showing more reaction to a stimulus than the average), or are more exposed (also called vulnerable), or both.

3.1 Health effects of disturbed sleep in children *(Bruni, Kahn)*

Although children appear to tolerate a single night of restricted sleep without a decrement in performance on brief tasks, perhaps more prolonged restriction and prolonged tasks similar to those required in school would show decrement. In addition, as children seem to require more time to recuperate fully from nocturnal sleep restriction than adults (Carskadon et al. 1981), with additional nights of partial sleep deprivation, cumulative sleepiness might become a significant problem.

Empirical data that directly addresses the effects of repeated sleep loss on children's mood or cognitive function are sparse. A range of clinical and observational data support a general picture that inadequate sleep results in tiredness, difficulties with focussed attention, low thresholds to express negative affects (irritability and easy frustration), as well as difficulty in modulating impulses and emotions. In some cases, these symptoms resemble attention deficit hyperactivity disorder.
Environmental noise experienced at home during night time is a sometimes unpredictable and most often discontinuous event (i.e., traffic noise, aircraft or train noise, noisy environment for other reasons i.e. proximity with a discotheque, etc.), that might lead to sleep disruption without leading to behavioural awakenings through the alteration of sleep microstructure, in a similar manner as other sleep disturbing events such as respiratory disturbances.
Therefore, considering clinical settings, we can assume that, in children, an experimental model of consequences of noise can be represented by the respiratory disturbances during sleep such as snoring, Upper Airway Resistance Syndrome (UARS) or Obstructive Sleep Apnea (OSA), either for the noise produced by snoring or by the effects on arousal system and sleep micro structure.
For this reason, we will describe the well-studied effects of sleep breathing disorders on children's health and then we will evaluate the indicators of sleep disruption from the point of view of sleep micro structure.
In the literature few data on medium and long term effect of disturbed sleep in children is available form the longitudinal point of view. Most reports focussed on respiratory disturbances during sleep as theoretical model to evaluate long term effect of disturbed sleep in children. In this review we will report the medium and long term negative consequences of disturbed sleep on cognitive functioning, behaviour, mental health, growth and cardiovascular system.

3.1.1 Sleep deprivation in children

The effects of sleep deprivation were evaluated in children. The findings only indirectly pertain to this general report, although repeated noise-induced sleep disruption favours sleep deprivation.

In another study, 15 healthy infants aged 78+/-7 days were studied during two nights; one night was preceded by sleep deprivation (kept awake for as long as possible beyond their habitual bedtime: median onset 150 min; range 0-210 min) (Thomas et al. 1996). Thirteen slept supine, 12 were breastfed, 4 were from smoking parents. Following sleep deprivation, infants maintained a greater proportion of quiet sleep (44 vs 39%; p=.002). There was no measurable change in arousal propensity by either graded photic (stroboscope) or auditory stimuli (1 kHz pure tone, delivered in the midline of the cot, from 73 dB and increased in 3 dB steps to 100 dB) during quiet sleep.

49 Finish children (26 boys) aged 7 to 12 years were interviewed together with their parents and school teachers, and recorded for 72 h with a belt-worn activity monitor during weekdays.
The objectively measured true sleep time was associated with teacher-reported psychiatric symptoms. The decreased amount of sleep was associated more with externalising than internalizing types of symptoms (aggressive and delinquent behavior, attention, social, and somatic problems) (Aronen et al. 2000).

In a survey, we could show that out 100 Belgian school children, 9 to 12 year old, those with poor sleep (insomnia) were also showing more frequent poor school performance (failure to comply with expected grades) than good sleepers. The relation between poor sleep and noisy environment was however not evaluated (Kahn et al. 1989).

3.1.2 Neuro cognitive manifestations

Several studies in adults have shown that sleep fragmentation and hypoxemia can result in daytime tiredness and loss of concentration, retrograde amnesia, disorientation, morning confusion, aggression, irritability, anxiety attacks and depression. One could hypothesize that sleep fragmentation and hypoxemia would affect the neuropsychological and cognitive performance also in children, where the impact of abnormal sleep may be even greater than in adults. In fact, neuro cognitive and behavioural deficits and school problems have been reported recently in children with Sleep Related Obstructive Breathing Disorders (SROBD).

3.1.3 Attentional capacity

This represents the ability to remain on task and appropriately attend to stimuli in the environment. Taken together the studies to date indicate that children with SROBD are less reflective, more impulsive, and show poorer sustained and selective attention. Blunden et al. (2000) reported that, compared to sixteen controls, sixteen children with mild SROBD showed reduced selective and sustained attention. Owens-Stively et al. (1997) suggested a dose response in attention-impulsivity with moderate-severe OSAS children showing greater deficits than mild OSAS children. Importantly, early treatment showed that attention deficits in children with OSAS are reversible (Guilleminault et al. 1982)). In another study, 12 children with moderate to severe OSAS showed significant post surgical reductions in inattention and improvement in aggressive and hyperactive behaviours and vigilance after surgical treatment (Ali et al., 1996).

3.1.4 Memory

Rhodes et al. (1995) found inverse correlations between memory and learning performance and the apnea hypopnea index in 14 morbidly obese children. Smaller deficits were observed by Blunden et al. (2000), who found in their sample of children with mild SROBD that mean global memory performance was in the lower end of the normal range compared to controls. A recent study using actigraphy in normal school-age children showed that lower sleep efficiency and longer sleep latency were associated with a higher percentage of incorrect responses in working memory tasks; shorter sleep duration was associated with performing tasks at the highest load level only. Also, controlling for age, gender, and socioeconomic status, sleep efficiency and latency were significantly associated with the mean incorrect response rate in auditory working memory tasks. This study showed that sleep quality (evaluated as sleep efficiency= 100* [sleep + light sleep]/duration) is more strongly associated with performance in working memory tasks than sleep duration, suggesting that in assessing sleep, attention should be directed not only at the amount of sleep but also at sleep quality.

3.1.5 Intelligence

Inspection of the mean IQ scores reported in the study by Rhodes et al. (1995) suggested that their sample of five obese children with moderate-severe OSAS performed in the borderline range whereas controls performed in the normal range. Blunden et al. (2000) showed smaller deficits in children with mild SROBD whose mean verbal and global IQ were in the lower end of the normal range.

It remains unclear as to whether the putative negative effects of SROBD on intelligence are global in nature or confined to specific areas such as verbal rather than performance or visuo-spatial intelligence and whether these impairments can be reversed.

3.1.6 Learning and school performance

It has been widely reported (Stradling et al., 1990; Guilleminault et al., 1996; Richards et al., 2000) that children with SROBD show reduced academic performance and learning. Weissbluth et al. (1983) found that poor academic achievers had a higher prevalence of night-time snoring (38% vs. 21%) and breathing difficulties (13% vs. 6%). Forty percent of 297 children with SROBD (22% snorers and 18% sleep-associated gas exchange abnormalities) were in the lowest 10th percentile of academic performance (Gozal, 1998) and SROBD in early childhood may continue to adversely affect learning in later years (Gozal and Pope. 2001). Gozal (1998) found in his sample of poor academic achievers that school grades improved post-adenotonsillectomy in treated but not untreated children.

Apart from SROBD, also healthy normal children with fragmented sleep (measured by actigraphy) showed lower performance on neurobehavioral functioning (NBF) measures, particularly those associated with more complex tasks and also had higher rates of behavior problems (Sadeh et al., 2002). Further, in normal children, without sleep disorders, also modest sleep restriction can affect children's NBF. Sadeh et al. (2003) monitored 77 children for 5 nights with activity monitors; on the 3rd evening, the children were asked to extend or restrict their sleep by an hour on the following 3 nights. Their NBF was reassessed on the 6th day following the experimental sleep manipulation and showed that sleep restriction led to improved sleep quality and to reduced reported alertness.

These studies suggest that fragmented sleep or insufficient sleep is highly relevant during childhood and that children are sensitive to modest alteration of their natural sleep duration.

Early reports documented that untreated OSAS can have long term negative effects such as failure to thrive, cor pulmonale, and mental retardation. These severe sequelae are less common now, due to the early diagnosis and treatment, but recent reports focused on other long term effects mainly related to neurocognitive deficits, such as poor learning, behavioral problems, and attention deficit hyperactivity disorder (Marcus, 2001).

Gozal et al. (2001) tried to determine the potential long-term impact of early childhood snoring. Analyzing questionnaires of 797 children in low academic performance group (LP) and 791 in high academic performance (HP) group, they found that frequent and loud snoring during early childhood was reported in 103 LP children (12.9%) compared with 40 HP children (5.1%). Therefore, children with lower academic performance in middle school are more likely to have snored during early childhood and to require surgery for snoring compared with better performing schoolmates. These findings suggest that children who experienced sleep-disordered breathing during a period traditionally associated with major brain growth and substantial acquisition of cognitive and intellectual capabilities may suffer from a partially irreversible compromise of their a priori potential for academic achievement.

Three major components that result from the intermittent upper airway obstruction that occurs during sleep in children could theoretically contribute to such neurocognitive deficits, namely episodic hypoxia, repeated arousal leading to sleep fragmentation and sleep deprivation, and periodic or continuous alveolar hypoventilation.

Schooling problems may underlie more extensive behavioral disturbances such as restlessness, aggressive behavior, excessive daytime sleepiness, and poor neurocognitive test performances. Nearly 20-30% of children affected by OSAS or loud and frequent snoring have important consequences on behavior with inattention and hyperactivity. Problems similar to symptoms of ADHD are linked to the presence of repeated sleep arousals, and

intermittent hypoxic events, inducing a lack of behavioral inhibition, with negative implications for working memory, motor control, and self-regulation of motivation and affect. In contrast with these data, recently Friedman et al. (2003) found a significant improvement of functions, at least in mild to moderate OSAS, when measured several months following after adenotonsillectomy, but they confirmed that their results could not rule out the possibility of partial irreversible damage to academic function even after treatment that may be detected only later in life and stated that also adults with deficits of neurocognitive executive functions related to the prefrontal area failed to improve significantly after treatment.
The negative long term effects may be mediated by the irreversible alteration of PFC and be related to structural changes of the brain as a consequence of both hypoxemia and sleep fragmentation induced by OSAS or other pathologies affecting sleep.
In a recent report Macey et al, (2002) demonstrated, in OSAS adults, gray matter loss in cerebral sites involved in motor regulation of the upper airway as well as in areas contributing to cognitive function (frontal and parietal cortex, temporal lobe, anterior cingulate, hippocampus, and cerebellum). It can be argued that, in critical stages of brain development (i.e. in childhood), such effects can determine even more severe consequences that could explain the negative long term effects.
It is speculative to think that the remodelling of brain could be also mediated by sleep and therefore sleep fragmentation could affect the process of brain plasticity (i.e. the capacity of the brain to modify its structure and function along time). Recent studies showing experience-dependent gene-expression of gene zif-268 during paradoxical sleep in rats exposed to a rich sensorimotor environment or the role of sleep for enhancing the remodelling of ocular dominance in the developing visual cortex are also in line with the hypothesis that sleep participates to neuronal plasticity and memory processes (Peigneux et al., 2001).

3.1.7 Neuro behavioural manifestations

Behavioural disturbances are common in children with SRODB with higher prevalence rates of both internalised (e.g. withdrawn, shy, anxious and psycho somatisation) and externalised (e.g. impulsivity, hyperactivity, aggression and delinquency) problematic behaviours (Blunden et al., 2001). The most frequently documented problematic behaviour in children with SROBD is inattention hyperactivity with a prevalence rate of 20-40% (Weissbluth et al., 1983; Ali et al., 1993). Conversely, children with inattention-hyperactivity showed a high prevalence rate of snoring (Chervin et al., 1997) and a co-diagnosis of Attention-Deficit Hyperactivity Disorder (ADHD) has been reported in 8-12% of children with OSAS (O' Brien and Gozal, 2002).
Few studies have documented that children with sleep disorders tend to have behavioral problems similar to those observed in children with attention deficit hyperactivity disorder. A survey of 782 children documented daytime sleepiness, hyperactivity, and aggressive behaviour in children who snored, with 27 and 38% of children at high risk for a sleep or breathing disorder displaying clinically significant levels of inattention and hyperactive behaviour, respectively (Ali et al., 1994).
At 3 years of age children with persistent sleep problems (n= 308) were more likely to have behaviour problems, especially tantrums and behavior management problems (Zuckerman et al. 1987).
In a study on16 children with a mean age of 12+/- 4 years, suffering from chronic pain due to juvenile rheumatoid arthritis and secondary poor sleep, polysomnographic recordings showed more night-time poor sleep, longer afternoon naptime and more daytime sleepiness than normal values from the literature (Zamir et al. 1998).

In a school survey, among 9 to 12 year old children (n= 1000), those with poor sleep (insomnia for more than 6 months) had poorer school performance, defined as failure to comply with expected grades, than good sleepers. Their learning problems were tentatively attributed to the long-term effect of poor sleep (Kahn et al. 1989).
A questionnaire administered on 4- to12 year-old children (n= 472) showed a relation between sleep problems and tiredness during the day (Stein et al. 2001).
In 9 to 12 year-old children (n= 77), shortening sleep by one hour was associated with reduced alertness and significant lowering of neurobehavioral functioning (Sadeh et al. 2003).
In school-age children (n= 140) recorded at home with an actigraph, a significant relation was shown between the presences of fragmented sleep, daytime sleepiness and lower performance on neurobehavioral functioning evaluated by various performance tests (Sadeh et al. 2000). These children also had higher rates of behavior problems, as reported by their parents (Sadeh et al. 2002).
In Finland, children, aged 7 to 12 years (n= 49), were interviewed together with their parents and schoolteachers and recorded for 72h with a belt-worn activity monitor during weekdays. The decreased amount of sleep was associated with symptoms, such as aggressive and delinquent behavior, attention, social, and somatic problems. The sleep findings were better associated with the teachers' than the parents reports, suggesting that parents may be unaware of their child's sleep deficiencies as the behavioral problems may be more evident at school than at home (Aronen et al. 2000).
A prospective long-term study on 2518 children conducted in Sweden, revealed that within a subgroup of 27 children with severe and chronic sleep problems, 7 children developed symptoms that met the criteria for attention-deficit/hyperactivity disorder by the age of 5.5 years (Thunström, 2002). Compared to the other children with sleep problems, these subjects had more frequent psychosocial problems in the family, bedtime struggles and long sleep latency at bedtime.
A questionnaire population-based, cross-sectional survey has been conducted in Massachusetts on 30195 children, aged 5 years (Gottlieb et al. 2003). Children described by their parents with sleep-disordered breathing (snoring, noisy breathing, apnea) were significantly more likely to have daytime sleepiness and problem behaviors, including hyperactivity, inattention and aggressiveness (all with odds ratio >2.0). These problem behaviors were suggestive of attention-deficit/hyperactivity disorder.
Similar findings were found in a group of 5 to 7 year-old children with periodic limb movement disorder were studied polygraphically and their recording compared with those of age-matched children with attention-deficit/hyperactivity disorder. Their repeated sleep fragmentation resulting from the periodic limb movement disorder favoured the development of symptoms similar to those seen in the attention-deficit/hyperactivity disorder (Crabtree et al. 2003).
The parents of 8.6-year old children (range 2 to 17 years) reported their child had difficult behaviors on the day that followed a 4-hour night-time sleep restriction (Wassmer et al. 1999). In a study, a 2-h sleep reduction induced by delayed bedtime, has been shown to increase daytime sleepiness, mainly during morning hours (Ishihara, 1998, 1999).
Following one night of 4 h of sleep deprivation imposed on children (aged 11 to 13 years), a decrease in performance tests has been observed (Carskadon et al. 1981).
Following one-night sleep loss, adolescents showed increased sleepiness, fatigue, and reaction time. They selected less difficult academic tasks during a set of tests, but the percentages of correct responses were comparable to those seen following a normal sleep night (Engle-Friedman et al. 2003).
Another study has been conducted on 82 children, 8 to 15 years of age. They were assigned

an optimised, 10-hour night of sleep, or a restricted 4-hour night of sleep. Sleep restriction was associated with shorter daytime sleep latency, increased subjective sleepiness, and increased sleepy and inattentive behaviors, but was not associated with increased hyperactive-impulsive behavior or impaired performance on tests of response inhibition and sustained attention (Fallone et al. 2001).

3.1.8 Mental health

A recent longitudinal study on the outcomes of early life sleep problems and the relation to behavior problems in early childhood stressed the importance of studying the natural history of sleep problems and their consequences to identify whether persistent or recurrent sleep problems at age 3 to 4 years are associated with comorbidities such as child behavior problems, maternal depression, and poor family functioning (Peiyoong et al., 2003).
The authors found that night waking at 3 to 4 years of age continued to be common. Seventy eight percent of mothers reported that their child awoke overnight at least once during the week, and of these waking children, 43% were reported to have awakenings 4 or more nights per week. Children with early sleep problems had significantly higher mean scores on Internalizing and Externalizing Behavior and the Aggressive Behavior and Somatic Problems subscales of the Child Behavior Checklist (CBCL).
It has been noted that within groups of children and adolescents with psychiatric, behavioral, or emotional problems, rates of sleep disorders are elevated (Sadeh et al., 1995). On the other hand, children and adolescents with disturbed sleep report more depression, anxiety, irritability, fearfulness, anger, tenseness, emotional instability, inattention and conduct problems, drug use, and alcohol use.
Only few longitudinal studies in adolescents evaluated the impact of insomnia on future functioning. In a large sample of 11-17 years old adolescents, followed for 1 year, using symptoms of DSM-IV criteria for insomnia, Roberts et al, (2002) found that nearly 18% of the youths 11-17 years of age report non restorative sleep almost every day in the past month, over 6% report difficulty in initiating sleep, over 5% waking up frequently during the night, another 3% had early-morning awakening almost every day, over 7% report daytime fatigue and 5% daytime sleepiness. Combining "often" and "almost every day" response categories dramatically increases prevalence, ranging from 60% for non restorative sleep to 23% for daytime fatigue and to 12% for waking up at night with difficulty to go back to sleep. The re-evaluation of sample at follow-up showed that insomnia predicted two indicators of psychological functioning: self-esteem and symptoms of depression (Roberts et al, 2002).

3.1.9 Growth impairment

Failure to thrive is a well known complication of disturbed sleep and childhood OSAS. The cause of poor growth is not known, although many different reasons have been implicated: a) poor caloric intake associated with adenotonsillar hypertrophy; b) excessive caloric expenditure secondary to increased work of breathing; c) abnormal growth hormone release secondary to loss of deep non-REM sleep. The relative roles of these factors are unclear (ATS, 1999, Marcus et al., 1994). Circulating concentrations of insulin-like growth factor-I (IGF-I) and IGF-binding protein 3 (IGFBP-3), reflect mean daily GH levels, and seem to correlate well with physiologic changes in GH secretion. In the operated children with initial OSAS a highly significant reduction in Apnea-Hypopnea Index (AHI) was found and both the IGF-I and the IGFBP-3 concentrations increased significantly. GH is released in a pulsatile fashion; the initial secretion is synchronized with the onset of slow-wave sleep (SWS) and strongly correlated with slow-wave activity, within 90 to 120 minutes from the onset of sleep (Nieminen et al., 2002). In OSAS children, the sleep architecture is relatively well-preserved,

but the microstructural alteration of slow-wave sleep due to microarousals induced by respiratory disturbance could play a role in the abnormal profile of GH secretion.

3.1.10 Cardiovascular complications

Children with OSAS had a significantly higher diastolic blood pressure (BP) than those with primary snoring. Multiple linear regression showed that blood pressure could be predicted by apnea index, body mass index, and age. The etiology of OSAS-related hypertension it is thought to be due to a number of factors, particularly sympathetic nervous system activation secondary to arousal, and to a lesser degree, hypoxemia. Although cortical arousals at the termination of obstructive apneas are less common in children than adults, children may manifest signs of subcortical arousal, including autonomic changes such as tachycardia. It is therefore possible that these subcortical arousals are associated with elevations of BP. A correlation between the frequency of obstructive apnea and BP, but no correlation between SaO2 and BP was found, suggesting that respiratory-related subcortical arousals rather than hypoxemia may be a major determinant of BP elevation in children (Marcus et al., 1998). Similarly to BP variations induced by OSAS, other studies suggest that chronic exposure to environmental noise during sleep could contribute to permanent increases in BP in otherwise healthy individuals and that no habituation to noise was apparent over three consecutive sleep sessions (Carter et al., 2002). This is further elaborated in Chapter IV, section 3.

3.1.11 Risk of accidents

Only one study was found that evaluated the association between sleep and wakefulness duration and childhood unintentional injury (Valent et al., 2001).
Two hundred ninety-two injured children who presented at the Children's Emergency Center of Udine, Italy, or their parents were interviewed after a structured questionnaire. Sleep or wakefulness status of the child was assessed retrospectively for each of the 48 hours before injury. For each child, the Authors compared the 24 hours immediately before the injury (hours 1-24; case period) with hours 25 to 48 (control period).
Overall, more children had longer hours of sleep during the control period than during the case period. A direct association between injury risk and sleeping <10 hours was found among boys (RR: 2.33; 95% CI: 1.07-5.09) but not among girls (RR: 1.00; 95% CI: .29-3.45). We also found a direct association between injury occurring between 4 PM and midnight, and being awake for at least 8 hours before injury occurred (both sexes, RR: 4.00; 95% CI: 1.13-14.17). Sleeping <10 hours a day was associated with an 86% increase in injury risk. A significantly increased risk did not emerge in all subgroups of patients but it was evident among children from 3 to 5 years of age, boys in particular. A fourfold increase in injury risk was also associated with being awake for at least 8 hours among males only. These findings demonstrated that inadequate sleep duration and lack of daytime naps are transient exposures that may increase the risk of injury among children. Results of a study on sleep disturbance and injury risk in young children show inadequate sleep duration and lack of daytime naps. Day-time naps are transient exposures that may increase the risk of injury among children. Among children (boys particular) from 3 to 5 years of age, sleeping less than 10 hours a day was associated with an 86% increase in injury risk. A fourfold increase in injury risk was also associated with being awake for at least 8 hours.

Daytime sleepiness in children is often manifested by externalizing behaviours noted by parents or teachers such as increased activity levels, aggression, impulsivity, as well as by poor concentration, instantiation irritability and moodiness (Fallone et al., 2002)

Analysing attendance to school, data shows that accidents took place at school (25.6%) and at home (22.0%), and there is a statistically highly significant greater total accident rate in boys than in girls. The most frequent injuries happening at school are fractures and dislocation of joints, being the head injuries more common among school injuries compared with spare-time injuries. Most injuries occurred when children are in sport areas and to be noted is that 25% of all injuries were caused by other pupils by intentional violence

3.1.12 Use of sleeping pills

Several studies demonstrated that the use of sleeping pills is common among children and that pediatricians are prone to prescribe these medications. Twenty-five percent of first-born infants had been given "sedatives" by 18 months (Ounstead, 1977). A research on parental report of 11,000 preschoolers showed that 12% took psychoactive drug, most commonly for sleep; 39% daily and 60% intermittently for 1-2 years (Kopferschmitt, 1992). Another study (Trott et al., 1995) revealed that 35% of prescriptions in children less than 1 year olds were for sleep disturbances and that sleep disturbances were also the most common reason for prescribing medications to preschoolers (23%). Two French surveys on adolescents showed that 10% to 12% of the respondents reported use of prescription or over-the-counter drugs for sleep disturbances. (Patois et al., 1993; Ledoux et al., 1994). Recently it has been reported that of 671 community-based US pediatricians 75% had recommended OTC and 50% prescription meds for insomnia during the past 6 months (Owens et al., 2003). Further, an Italian survey showed that pharmacological treatment for sleep problems was prescribed during the past 6 months by 58.54% of pediatricians and by 61.21% of child neuropsychiatrists (Bruni et al., 2004).

3.2 Basic individual factors: Gender, age(*Åkerstedt*)

Gender turns up an important predictor of disturbed sleep in virtually all epidemiological studies. (Bixler et al., 1979; Karacan et al., 1976, Ancoli-Israel, 1999 #4652; Sateia et al.. 2000; Leger Pt al., 2000). On the other hand there does not seem to be much of a difference in polysomnographical parameters between males and females, except for the former loosing SWS with increasing age and having a slightly reduced sleep efficiency also with increasing age (Williams et al., 1974) (Hume et al., 1998). Ehlers et al timed the start of differences between genders to 20-40 years (Ehlers and Kupfer, 1997). Also spectral analysis indicates slightly larger amounts of low frequency activity in females (Dijk et al., 1989a; Dijk et al., 1989b). In addition, men seem to run a higher risk for morbidity and mortality related to sleep problems than women (Nilsson et al.; 2001). The inconsistency between polysomnography and subjective measures has not been resolved but it may be important that most polysomnographical studies have controlled for anxiety and depression. Thus, it is conceivable that the higher level of subjective complaints in women reflects a higher prevalence of anxiety. The latter is a speculation, however.

A confounding factor in gender comparisons is that also phase in female biological cycles is usually controlled for in polysomnographical studies, meaning that potential effects of, for example, menstruation, may not receive its proper weight. A recent review has gone through the literature in this area (Moline et al., 2003). It found that the luteal phase of the menstrual cycle is associated with subjective sleep problems, but this has not been supported by polysomnographical studies. Pregnancy affects sleep negatively already in the first trimester and the effects mainly involve awakenings and difficulties getting back to sleep. Napping is a frequent coping method. The postpartum period is often associated with severe sleep

disruption, mainly due to feeding and comforting the infant. There seems to be some relation between sleep disruption and post-partum mood, but nothing is known about the causal relations. Menopause seems to involve disrupted sleep in relation to hot flashes, depression/anxiety and sleep disordered breathing. Estrogen is associated with improved sleep quality but it is not clear whether the effects is via a reduction of hot flashes. Estrogen also improves sleep-disordered breathing.

With respect to background factors, age is an established predictor of disturbed sleep (Bixler et al., 1979; Karacan et al., 1976; Ancoli-Israel and Roth, 1999; Ribet and Derriennic, 1999; Sateia et al., 2000; Leger et al., 2000). Interestingly, though, older age may be related to a lower risk of impaired awakening (Akerstedt et al., 2002c), that is, in this study it was easier to wake up and one felt more well rested with increasing age, while at the same time, sleep quality was lower. The increased risk of disturbed sleep is consistent with the increasingly strong interference of the circadian morning upswing of metabolism with increasing age (Dijk and Duffy, 1999). Thus sleep maintenance is impaired and when sleep is interrupted "spontaneously", the awakening is, by definition, easily accomplished and will be lacking in inertia. This ease of awakening may be interpreted as "being well rested", and obviously the need for sleep is not great enough to prevent an effortless transition into wakefulness.

In addition, sleep homeostasis seems to be weakened with age in the sense that sleep becomes more fragmented and SWS or power density in the delta bands decreases (Williams et al., 1974) (Bliwise, 1993; Dijk et al., 1999). As mentioned above, the effects are more pronounced in males, a fact may be linked to reduced levels of growth hormone and testosterone.

3.3 Persons exposed to stressors as a risk group (*Åkerstedt*)

A number of epidemiological studies point to a strong link between stress and sleep (Akerstedt, 1987; Ancoli-Israel and Roth, 1999; Urponen et al., 1988). In fact, stress is considered the primary cause of persistent psychophysiological insomnia (Morin et al., 2003). That stress can affect proper sleep seems obvious, but A. N. Vgontzas et all at Pennsylvania State University College of Medicine have found another reason why middle-aged men may be losing sleep. It's not just because of what they worry about. Rather, it's due to "increased vulnerability of sleep to stress hormones".
As men age, it appears they become more sensitive to the stimulating effects of corticotropin-releasing hormone (CRH). When both young and middle-aged men were administered CRH, the older men remained awake longer and slept less deeply. (People who don't get enough of this "slow-wave" sleep may be more prone to depression.)
The increased prevalence of insomnia in middle-age may, in fact, be the result of deteriorating sleep mechanisms associated with increased sensitivity to arousal-producing stress hormones, such as CRH and cortisol.
In another study, the researchers compared patients with insomnia to those without sleep disturbances. They found that "insomniacs with the highest degree of sleep disturbance secreted the highest amount of cortisol , particularly in the evening and nighttime hours," suggesting that chronic insomnia is a disorder of sustained hyperarousal of the body's stress response system Also, recent epidemiological studies have shown a connection between disturbed sleep and later occurrence of stress-related disorders such as cardiovascular disease (Parish and Shepard, 1990; Nilsson et al., 2001; Leineweber et al., 2003) and diabetes type II (Nilsson et al., 2002). The mechanism has not been identified but both lipid as well as glucose

metabolism ìs impaired in relation to experimentally reduced sleep (Akerstedt and Nilsson, 2003).

Burnout is another result of long term stress and a growing health problem in many western countries (Weber and Jaekel-Reinhard, 2000). In Sweden, burnout is estimated to account for most of the doubling of long-term sickness absence since the mid-nineties (RFV, 2003). The characteristic clinical symptoms of the condition are excessive and persistent fatigue, emotional distress and cognitive dysfunction (Melamed et al., 1992; Kushnir and Melamed, 1992). Self-reports of disturbed sleep are pronounced in subjects scoring high on burnout (Melamed et al., 1999; Grossi et al., In press). Since shortened and fragmented sleep is related to daytime sleepiness and impaired cognitive performance (Bonnet, 1986b; Bonnet, 1986a; Bonnet, 1985; Dinges et al., 1997; Gillberg and Akerstedt, 1998; Akerstedt, 1990), disturbed sleep might provide an important link between the state of chronic stress and the complaints of fatigue and cognitive dysfunction seen in burnout.

Partinen et al (1984) investigated several occupational groups and found disturbed sleep to be most common among manual workers and much less so among physicians or managing directors. Geroldi et al (1996) found in a retrospective study of older individuals (above the age of 75) that former white collar workers reported better sleep than blue collar workers. Kupperman et al (1995) reported less sleep problems in subjects satisfied with work.

In what seems to be the most detailed study so far, Ribet et al (1999) studied more than 21000 subjects in France, using a sleep disturbance index and logistic regression analysis. It was found that shift work, a long working week, exposure to vibrations, and "having to hurry" appeared to be the main risk factors, controlling for age and gender. Disturbed sleep was (Bixler et al., 1979; Karacan et al., 1976; Ancoli-Israel and Roth, 1999), more frequent in women, and in higher age groups.

The particular stressor linked to disturbed sleep may be working under high demands. (Urponen et al., 1988; Ancoli-Israel and Roth, 1999; Ribet and Derriennic, 1999; Akerstedt et al., 2002b)]. High demands is the classical work stress factor and when combined with low decision latitude it has been shown to be related to cardiovascular disease (Theorell et al., 1998) and absenteeism (North et al., 1996). Interestingly, when "persistent thoughts. about work" was added to the regression in the study by Akerstedt et al (2002b) this variable took over part of the role of work demands as a predictor. This suggests that it may not be work demands per se that are important, but rather their effect on post-work unwinding. In two studies it has been demonstrated that even moderate worries about being awakened during the night or of having a negative feeling about the next day will affects sleep negatively, mainly reducing SWS (Torsvall and Akerstedt, 1988; Kecklund and Akerstedt, 1997). On the other hand there is very little data to connect real life stress with polysomnograhical indicators of disturbed sleep. Most studies have used rather innocuous and artificial stressor in a lab environment. Field studies of stress are virtually lacking, with some exceptions (Hall et al., 2000).

Also lack of social support at work is a risk indicator for disturbed sleep (Akerstedt et al., 2002b). We have not been able to find much previous data of this type but poor (general) social support has been associated with sleep complaints in Vietnams veterans (Fabsitz et al., 1997). On the other hand, there are a number of studies indicating a close connection with poor social support for, for example, cardiovascular disease (Arnetz et al., 1986), or muscle

pain (Ahlberg-Hultén et al., 1995).
Interestingly, the metabolic changes seen after sleep curtailment in normals or in insomniacs and sleep apneics are similar to those seen in connection with stress. That is, lipid and glucose metabolism are increased, as are cortisol levels (Spiegel et al., 1999; Vgontzas et al., 2001; Vgontzas et al., 2000). Together with the prospective links to stress related diseases such as diabetes: type II and cardiovascular disease discussed above, and with mortality (Kripke et al., 1979; Kripke et al., 2002; Akerstedt et al., 2002a; Dew et al., 2003), the findings could suggest that disturbed sleep may be an important mediator in the development of stressrelated diseases.

3.4 Shift work as a risk factor for sleep disturbance and health effects (*Åkerstedt*)

The dominating health problem reported by shift workers is disturbed sleep and wakefulness. At least 3/4 of the shift working population is affected. (Akerstedt, 1988). When comparing individuals with a very negative attitude to shift work with those with a very positive one, the strongest, discriminator seems to be the ability to obtain sufficient quality of sleep during daytime (Axelsson et al., submitted). EEG studies of rotating shift workers and similar groups have shown that day sleep is 1-4 hours shorter than night sleep (Foret and Lantin, 1972; Foret and Benoit, 1974; Matsumoto, 1978; Tilley et al., 1981; Torsvall et al., 1989; Mitler et al., 1997). The shortening is due to the fact that sleep is terminated after only 4-6 hours without the individual being able to return to sleep. The sleep loss is primarily taken out of stage 2 sleep and stage REM sleep (dream sleep). Stages 3 and 4 ("deep" sleep) do not seem to be affected. Furthermore, the time taken to fall asleep (sleep latency) is usually shorter. Also night sleep before a morning shift is reduced but the termination is through artificial means and the awakening usually difficult and unpleasant. (Dahlgren, 1981a; Tilley et al., 1982; Akerstedt et al., 1991; Kecklund, 1996)

Interestingly, day sleep does not seem to improve much across series of night shifts (Foret and Benoit, 1978; Dahlgren, 1981b). It appears, however, that night workers sleep slightly better (longer) than rotating workers on the night shift (Kripke et al., 1971; Bryden and Holdstock, 1973; Tepas et al., 1981). The long term effects of shift work on sleep are rather poorly understood. However, Dumont (1988) found that the amount of sleep/wake and related disturbances in present day workers were positively related to their previous experience of night work. Guilleminault (1982) found an over representation of former shift workers with different clinical sleep/wake disturbances appearing at a sleep clinic. Recently, we have shown that in pairs of twins discordant on night work exposure, the exposed twin reports somewhat deteriorated sleep quality and health after retirement (Ingre and Akerstedt, in press).

The main reason for the short daytime sleep is the influence exerted by the circadian rhythm. The more sleep is postponed from the evening towards noon next day, the more truncated it becomes and when noon is reached the trend reverts (Foret and Lantin, 1972; Akerstedt and Gillberg, 1981) . Thus, sleep during the morning hours is strongly interfered with, despite the sizeable sleep loss that, logically, should enhance the ability to maintain sleep. (Czeisler et al., 1980). Also homeostatic influences control sleep. For example, the expected 4-5 hours of daytime sleep, after a night spent awake, will be reduced to 2 hours if a normal night sleep precedes it and to 3.5 hours if a 2-hour nap is allowed (Akerstedt and Gillbèrg, 1986). Thus, the time of sleep termination depends on the balance between the circadian and homeostatic influences. The circadian homeostatic regulation of sleep has also been demonstrated in great

detail in studies of forced or spontaneous desynchronization under conditions of temporal isolation and ad lib sleep hours (Czeisler et al.,. 1980; Dijk and Czeisler, 1995).

3.4.1 Alertness, performance and safety

Night oriented shift workers complain as much of fatigue and sleepiness as they do about disturbed sleep (Akerstedt, 1988). The sleepiness is particularly severe on the night shift, hardly appears at all on the afternoon shift, and is intermediate on the morning shift. The maximum is, reached towards the early morning (0500h to 0700h). Frequently, incidents of falling asleep occur during the night shift (Prokop and Prokop, 1955; Kogi and Ohta, 1975; Coleman and Dement, 1986). At least 2/3 of the respondents report that they have experienced involuntary sleep during night work.
Ambulatory EEG recordings verify that incidents of actual sleep occur during night work in, for example, process operators (Torsvall et al., 1989) . Other groups, such as train drivers or truck drivers show clear signs of falling-asleep incidents while driving at night (Caille and Bassano, 1977; Torsvall and Akerstedt, 1987; Kecklund and Akerstedt, 1993). This occurs towards the second half of the night and appears as repeated bursts of alpha and theta EEG activity; together with closed eyes and slow undulating eye movements. As a rule the bursts are short (1-15 seconds) but frequent, and seem to reflect let-downs in the effort to fend off sleep. Approximately 1/4 of the subjects recorded show the EEG/EOG patterns of fighting with sleep. This is clearly a larger proportion than what is found in the subjective reports of falling asleep episodes.

As may be expected, sleepiness on the night shift is reflected in performance. One of the classics in this area is the study by Bjerner et al (1955) who showed that errors in meter readings over a period of 20 years in a gas works had a pronounced peak on the night shift. There was also a secondary peak during the afternoons. Similarly, Brown (1949) demonstrated that telephone operators connected calls considerably slower, at night. Hildebrandt et al (1974) found that train drivers failed to operate their alerting safety device more often at night than during the day. Most other studies of performance have used laboratory type tests and demonstrated, for example, reduced reaction time or poorer mental arithmetic on the night shift (Tepas et al., 1981; Tilley et al., 1982). Flight simulation studies have furthermore shown that the ability to "fly" a simulator (Klein et al., 1970), or to carry out a performance test (Dawson and Reid, 1997) at night may decrease to a level corresponding to that after moderate alcohol consumption (>0.05% blood alcohol) Interestingly, Wilkinson et al (1989) demonstrated that reaction time performance on the night shift (nurses) was better in permanent than rotating shift workers.

If sleepiness is severe enough, interaction with the environment will cease and if this coincides with a critical need for action an accident may ensue. Such potential performance lapses due to night work sleepiness were seen in several of the train drivers discussed earlier (Torsvall and Akerstedt, 1987). The transport area is where most of the available accident data on night shift sleepiness has been obtained (Lauber and Kayten, 1988). Thus Harris (Harris, 1977) and Hamelin (1987) demonstrated that single vehicle accidents have, by far, the greatest probability of occurring at night. So does fatigue-related accidents (Home and Reyner, 1995) but also most other types of accidents, for example head-on collisions, rear end collisions (Akerstedt et al., 2001). The National Transportation Safety Board ranks fatigue as one of the major causes of heavy vehicle accidents (NTSB, 1995).

From conventional industrial operations very little relevant data is available but fatal work accidents show a higher risk in shift workers (Akerstedt et al., 20.02a) and accidents in the automotive industry may exhibit night shift effects (Smith et al., 1994) and an interesting analysis has been put forward by the Association of Professional Sleep Societies ' Committee on Catastrophies, Sleep and Public Policy (Mitler et al.; 1988). Their consensus report notes that the nuclear plant meltdown at Chernobyl occurred at 01.35h and was due to human error (apparently related to work scheduling). Similarly, the Three Mile Island reactor accident occurred between 04.00h and 06.00h and was due to, not only the stuck valve that caused a loss of coolant water but, more importantly, to the failure to recognize this event leading to the near meltdown of the reactor. Similar incidents, although with the ultimate stage being prevented, occurred 1985 at he David Beese reactor in Ohio and at the Rancho Seco reactor in California. Finally, the committee also states that the NASA Challenger space shuttle disaster stemmed from errors in judgement made in the early morning hours by people who had insufficient sleep (through partial night work) for days prior to the launch. Still, there is very limited support for the notion that shift work outside the transport area actually carries a higher over all accident risk.

As with sleep, the two main factors behind sleepiness and performance impairment are circadian and homeostatic factors. Their effects may be difficult to separate in field studies but are clearly discernible in laboratory sleep deprivation studies (Fröberg et al., 1975) as well as in studies of forced desynchronization (Dijk et al., 1992). Alertness falls rapidly after awakening but gradually levels out as wakefulness, is extended. The circadian influence appears as a sine-shaped superimposition upon this exponential fall in alertness. Space does not permit a discussion of the derivation of these functions, but the reader is referred to Folkard and Akerstedt (Folkard and Akerstedt, 1991) in which the "three-process model of alertness regulation" is described. This model has been turned into computer software for predicting alertness and performance and to some extent accident risk.

3.4.2 Health effects

Gastrointestinal complaints are more common among night-shift workers than among day workers. In a review of a number of reports covering 34047 persons with day or shift work was found ulcers occurring in 0.3%-0.7% in day workers, in 5% of persons with morning and afternoon shifts, in 2.515% of persons with rotating shift systems with night shifts, and in 10%-30% in ex-shift workers (Angersbach et al., 1980). Several other studies have come to similar conclusions (Thiis-Evensen, 1958; Segawa et al., 1987; Harrington, 1994) Other gastrointestinal disorders, including gastritis, duodenitis and dysfunction of digestion are more common in shift workers than in day workers (Koller, 1983).

The pathophysiologic mechanism underlying gastrointestinal disease in shift workers is unclear, but one possible explanation is that intestinal enzymes and intestinal mobility are not synchronized with the sleep/wake pattern. Intestinal enzymes are secreted with circadian rhythmicity, and shift , workers' intake of food is irregular compared with intestinal function (Suda and Salto, 1979; Smith et al., 1982). A high nightly intake of food may be related to increased lipid levels (Lennernás et al., 1994) and eating at the circadian low point may be associated with altered metabolic responses (Hampton et al., 1996). In addition, reduced sleep affects lipid and glucose metabolism (Spiegel et al., 1999).

A number of studies have reported a higher incidence of cardiovascular disease, especially

coronary heart disease, in male shift workers than in men who work days (for review see (Kristensen,.1989; Boggild and Knutsson, 1999). In a study of 504 paper mill workers followed for 15 years was found a dose-response relationship between years of shift work and incidence of coronary heart disease in the exposure interval 1-20 years of shift work (Knutsson et al., 1986). A study of 79000 female nurses in the United States gave similar results (Kawachi et al., 1995) as did a study with more than 1 million Danish men (Tiichsen, 1993) and a cohort of Finnish workers (Tenkanen et a1.,1997). As with gastrointestinal disease, a high prevalence of smoking among shift workers, might contribute to the increased risk of coronary heart disease, but smoking alone cannot_ explain the observed excess risk (Knutsson, 1989b). Another possibility is disturbances of metabolic parameters such as lipids and glucose for which there is some support as discussed above.

Only a few studies have addressed the issue of pregnancy outcome in shift workers. In one study of laboratory employees shift work during pregnancy was related to a significantly increased risk of miscarriage (RR 3.2) (Axelsson et al., 1984). Another study of hospital employees also demonstrated an increased risk of miscarriage (RR=1.44, 95% CI 0.83-2.51) (Axelsson and Rylander, 1989). Lower birth weight of infants of mothers who worked irregular hours has been reported (Axelsson and Rylander, 1989; Nurminen, 1989). No teratogenic risk associated with shift work was reported (Nurminen, 1989).

The mortality of shift and day workers was studied by Taylor and Pocock (Taylor and Pocock, 1972), who studied 8603 male manual worker in England and Wales between 1956 and 1968. Day, shift, and ex-shift workers were compared with national figures. SMR can be calculated from observed and expected deaths reported in the paper. SMRs for deaths from all causes were 97, 101 and 119 for day, shift, and ex-shift workers respectively. Although the figures might indicate an increasing trend, the differences were not statistically significant. However, the reported SMR close to 100 is remarkable because the reference population was the general male population. Most mortality studies concerned with occupational cohorts reveal SMRs lower than 100, implying a healthy worker's effect (Harrington, 1978). The same study showed significantly increased incidence of neoplastic disease in shift workers (SMR 116). A Danish study of 6.000 shift workers failed to demonstrate any excess mortality in shift workers (Boggild et al., 1999).

Rather little evidence exists on the connection between shift work and cancer. The mortality study by Taylor and Pocock (Taylor and Pocock, 1972) reported a increased incidence of neoplasm's in shift workers compared with the general population. A recent Danish case-control study reported an increased risk of breast cancer among 30-45-year old women who worked mainly nights (Hansen, 2001). In 75.000 nurses those with more than 15 years of night work showed an increased risk of colorectal cancer (Schernhammer et al., 2003). If the results are confirmed a possible mechanism may be the low levels of the hormone melatonin, due to light exposure during the night with a subsequent suppression of melatonin.

Very few studies are available but Koller et al (1978) found a prevalence of endocrine and metabolic disease of 3.5% in shift workers and 1.5% in day workers. Kawachi et al found in a prospective study of shift workers that the age-standardized prevalence was 5.6% at 15 years of shift work experience compared with 3.5% for no exposure (Kawachi et al., 1995). Nagaya et al found that markers of insulin resistance was more frequent in shift workers above the age of 50 than in day workers (Nagaya et al., 2002). Other indicators, such as body mass index, glucose levels, etc give a rather inconclusive impression as indicated in a review by Boggild

and Knutsson (Boggild and Knatsson, 1999).

Another contributing factor to gastrointestinal diseases might be the association between shift work and smoking. A number of studies have reported that smoking is more common among shift workers (Angersbach et al., 1980; Knutsson et al., 1988). Studies concerned with alcohol consumption comparing day workers and shift workers have produced conflicting results (Smith et -al., 1982; Knutsson, 1989a; Romon et al., 1992), probably due to local cultural habits. One study, which used g-Glutamyltransferase as marker of alcohol intake, did not indicate that the shift workers had higher intake of alcohol than the day workers (Knutsson, 1989a).

Sickness absence is often used as a measure of occupational health risks. However, sickness leave is influenced by many irrelevant factors and cannot be considered as a reliable measure of true morbidity. Studies on sickness absence in day and shift workers have revealed conflicting results and there is no evidence that shift workers have more sickness absence than day workers. For review, see (Harrington, 1978).

3.4.3 Conclusion

Shift work or similar arrangements of work hours clearly affects sleep and alertness and there is a moderate risk of cardiovascular and gastrointestinal disease. Other diseases such as cancer or diabetes may be related to shift work but the evidence is as yet rather weak.

The present review suggests that the risk of disturbed sleep increases with age but there also seems to be a recent stress related increase in sleep disturbances in young adults. The long term health consequences are not understood.

The relation between gender and disturbed sleep is confusing. Females, as a rule complain more of sleep problems, but do not exhibit any objective indications of more disturbed sleep at least not among otherwise healthy women. With increasing age the sleep of males deteriorate whereas that of women is relatively well upheld. Pregnancy, however, is a period of increased risk of disturbed sleep , whereas the menstrual cycle and menopause show less evidence of disturbed sleep. Clearly there is a great need of longitudinal research on gender and sleep and in particular on the possible health consequences around pregnancy.

Stress due to work or family seems to be one of the major causes of disturbed sleep. The effects on the risk of insomnia is well established, but reduced sleep in itself seems to yield the same physiological changes as stress. This suggests that several of the major civilization diseases in Europe and the US (diabetes, cardiovascular disease, burnout) could be mediated via disturbed sleep. This link clearly warrants longitudinal studies with interventions.

Shift workers constitute a group that suffers from disturbed sleep for most of their occupational life. The reason is the interference of work hours with the normal timing of sleep: This leads to an increased risk of accidents, directly due to excessive sleepiness, but also to cardiovascular and gastrointestinal disease, although it is not clear whether the latter effects are sleep related or due to circadian factors - or to a combination. Recent studies also suggest that breast cancer may result from shift work due to the effects of light on melatonin secretion. This still needs verification, however. Future research needs to identify countermeasures, the reasons for large individual differences in tolerance and the possible

cancerogenic and other effects.

The conclusions above should be seen against the profound effects of reduced or fragmented sleep on the neuro endocrine (including glucose and lipid regulation) and immune systems as well as the effects on mortality, diabetes and cardiovascular disease.

3.5 Conclusion

Children, elderly, pregnant women, people under stress and shiftworkers are vulnerable to (noise) disturbance of their sleep.

4 Accidents related to sleep quality *(Yovanovic)*

As already stated in 3.1.10 children with disturbed sleep present cognitive dysfunction and behavioural disturbances, abnormal growth hormone release, increase of diastolic blood pressure, increased risk of accidents and use of sleeping pills.

Regarding sleep disturbance and accidents in adults data shows that 15 - 45% of all patients suffering from sleep apnea, 12 - 30% of all patients suffering from narcolepsy and 2 - 8% of all patients suffering from insomnia have at least one accident (in a life time) related to sleepiness (statistics from the Stanford Sleep Disorders Clinic).

As already discussed in section 3.5, the biggest industrial catastrophes like the Three Mile Island, Bhopal, Chernobyl and Exxon Valdez disaster occurred during the night shift. The shift schedules, fatigue, and sleepiness were cited as major contributing factor to each incident .

The LARES (Large Analysis and Review of European housing and health Status) study is one of the few studies analysing this issue directly. The results show that the likelihood of home accidents is significantly greater when the individual is tired all the time or most the time and there is an association between sleep disturbance and accidents, with 22% of those reporting an accident also reported having their sleep disturbed during the previous 4 weeks.

The data available to document the impact of environmental noise on sleep deprivation and accidents is largely inadequate. There is no estimation of relative risk.
Further research is needed in order to identify the accident related burden of diseases attributable to noise during the night time.

5 Animal studies

As pet owners know, cats sleep (most of the time it seems) and dogs too. But do fish sleep? And flies? Yes, most animals sleep, and they even show the same phenomena as in humans; from deep sleep, dream sleep to sleep disorders. There are also many differences and weird behaviour like sleeping with only one brain half at the time (dolphins and ducks).
As Ising points out (Annex III), in animal experiments it is possible to assess the complete causal chain from noise exposure via physiological reactions and biological risk factors to morbidity or even mortality. However, a quantitative application of the results to humans is not possible. Instead, the method is useful in studying the patho-mechanisms qualitatively.
Rechtschaffen (1965) studied sleep deprivation in rats, showing that total sleep deprivation

leads to mortality in 16 to 20 days. As the animals in the last stage died from microbial infection, Everson (2000) proceeded to show early infection of lympal nodes and other tissues and hypothesised that daily sleep of some amount is necessary to maintain an intact immune system which will prevent bacterial invasion. A view that has been challenged.
Surprisingly, sleep in the common fruit fly - Drosophila melanogaster - has many similarities with mammalian sleep, including sleep-deprivation leading to impaired performance. Genetic studies in fruit flies (Cirelli, 2005) lead to mutant flies that can get by on 30% less sleep than their normal counterparts, thanks to a single mutation in one gene. While they sleep 30% less they show no immediate ill effects. The lifespan of the flies is however reduced by 30%.
These animal models certainly lead to believe that sleep is a biological necessity, and tampering with it is dangerous for survival.
As Ising shows (Annex III) noise may play a role in this. Under stressful circumstances the death rate of rats is increased when noise levels are increased from “ambient” to Leq=69 dB(A). Are noise and sleep deprivation stressors that both lead to early death? Is the noise effect due to sleep deprivation? A careful planned study may sort this out. Still remains the question in how far this is relevant to humans.

6 Conclusions

From the evidence presented so far it can de deduced that sleep is important for human functioning. Why exactly is less evident, but it is clear that disturbed sleep (either from internal factors or from external factors) leads to or is at least associated with fatigue, cognitive performance, depression, viral illness, accidents, diabetes , obesity and cardiovascular diseases. Animal experiments show that sleep deprivation shortens lifespan. That - in comparison- relatively mild effects turns up in human sleep deprivation experiments could be due to the short period (about 10 days in controlled experiments) and the limitation to young and healthy adults.
The central position of sleep in human functioning is summarized in figure 9.
In figure 10 relations with sufficient evidence are indicated with solid lines, while relations

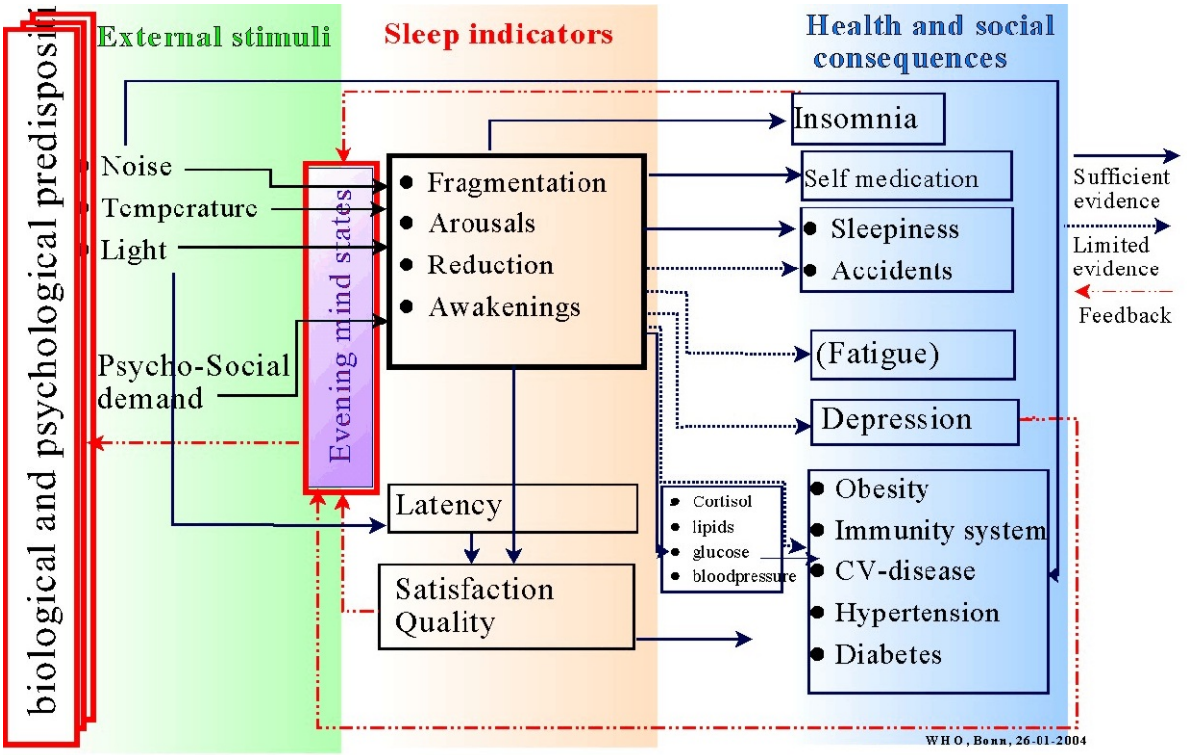


Figure 9. Expert view on the relations between sleep and health, WHO, 2004

for which limited evidence exist are indicated with interrupted lines. Feedback connections are in red and double-dotted.
The presence of feedback loops in the system is an indication that it may be difficult to prove direct cause-effect relations. An example is the relation between sleep quality and depression. They are strongly associated, but it is uncertain if depression causes bad sleep, or bad sleep causes depression (see also Ch.IV.8.11). This may also depend on one of the many other factors, so it could be different for different personality types.
Impaired sleep is widely considered as health problem per se, and in this chapter it has been shown that there are many internal and external causes. In the next chapter the relation between noise and sleep quality is further unravelled.

CHAPTER III. EFFECTS OF NIGHT TIME NOISE ON SLEEP

Best travel tip: Never ever forget to pack ear plugs.
Virginia Jealous, Lonely Planet Author

In this chapter the direct effects of noise on sleep are discussed; as well acute effects of a single exposure as the effects after a night exposure. The health consequences after prolonged exposure to noise will be the topic of chapter IV.

1 Short term effects of transportation noise on sleep with specific attention to mechanisms and possible health impact *(Miedema/Muzet)*

1.1 Introduction

In this section reactions to single events are presented. Where in chapter II normal sleep and sleep disorders are described in medical terms, here we zoom in on the mechanisms underlying the relation between noise and sleep quality. How does a sound penetrate in the brain and cause a disruption of sleep? .

1.2 How noise influences normal sleep

Noise can induce changes in the EEG or in autonomic variables that are called arousals or phasic activations. Similar brief episodes of activity also occur without noise in normal sleep, and, more frequently, in sleep that is otherwise disturbed, e.g. by apnea. Arousal during sleep is not a uniform concept and has been defined differently by different researchers. Commonly, the occurrence of alpha rhythms is required for EEG arousal. Depending on the additional requirements and on the length of time that the slower cortical rhythms are interrupted, arousals have been called e.g. micro-arousal, minor arousal, EEG awakening or transient activation phases. EEG awakening requires an interruption of the sleep patterns of at least 15 s (half the period) when sleep staging is scored by periods of 30 s, but need not be experienced consciously. Because normal REM sleep is a state characterized by cerebral arousal with frequently occurring alpha rhythms, additional criteria are needed to define arousal from REM. Criteria used are increased heart rate, EMG, or irregular respiration. However, since the mechanisms of such autonomic responses appear to be at least partly different from the causal mechanisms of EEG arousal, such definitions seem to make arousal from sleep a heterogeneous concept that may not have simple relationships with noise exposure.
EEG arousals lasting at least 30 s have been found to occur as often as 4 (95% CI: 1- 15) times per hour during sleep on average, while micro-arousals occurred 21 (95% CI: 7 – 56) times per hour (Mathur and Douglas, 1995). Since these figures are from a laboratory study, they almost certainly are higher than the figures that hold for the natural situation at home. Sleep pressure decreases the density of micro-arousals (Sforza et al., 2004). While the number of EEG arousals(during sleep stage I and II) increases with age (Mathur and Douglas, 1995; Boselli et al., 1998) possibly only for men (Hume et al., 2003), their average length is stable and circa 15 s (Boselli et al., 1998). Also the threshold for auditory arousal decreases with age (Busby and Pivik, 1983; Zepelin et al., 1984; Busby et al., 1994) and towards the end of the night (Basner et al, 2004). Recovery after EEG awakenings takes longer for noise-induced awakenings than for spontaneous awakenings (Basner et al., 2004). The time required for falling asleep again depends on the sound level and especially for loud events this latency is considerably longer than after spontaneous awakenings. Thus, in general noise induced EEG

awakenings are more disruptive than spontaneous awakenings and therefore will more often be experienced consciously and remembered afterward. In common situations with aircraft overflight noise at home, (minor)arousals were found in 10.3 % of the 64 s intervals without aircraft noise and this percentage was found to be increased by circa 4 % up to 14.3% in intervals with an aircraft noise event (Hume et al., 2003). Thus, in that particular (exposure) situation, about 1 in 24 aircraft over flights caused a(minor) arousal.

1.3 Mechanisms

Activity in the auditory system up to the brainstem nucleus inferior colliculus occurs within 10 ms after the onset of a sound. This early activity appears to be obligatory and is hardly affected by the state (sleeping or awake). Being asleep or awake does influence later activity. The auditory pathways proceed from the inferior colliculus to the thalamus, and from there to the auditory cortex. The state (sleep or awake) affects the activity in the thalamocortical circuits, which occurs after 10 – 80 ms. In particular, during slow wave sleep (SWS) the transmission of auditory information through the thalamus is suppressed. This is not the case during Rapid Eye Movement (REM) sleep or when awake.
Thus while in all (sleep) stages, sound activates the auditory system up to the inferior colliculus, the sound-induced activation of higher areas is suppressed in SWS. Therefore, further activation depending on those higher areas (e.g., extracting meaning) is not likely to occur as a primary reaction to sound during SWS. For understanding arousal during SWS, it is important that the inferior colliculus and the earlier auditory nucleus of the lateral lemniscus, and already the (dorsal and ventral) cochlear nuclei project to the reticular arousal system. Presumably, sound is always capable of arousing the sleeping subject through these connections. The ascending arousal system is heterogeneous and encompasses mono-aminergic, glutamatergic,and cholinergic nuclei that can directly or indirectly activate the thalamus and cortex. An important indirect route is the activation of the basal forebrain, which can activate the cerebral cortex through widespread, mainly cholinergic projections. The activation of the thalamus and cortex is indicated by increase in EEG rhythm frequency and reduction of the inhibition in the thalamic sensory relay nuclei. As a consequence of the latter effect, subsequent sound-induced activation may pass the thalamus and may be subject to more elaborate processing than initial sound. It can be speculated that sound in this way also reduces the threshold for somatosensory information that initiates body movements so that more body movements are observed when exposed to sound. The occurrence of habituation of cortical responses suggests an active roleplayed by a part of the brain that blocks or at least limits the impact of the activated ascending pathways.
The parasympathetic autonomic nervous system seems to be responsible for the bradycardia observed in Non-REM sleep and mainly in tonic REM sleep through the increase in vagal activity(Guazzi et al., 1968). The variability of heart rate in REM sleep could be placed under the same control as vagotomy strongly reduced the heart rate instability (Baust et al., 1969). During falling asleep, respiration is unstable and alternates hypo and hyper ventilation episodes. This respiration, called “periodic respiration” (Mosso, 1886), disappears when stable sleep occur(stage 2). The main hypotheses concerning this periodic ventilation refer to metabolic control and chemoreceptor responses to levels of $PaCO_2$ and PaO_2 (Chapman et al., 1988). In stable Non-REM sleep, respiration is regular in amplitude and frequency, although ventilation per minute is lower than during awakening. In REM sleep, respiration appears irregular with sudden variations in amplitude and frequency. This irregularity appears to be not modifiable by metabolic factors and, therefore, it is possibly linked to mechanisms leading to REM expression. The non habituation of the cardiovascular responses would be

explained by the absence of an inhibitory influence on the part of the arousal system that affects the centres regulating the autonomous response.

1.4 EEG response

The sleep polygraph continuously records electroencephalogram (EEG) activity, eye movement (EOG) and muscle tone (EMG). These data are used to classify sleep into various stages, and to assess time of falling asleep and wake-up time. Also sleep variables such as total sleep time and total time spent in Slow Wave Sleep (SWS, consisting of sleep stages III and IV, the stages of deep sleep) and in the stage of Rapid Eye Movement (REM, also called dream or paradoxical sleep) can be assessed on the basis of sleep polygraph recordings. Polygraphic indicators of responses to individual noise events are changes from a deeper to a less deep sleep, and EEG awakening. Several field studies (Basner et al., 2004; Hume et al., 2003; Pearsons et al 1973; Vallet et al.,1983; Vernet, 1979) have been conducted regarding noise-induced changes in sleep stage and awakening using EEG recordings. Transition from a deep stage of sleep to shallower sleep stage can be the direct consequence of a nocturnal noise event. Although not perceived by the sleeper, these transitions modify the sleep architecture and may reduce the amount of SWS (Basner et al.,2004; Carter, 1996) and the amount and rhythmicity of REM sleep may be markedly affected (Naitoh et al., 1975; Thiessen, 1988). In addition to their results from a laboratory study, Basner et al. (2004) present results from a field study with valid data for 63 subjects (age: 18 – 65 years) with 15 556 aircraft noise events included in the final analyses. They established a curve that gives the probability of awakening as a function of L_{Amax} with a model that assumed a background noise level just prior to the aircraft noise event of 27 dB(A). The L_{Amax} threshold for noise induced awakenings was found to be about 35 dB(A). Above this threshold the probability of noise induced awakenings increase monotonically up to circa 10 % when L_{Amax} = 73 dB(A). This is the extra probability of awakening associated with the aircraft noise event, on top of the probability of awakening spontaneously in a 90 s interval.
Some arousals provoked by noise events are so intense that they induce awakening. Frequent awakening leads to sleep fragmentation and global sleep disturbance. The noise threshold for awakening is particularly high in deep slow wave sleep (stages 3 and 4) while it is much lower in shallower sleep stages (stages 1 and 2). In REM sleep the awakening threshold is variable and depending on the stimulus significance. Total sleep time can be reduced by both longer time to fall asleep and premature final awakening. It has been reported that intermittent noises with maximum noise levels of 45 dB(A) and above, can increase the time to fall asleep by a few to 20 minutes (Öhrström, 1993). In the morning hours, the sleeper can be more easily awakened by ambient noise and has more difficulty to go back to sleep because sleep pressure is progressively reduced with time (Keefe et al., 1971; Rechtschaffen et al., 1966). Terzano et al. (1990, 1993) showed that with increasing intensity of sound pressure level (white noise at 45, 55, 65 and 75 dBA), white noise induced a remarkable enhancement of cyclic alternating patterns (CAP)/NREM, characterized by a linear trend from the lowest to the highest intensities, revealed by a significant increase in the CAP rate already at 45 dB(A). Noise decreased mainly SWS, REM and total sleep time, and increased wake after sleep onset, Stage 1 NREM and CAP rate (Terzano et al., 1993). For CAP/NREM values between 45% and 60%, subjects generally recalled a moderate nocturnal discomfort and values of CAP/NREM over 60% corresponded to a severe complaint.
This result corroborates previous findings described by Lukas (1972) who reported that reactions less intense than a sleep stage change correlate better to the noise intensity than awakening reactions.

1.5 Cardiovascular response

For sleeping persons mean heart rate, mean systolic and diastolic blood pressure, and variability in heart rate are usually assessed. Indicators of responses to individual noise events are instantaneous changes in (variability of) heart rate and changes in systolic blood pressure. Several field studies (Carter et al., 1994; Hoffman et al., 1995) have been conducted regarding momentary change in heart rate. Intermittent noise during sleep has been found to induce a biphasic cardiac response and a transient constriction of peripheral vessels together with a short phasic activation in the EEG, while no other behavioural effect can be seen (Muzet and Ehrhart, 1978). This biphasic cardiac response starts with an increase in heart rate, probably due to a phasic inhibition of the parasympathetic cardio-inhibitory centre, followed by a compensatory decrease due to a phasic decrease in ortho-sympathetic activity (Keefe et al., 1971; Muzet and Ehrhart, 1980). The vasoconstrictive response was reported to be due to the sympathetic peripheral stimulation provoked by the auditory reflex (Kryter and Poza, 1980). More recently, it has been shown that beat by beat blood pressure changes can be induced by suddenly occurring noises (Carter et al., 2002). Although habituation in some effect parameters can occur in a few days or weeks, this habituation is not complete and the measured modifications of the cardiovascular functions remain unchanged over long periods of exposure time (Muzet and Ehrhart, 1980; Vallet et al., 1983). Most striking is that none of the cardiovascular responses show habituation to noise after a prolonged exposure, while subjective habituation occurs within a few days. In people that are used to sleep in a noisy surrounding, noise-induced changes in heart rate are dependent on the maximum sound level of a noise event, but not on the EEG sleep stage (Hofmann et al., 1995).

1.6 Body movement

Motility is the term used for accelerations of the body or body parts during movement. It is measured with actimeters, usually worn on the wrist in field research and the laboratory. In (Brink, 2006) a more sophisticated method is described which is based on the bed being placed on accelerometers. This allows to track whole body movements.
Motility is related to many variables of sleep and health (Reyner, 1995; Reyner and Horne, 1995; Patterson et al., 1993, Passchier-Vermeer et al., 2002). Clinical research shows that the sleepwake cycle (assessed by polysomnography EEG, EOG, EMG) passes through the 24-hour period synchronously with the rest-activity cycle (assessed by actimetry) (Borbely et al., 1981; Cole et al., 1992, 1995). A number of investigations have compared the results of polysomnographic recordings (number of EEG-awakenings during sleep period, duration of sleep period, sleep onset time, wake-up time) with results of actimetry. The correlation between actimetrically assessed duration of sleep period, sleep onset time, wake-up time and similar variables assessed with polysomnography were found to be very high (correlation coefficients between individual test results in the order of 0.8 to 0.9).
Measures of instantaneous motility are the probability of motility and the probability of onset of motility in a fixed time interval, e.g. a 15, 30, or 60 s interval. Increased instantaneous motility during sleep is considered to be a sensitive behavioural marker of arousal, but the relation with arousal is not simple. Also other factors, such as the need to relieve the pressure on body parts for better blood circulation, cause motility, and spontaneously occurring arousals are part of the normal sleep process. The noise-induced probability of (onset of) motility is the difference between the probability of (onset of) motility during noise events minus the probability in the absence of noise.

(Onset of) motility and (minor) arousal found on the basis of EEG recordings are highly correlated. In the UK sleep disturbance study, Ollerhead et al. (1992) found for their study population that during sleep there is on average an EEG (minor) arousal in 40% of the 30-s intervals with onset of motility. Unfortunately, it is unknown whether this 40% is also valid for noise-induced awakenings. In 12% of the 30-s intervals with an EEG (minor) arousal, motility does not occur. Several field studies (Fidell et al., 1998, 2000; Flindell et al. 2000; Griefahn et al., 2000; Horne et al., 1995; Passchier-Vermeer et al 2002, 2004) have been conducted regarding noise-induced instantaneous motility. For this effect, relationships have been established with SEL or L_{Amax} , for aircraft noise only. In Passchier-Vermeer et al. (2002) relationships between noise-induced increase in motility (*m*) or noise-induced increase in onset of motility (*k*) in the 15- s interval with the maximum noise level of an overflight, and L_{Amax} or SEL have been approximated by quadratic functions (see, e.g., figure 11). It may be noted that the threshold of motility (L_{Amax} = 32 dB(A)) is in the same range as the threshold found by Basner et al (2004) for EEG awakenings, with a definition that also encompassed transitions to steep stage I (L_{Amax} = 35 dB(A)). The probability of motility at 70 dB(A) of about 0.07 is lower than the probability of noise-induced EEG awakening at L_{Amax} = 73 dB(A) of about 0.10. There is no a priori reason to expect the above threshold probabilities to be the same for these two measures of sleep disturbance, but, taking into account that motility is assessed for shorter intervals (15 s versus 90 s), the differences in probabilities above threshold appear to be limited.
One of the variables influencing the relationships between noise-induced instantaneous motility and L_{Amax} or SEL, is the long-term aircraft noise exposure during sleep. The probability of instantaneous aircraft noise-induced motility is lower when the long-term exposure is higher. This may be partly due to the higher base rate motility in quiet intervals in higher long-term exposure, which is used as a reference for the instantaneous noise-induced motility. Other factors influencing the relationships between instantaneous motility and L_{Amax} or SEL, are the point of time in the night, and time since sleep onset. E.g., after 7 hours of sleep, noise-induced motility is about 1.3 larger than in the first hour of sleep. Age has only a slight effect on noise-induced motility, with younger and older people showing a lower motility response than persons in the age range of 40 to 50 years.

1.7 Behavioural awakening in adults

In Passchier-Vermeer (2003a) a review is published of 9 studies on awakening by noise. It was found that these studies had different definitions of what constituted an “awakening”. In this review, however, all awakening data were collected on *behavioural awakening*: these are awakenings that were followed by an action (like pressing a button) from the sleeper. The number of awakenings defined in this manner is much smaller than the number of sleep stage changes which lead to EEG-patterns similar to wakefulness.
Data were available for rail traffic noise, ambient (probably road) noise, civil aviation noise and military aviation noise.

The rail traffic noise study is very small (only 20 subject nights), but showed no awakenings. The study states that "*there is some evidence, be it very limited, that railway noise events, in the range of SEL_i considered (up to 80 dB(A)), do not increase (the) probability of awakening*".

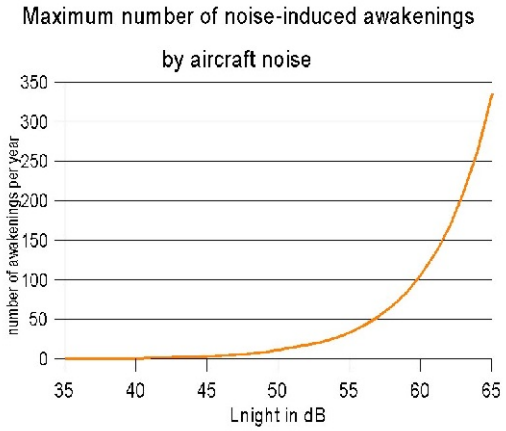


Figure 10. Worst case prediction of noise induced behavioural awakenings.L_{night}, converted from inside relation with [3]

Ambient noise also showed no effect on the probability of awakening, but as it is uncertain exactly what noise is meant, no firm conclusions could be drawn.
Military aircraft noise showed a very strong effect, but this study is of limited applicability since the few subjects (military) lived near the end of the runway.
For civil aviation noise there were sufficient data to derive a dose-effect relation:

$$\text{percentage of noise-induced awakenings} = -0.564 + 1.909*10^{-4}*(SEL_{inside})^2 \quad [4]$$

where SEL_{inside} is the Sound Exposure Level of an aircraft noise event in the bedroom.
This relation is confined to commercial aircraft noise over the intervals $54<SEL<90$ (ca $37<L_{Amax}<82$) and the number of events/night $1< N< 10$.
With this relation, it is possible to calculate for an individual L_{night} the expected number of noise-induced behavioural awakenings. This requires all single contributions over the year to this L_{night} to be known. Alternatively (if, for instance a future situation has to be estimated for which no exact data are available) a worst case scenario can be calculated. Figure 10 represents the results of this worst case approach (converted to L_{night}, see section I.3.4), and so gives the maximum number of awakenings n_{max} that may be expected.

$$n_{max} = 0.3504*10^{(Lnight-35.2)/10} \quad [5]$$

It can be demonstrated that the number of awakenings reaches a maximum when the $SEL_{,inside}$ value is 58.8 dB(A).
It should be noted that, on average, 600 spontaneous awakenings per person are reported per year. This also explains why so many more awakenings are reported than can be attributed directly to aircraft noise. At 55 L_{night}, nearly 100 overflights per night with $SEL_{,inside}$=58.8, or 1 per 5 minutes are possible. It is therefore very likely that an overflight coincides with a spontaneous awakening.

1.8 Dose-effect relations for body movements during sleep

In Passchier-Vermeer et al. (2002) motility is registered in 15-second intervals. A distinction is made between 2 variables:
- the *presence of motility* in the interval (indicated by *m*) and
- the *onset of motility*,
  meaning the presence of motility when there was no motility in the preceding interval

(indicated by *k*).

Relations between a noise-induced increase in motility (*m*) or a noise-induced increase in the onset of motility (*k*) in the 15-s interval with the maximum sound level of an overflight, and $L_{Amax,inside}$ or $SEL_{,inside}$ have been approximated by quadratic functions with the following format:

$$m = b*(L_{Amax,inside} - a) + c*(L_{Amax,inside} - a) \quad [6]$$

The coefficients a, b and c are given in Table III.1.1. The value of a is the value below which *m* or *k* is zero. Figure 11 shows the relationship between *m* and $L_{Amax,inside}$ together with the 95% confidence interval. Relations apply to $L_{Amax,inside}$ and $SEL_{,inside}$ values of at most 70 and 80 dB(A), respectively.

Table III.1.1: Coefficients of the quadratic equation (formula [6]) of *m* and *k* as a function of $L_{Amax,inside}$ or $SEL_{,inside}$ for the 15-s interval in which an indoor maximum sound level of an aircraft noise event occurs. The equations are applicable in the $L_{Amax,inside}$ range from 'a' up to 70 dB(A), or $SEL_{,inside}$ range from 'a' up to 80 dB(A). Below 'a', *m* and *k* are zero.

	(Aircraft) noise-induced increase of probability of motility (m)	(Aircraft) noise-induced increase of probability of onset of motility (k)
range	$32 < L_{Amax,inside} < 70$ dB(A) *(see figure 11)*	$32 < L_{Amax,inside} < 70$ dB(A)
a	32	32
b	633	415
c	$3.14x10^{-5}$	$8.84x10^{-6}$
range	$38 < SEL_{,inside} < 80$ dB(A)	$40 < SEL_{inside} < 80$ dB(A)
a	38	40
b	532	273
c	$2.68x10^{-5}$	$3.57x10^{-6}$

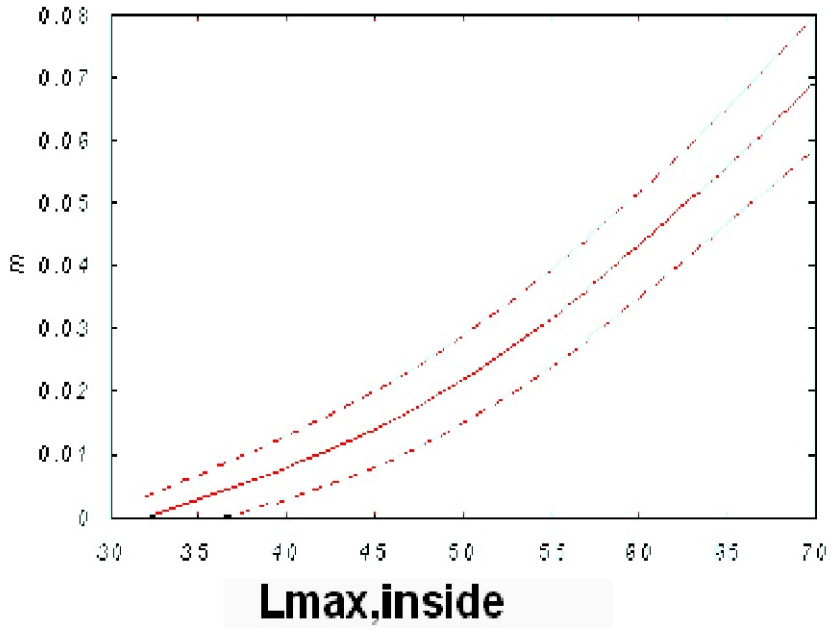


Figure 11. Probability of (aircraft) noise-induced motility (m) at the 15-s interval in which the indoor maximum sound level occurs (solid line) and the 95% confidence interval, as a function of $L_{Amax,inside}$ bedroom (Passchier-Vermeer et al[18]., 2002).

The study report also gives the upper boundaries for motility, based on the relationship between L_{Amax}, SEL and L_{night}(figure 12). This figure is mathematically derived from relation [6] as described in Annex I.

This area of study is still under development. In the study report (Miedema, H.M.E,2002) a detailed account is given of the relation between the study used for the data presented here (Passchier-Vermeer 2002), earlier studies like the much quoted CAA-study (Ollerhead et al., 1992, 1994) and earlier work done in the US.

1.9 Individual sensitivity

Sensitivity to noise may vary greatly from one individual to another. Primary self-evaluation of sensitivity to noise has been used as a factor to evaluate highly sensitive and non sensitive

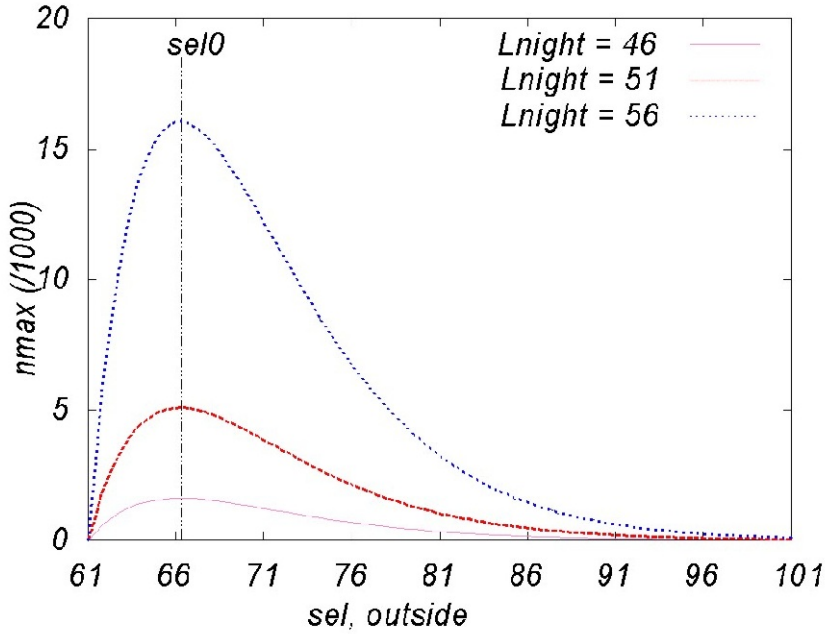


Figure 12. Maximum number of noise induced motility for 3 values of L_{nighr} Converted from inside relation with [3]

groups and to compare their reactions to noise exposure during daytime and night time (Di Nisi et al., 1990). In this study, self-declared highly sensitive individuals had higher cardiovascular response rate to noise than non sensitive people during their waking exposure, while there was no difference in sensitivity to noise between these two groups during their night time exposure while they were asleep.

The physiological sensitivity to noise depends also on the age of the sleeper. Thus, while EEG modifications and awakening thresholds are, on the average, 10 dB(A) higher in children than in adults, their cardiovascular sensitivity to noise is similar, if not higher, than the older group ([Muzet et al., 1981; see also Appendix 4). Elderly people complain much more than younger adults about environmental noise. However, their spontaneous awakenings occurring during the night sleep are also much more numerous. Therefore, it is difficult to conclude if elderly people are more sensitive to noise or if they hear noise because they are often awake during the night. This natural fragmentation of their night sleep tends also to lengthen their return to the sleeping state and this account for a significant part in their subjective complaints.

Differences in sensitivity to noise have been found between both sexes. Thus, young men seem to complain more about noise-disturbed sleep than young females (Muzet et al., 1973). However, this difference seems to reverse for populations over 30 years of age and then

females (often mothers) appear to be more sensitive to noise than males (Lukas, 1972).

1.10 Use of instantaneous effects in predictions over a longer time period

It is tempting to use the relations between single exposures and measured effects in long term predictions. Although this is perhaps possible, a word of caution is appropriate.
In general, the reactions are calculated by looking at a certain time frame around an exposure, usually in the order of a few minutes. The second limitation is that order and follow up effects are neglected. Time and order effects of identical events on motility have been described by (Brink, M., Wirth, K., Schierz, C. , 2006). Effects of early morning aircraft overflights on sleep and implications for policy making. Euronoise 2006, Tampere, Finland]. Only if the situation that is modelled resembles the one that was used in the single exposure analysis, no major deviations are to be expected. Reactions to noise events are generally not independent from each other. Each event may alter a subjects tendency to awake at the next event, even if no awakening reaction is detected for that particular event. If e.g. each event would additionally increase the probability of awakening at the next event, the total probability of awakening per night would be greater than predicted by mere summation of the single event probabilities. Most likely, this underestimation of probability will occur when events in the real situation follow in close succession, whereas events in the single exposure analysis did not. Such limitations can to some degree be overcome through applying advanced statistical methods such as put forward by Basner, M. (2006).
A third limitation is that an overall increase in the base line could go undetected.
If the situation that is calculated resembles the one that was used in the single exposure analysis, probably no major deviations are to be expected. Care should be taken to extrapolate outside the boundaries given in number of events or L_{Amax} . Calculations for Amsterdam Airport show a good agreement between the number of calculated awakenings per year (based on the actual SEL-data) and the self -reported number of awakenings. This number is a factor 2 lower than the worst case scenario presented in 1.7.

2 Chronic effects: Chronic increase of motility

Mean motility - all body movements counted together - during sleep is strongly related to age and is also a function of noise exposure during the sleep period. The relationships between mean motility and $L_{night,\ inside}$ are shown in figure 13. Mean motility during sleep is lowest at the age of 45 years, and greater higher and lower ages. The relation between mean motility, and $L_{night,inside}$ and age is:

$$Mean\ motility = 0.0587 + 0.000192*L_{night,inside} - 0.00133*age + 0.0000148*age^2 \quad [7]$$

The relation between the increase in mean noise-induced motility, m_{night}, and $L_{night,inside}$ is:

$$m_{night} = 0.000192*L_{night,inside}, \quad [8]$$

assuming, as described in section 3.4, that $L_{night,inside}=L_{night} - 21$:

$$m_{night} = 0.000192*L_{night} - 0.004032 \quad [8a]$$

The increase in m_{night} is 22% over the base line motility (0,03 on the average) if indoor $L_{night,inside}$ increases from 0 (absence of aircraft noise) to 35 dB(A) (living close to a runway).

This increase is independent of age, although the absolute level varies.

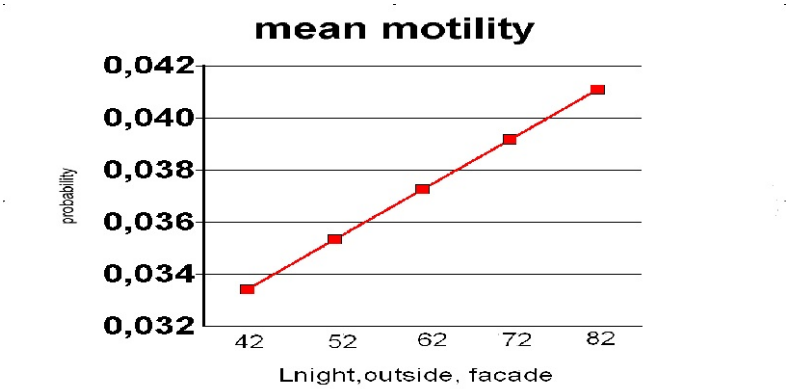


Figure 13 Increase in mean motility (body movements during sleep). Converted from inside relation with [3]

Other chronic effects like the use of sleeping pills, changes in blood pressure and changes in levels of stress hormones are discussed in the next chapter

3 Conclusions

During sleep the auditory system remains fully functional. Incoming sounds are processed and evaluated and although physiological changes continue to take place, sleep itself is protected because awakening is a relative rare occurrence. Adaptation to a new noise or to a new sleeping environment (for instance in a sleep laboratory) is rapid, demonstrating this active protection. The physiological reactions do not adapt, as is shown by the heart rate reaction and the increase of average motility with sound level. The autonomous physiological reactions are a normal reaction to these stimuli, but the question is if prolonged “abuse”of this system leads to adverse consequences for the organism. The next chapter tries to answer that.

CHAPTER IV. EFFECTS OF NIGHT TIME NOISE ON HEALTH AND WELL BEING

The sick die here because they can't sleep,(...)
For when does sleep come in rented rooms?
It costs a lot merely to sleep in this city!
That's why everyone's sick: carts clattering
Through the winding streets, curses hurled
At some herd standing still in the middle of the road,
Could rob Claudius or a seal of their sleep!
Juvenalis, 1st century AD

1 Introduction

In the chapters II and III sufficient evidence was presented to support the hypothesis for the simplified model presented in chapter I: sleep disturbance is connected to health impairment, and noise is an important factor that causes sleep disturbance. The ful model (figure 9, chapter II) showed why it is difficult to find evidence for a direct relation between noise exposure at night and health outcomes. Noise is but one of the internal and external factors that cause sleep disturbance and feedback loops obscure the view on the cause and effect chain. In this chapter the evidence for the direct relation is presented.

2 Self-reported (chronic) sleep disturbances

Self-reported sleep disturbance is investigated by means of a questionnaire containing questions regarding sleep disturbance. Often sleep disturbance is not the main focus of the questionnaires used in studies of self-reported noise effects. This means that considerable effort is needed to harmonise the different response categories. The relationships for self-reported sleep disturbance are based on analyses of the 15 data sets with more than 12000 individual observations of exposure-response combinations, from 12 field studies (Miedema, 2003; Miedema, 2004).
The curves are based on data in the L_{night} (outside, most exposed facade) range 45-65 dB(A). The polynomial functions are close approximations of the curves in this range and their extrapolations to lower exposure (40-45 dB(A)) and higher exposure (65-70 dB(A)). The formulae of these polynomial approximations for **road traffic** are as follows:

$$\%HSD = 20.8 - 1.05*L_{night} + 0.01486*(L_{night})^2 \quad [9]$$

$$\%SD = 13.8 - 0.85*L_{night} + 0.01670*(L_{night})^2 \quad [10]$$

$$\%LSD = -8.4 + 0.16*L_{night} + 0.01081*(L_{night})^2 \quad [11]$$

in which SD=Sleep Disturbed; H=Highly; L=Lowly

for **aircraft**:

$$\%HSD = 18.147 - 0.956*L_{night} + 0.01482*(L_{night})^2 \quad [12]$$

$$\%SD = 13.714 - 0.807*L_{night} + 0.01555*(L_{night})^2 \quad [13]$$

$$\%LSD = 4.465 - 0.411*L_{night} + 0.01395*(L_{night})^2 \quad [14]$$

and for **railways**:

$$\%HSD = 11.3 - 0.55*L_{night} + 0.00759*(L_{night})^2 \quad [15]$$

$$\%SD = 12.5 - 0.66*L_{night} + 0.01121*(L_{night})^2 \quad [16]$$

$$\%LSD = 4.7 - 0.31*L_{night} + 0.01125*(L_{night})^2 \quad [17]$$

in which again SD=Sleep Disturbed; H=Highly; L=Lowly

The above relations represent the current best estimates of the influences of L_{night} on self-reported sleep disturbance for road traffic noise and for railway noise, when no other factors are taken into account. Figure 14 illustrates the relations [9] [12] and [15] for persons highly disturbed by road, aircraft and rail noise.

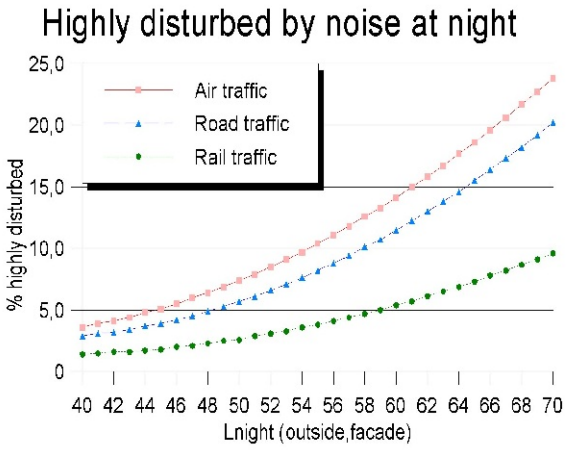


Figure 14. Highly sleep disturbed by noise at night. From EC, 2004.

With regard to the relations for aircraft noise it should be noted that the variance in the responses is large compared to the variance found for rail and road traffic. This means that the uncertainty regarding the responses for night-time aircraft noise is large, and such responses can be considered as indicative only. In Miedema, 2004 the following causes are suggested:
- The time pattern of noise exposures around different airports varies considerably due to specific night-time regulations;
- the sleep disturbance questions for aircraft noise show a large variation;
- the most recent studies show the highest self-reported sleep disturbance at the same L_{night}, level. This suggests a time trend.

For industrial noise there is an almost complete lack of information, although there are some indications(Vos, 2003) that impulse noise may cause considerable disturbance at night.

3 Complaints

According to the Health Council of the Netherlands (2004), the submission of a complaint about noise is symptomatic of reduced well-being.
Complaints about noise are widespread, and night noise seems to cause more complaints than day time noise at the same level. In (Hume, 2003b) it was found that around Manchester airport complaints per 1000 aircraft traffic movements rose from an average of 10 in daytime hours to up to 80 in the night. When linking part of the complaints to measured noise levels, an increase from an average of 1 complaint at 70 PNLdB (ca 58 L_{Amax}) to 2 at 114 PNLdB (ca 102 L_{Amax}) was found.
Due to differences in complaint cultures and registration practices, it is difficult to make comparisons between complaint registrations. Around Amsterdam Airport a relation between complaints and L_{Aeq} was found (Ministerie Verkeer en Waterstaat, 2005). The threshold for complaints is around 45 Lden, and increases to 7% of the population at 72 Lden. Night time

complaints follow the same pattern, and the threshold for night complaints is 35 L_{night}. In figure 15 the mean percentage shows a definite relationship with L_{night}. The 95-percentile indicates that the threshold is 35 L_{night}.

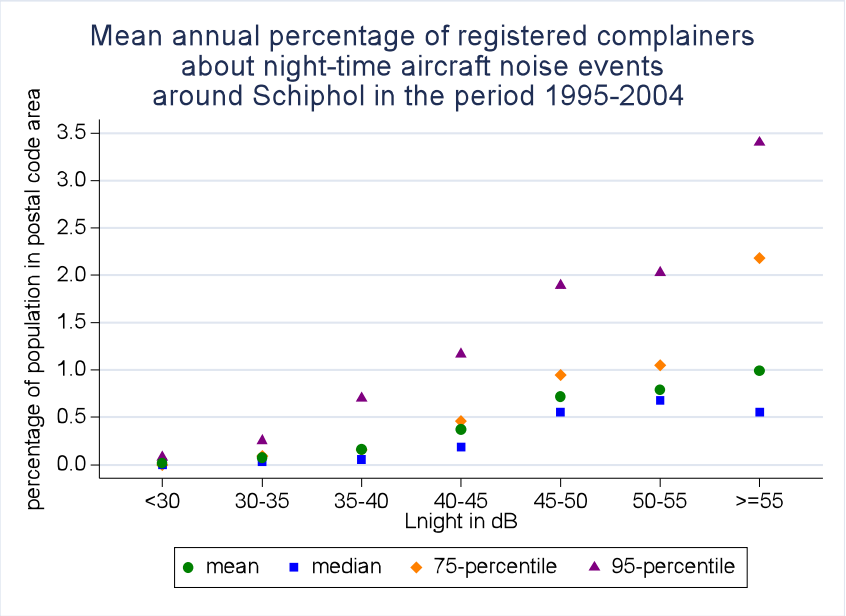


Figure 15 Dose-effect relation for persons having complained at least once during a year between 1994 and 2004. Courtesy RIVM, based on Houthuijs, 2006.

4 Neighbourhood noise and noise from neighbours (*Health Council of the Netherlands*)

Inventory studies in the Netherlands indicates that sleep disturbance attributable to the most annoying forms of neighbourhood noise and noise from neighbours (contact noise and human noises in the environment) is on a similar scale to disturbance attributable to the most annoying sources of road traffic noise (mopeds and passenger cars). It is reasonable to assume that chronic sleep disturbance is in the long term liable to have consequences for health and well-being. The sound pressure level and other noise characteristics are liable to determine the nature of the influence to some extent, but certain other factors play a more prominent role than is the case with traffic noise. These factors include appreciation of the noise and of the party responsible for the noise, as well as the hearer's personal circumstances. However, scientific understanding of the relative importance of and interaction between acoustic and non-acoustic factors is not sufficient for the Committee to draw any definitive conclusions regarding the relationship between, on the one hand, exposure to night-time neighbourhood noise and noise from neighbours and, on the other, health and well-being.

Leidelmeier and Marsman (1997) carried out an interview-based study of 1242 households in the Netherlands, in which subjects were asked about daytime and night-time noise from neighbours and any associated annoyance. Distinction was made on the basis of the part of the house in which the noises were audible and any associated annoyance was experienced. Subjects proved least tolerant of noise from their neighbours that was audible in the master bedroom. The researchers distinguished five types of noise, which are listed below, along with the percentage of subjects who indicated hearing the relevant type of

noise from a neighbouring dwelling at night in the master bedroom:

Type of noise	Subjects hearing at night in bedroom
Contact noise	22 %
Noise from sanitary fittings central heating etc	19 %
Noise from radio TV and hi-fi	12 %
Do-it-yourself noises	8 %
Pets	6 %

Where each of the five investigated types of noise were concerned, roughly 10 to 15 per cent of subjects indicated that they felt it was unacceptable for the noise to be audible during the day. Overall, nearly 30 per cent of subjects said that sanitary fittings should not be audible at night, while approximately 50 per cent felt each of the other four types of noise were unacceptable by night.

In 1993, Kranendonk et al produced a synthesis of the research conducted up to that point in time into the annoyance associated with noise from neighbours. Subsequently, in 1998, Van Dongen et al published a report on the relationship between noise from neighbouring dwellings and the airborne and contact noise attenuating indices Ilu, Ilu;k, and Ico, drawing on data from a questionnaire-based survey of the residents of six hundred dwellings, whose acoustic quality was determined in 202 cases. The results of the two studies are reasonably consistent. Both found that the chief causes of annoyance were loud radios, hi-fi's and TVs, audible and sometimes intelligible voices, the slamming of doors and footsteps on floors and staircases. In both cases, it proved that, when Ilu had a value of 0 (the minimum requirement for new homes), 10 per cent of subjects reported high annoyance and 15 per cent reported annoyance caused by noise from neighbouring dwellings. These figures are not specific to night-time noise, but apply to annoyance over a twenty-four-hour period.

On the basis of the findings outlined above, the Committee concludes that the standard of inter-dwelling sound attenuation presently required does not provide sufficient protection to prevent annoyance caused by noise from neighbours. Since people are less tolerant of the noise their neighbours make at night-time than of their neighbours' evening or daytime noise, it may be assumed that much of the annoyance associated with noise from neighbours relates to the influence of such noise on sleep

5 Cardiovascular Effects of Noise - Findings from Epidemiological Studies [*Babisch*]

5.1 Introduction

It is a common experience that noise is unpleasant and affects the quality of life. It disturbs and interferes with activities of the individual including concentration, communication, relaxation and sleep (WHO, 2000; WHO Regional Office for Europe, 2000; Schwela, 2000). Besides the psychosocial effects of community noise, there is concern about the impact of noise on public health, particularly regarding cardiovascular outcomes (Suter, 1992; Passchier-Vermeer and Passchier, 2000; Stansfeld et al., 2000). Non-auditory health effects of noise have been studied in humans for a couple of decades using laboratory and empirical methods. Biological reaction models have been derived, which are based on the general stress concept (Selye, 1956; Henry and Stephens, 1977; Ising et al., 1980; Lercher, 1996). Amongst

other non-auditory health endpoints, short-term changes in circulation including blood pressure, heart rate, cardiac output and vasoconstriction as well as stress hormones (epinephrine, norepinephrine and corticosteroids) have been studied in experimental settings for many years (Berglund and Lindvall, 1995; Babisch, 2003). Classical biological risk factors have been shown to be elevated in subjects that were exposed to high levels of traffic noise (Manninen and Aro, 1979; Lercher and Kofler, 1993; Algers et al., 1978; Arguelles et al., 1970; Dugué et al., 1994; Marth et al., 1988; Rai et al., 1981; Schulte and Otten, 1993a; Verdun di Cantogno et al., 1976; Yoshida et al., 1997; Knipschild and Sallé, 1979; Eiff et al., 1981a; Goto and Kaneko, 2002; Eiff et al., 1974; Babisch et al., 1990; Babisch and Gallacher, 1990). Although not in all these studies other risk factors were controlled for consistently, the hypothesis emerged that persistent noise stress increases the risk of cardiovascular disorders including high blood pressure (hypertension) and ischaemic heart disease:

- Sound/noise is a psychosocial stressor that activates the sympathetic and endocrine system.
- Acute noise effects do not only occur at high sound levels in occupational settings, but also at relatively low environmental sound levels when, more importantly, intended activities such as concentration, relaxation or sleep are disturbed.

The questions that need to be answered are:

- Do these changes observed in the laboratory habituate or persist under chronic noise exposure?
- If they habituate, what are the physiological costs? If they persist, what are the long-term health effects?

The answers to these questions come from epidemiological noise research. Large-scale epidemiological studies have been carried out for a long time (Babisch, 2000). The studies suggest, that transportation noise is associated with adverse cardiovascular effects, in particular ischaemic heart disease. The epidemiological evidence is constantly increasing (Babisch, 2002; Babisch, 2004a). The biological plausibility of the association derives from the numerous noise experiments that have been carried out in the laboratory. There is no longer any need to prove the noise hypothesis as such. Decision-making and risk management, however, rely on a quantitative risk assessment which requires an established dose-response relationship. Since many of the stress indicators and risk factors that have been investigated in relation to noise, are known to be classical cardiovascular risk factors, the hypothesis has emerged that chronic noise exposure increases the risk of hypertension, arteriosclerosis and ischaemic heart diseases. Its relevance for public health comes from the high prevalence of cardiovascular diseases in developed and industrialized countries. It is unclear as to what extent chronically repeated noise-induced sleep disturbance contributes to the development of somatic health disorders. Only a few epidemiological studies address this particular issue. Epidemiological noise research has seldom distinguished between day and night exposures, or between the exposure of the living room and the bedroom. However, some deduction can be made from daytime to night-time exposure.

5.2 Noise and stress - Reaction model

The auditory system is continuously analysing acoustic information, which is filtered and interpreted by different cortical and sub-cortical brain structures. The limbic system, including the hippocampus and the amygdala, plays an important role in the emotional processing pathways (Spreng, 2000). It has a close connection to the hypothalamus that controls the autonomic nervous system and the hormonal balance of the body. In laboratory studies, changes in blood flow, blood pressure and heart rate were found in reaction to noise stimuli as well as increases in the release of stress hormones including the catecholamines adrenaline and noradrenaline, and the corticosteroid cortisol (Babisch, 2003; Berglund and

Lindvall, 1995; Maschke et al., 2000). Such changes also occur during sleep without the involvement of cortical structures. The amygdala has the capacity to learn due to it's plasticity, particularly with respect to the meaning of sound stimuli (e.g. danger of an approaching lorry) (Spreng, 2000; Spreng, 2004). Acoustic stimulation may act as an unspecific stressor that arouses the autonomous nervous system and the endocrine system. The generalised psycho-physiological concept given by Henry and Stephens can be applied directly to noise-induced stress reaction (Henry, 1992). The stress-mechanism as such is genetically determined. It may be modified by experience and environmental factors. Its biological function is to prepare the organism to cope with a demanding stressor. The arousal of the sympathetic and endocrine system is associated with changes in physiological functions and the metabolism of the organism, including blood pressure, cardiac output, blood lipids (cholesterol, triglycerides, free fatty acids, phosphatides), carbohydrates (glucose), electrolytes (magnesium, calcium), blood clotting factors (thrombocyte aggregation, blood viscosity, leukocyte count) and others (Friedman and Rosenman, 1975; Lundberg, 1999; Cohen et al., 1995). In the long term, functional changes and dysregulation may occur, thus increasing the risk of manifest diseases. *Figure 16* shows the principal reaction schema used in epidemiological noise research for hypothesis testing (Babisch, 2002). It simplifies the cause-effect chain i.e.: sound - annoyance (noise) - physiological arousal (stress indicators) - (biological) risk factors - disease - and mortality (the latter is not explicitly considered in the graph). The mechanism works 'directly' through synaptic nervous interactions and 'indirectly' through the emotional and the cognitive perception of the sound. It should be noted that the 'direct' pathway is relevant even at low sound levels particularly during sleep, when the organism is at its nadir of arousal. The objective noise exposure (sound level) and the subjective noise "exposure" (annoyance) may serve independently as exposure variables in the statistical analyses of the relationship between noise and health endpoints.

Principally, the effects of environmental noise cannot directly be extrapolated from results of occupational noise studies. The two noise environments cannot simply be merged into one sound energy-related dose-response model (e. g., a simple 24 hour average noise level measured with a dose-meter). Noise effects are not only depending on the sound intensity but also on the frequency spectrum, the time pattern of the sound and the individuals' activities, which are disturbed. Therefore, epidemiological studies carried out under real-life conditions can provide the basis for a quantitative risk assessment provided that have adequate control over

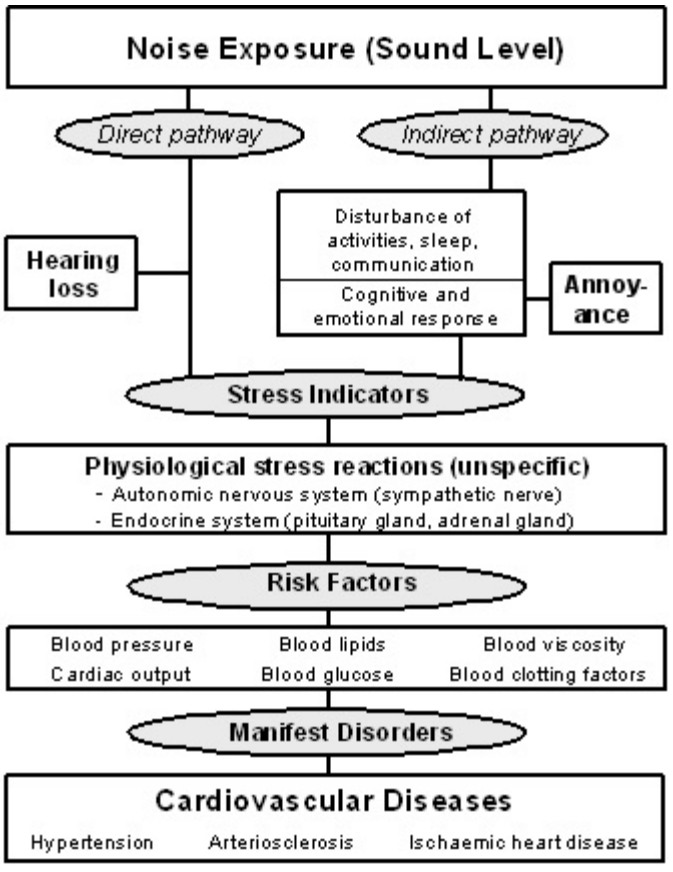


Figure 16 Noise effects reaction scheme (Babisch, 2002)

confounding and exposure variables. Other noise sources might act as confounders and/or effect modifiers on the association of interest. It was shown that the effects of road traffic noise (at home) were stronger in subjects that were also exposed to high noise levels at work (Babisch et al., 1990).

5.3 Previous reviews on environmental noise and cardiovascular risk

Causality in epidemiology can never be completely proven (Christoffel and Teret, 1991; Schlesselman, 1987; Weed, 2000). It is a gradual term for which evidence is increasing with the increasing number of facts. However, the magnitude of effect, presence of dose-response relationship, consistency with other studies in different populations and with different methodology and biological plausibility are commonly accepted arguments for a causal relationship (Bradford Hill, 1965; Evans, 1976; Morabia, 1991; Weed and Hursting, 1998). Classical, systematic and quantitative reviews have been published in the past, summarizing the results of studies that have be carried out up to the end of the last century, and assessing the evidence of the relationship between community noise and cardiovascular disease outcomes (Babisch, 2000; Berglund and Lindvall, 1995; Passchier-Vermeer and Passchier, 2000; Health Council of the Netherlands, 1994; Health Council of the Netherlands, 1999; Health Council of the Netherlands, 2004; IEH, 1997; Porter et al., 1998; Morrell et al., 1997), including a classical review and synthesis report by Babisch (Babisch, 2000) and a systematic review (meta-analysis) by v. Kempen et al. (Kempen et al., 2002). The status of evidence of the relationship between transportation noise and cardiovascular health as concluded by the year 2000 in the literature has been summarized as follows (Babisch, 2002; Babisch, 2004a).

Biochemical effects:	limited evidence
Hypertension:	inadequate or limited or sufficient evidence
Ischaemic heart disease:	limited or sufficient evidence

In a meta-analysis it was concluded that the risk of hypertension due to aircraft noise was 1.26 per increase of 5 dB(A) (95% CI: 1.14-1.39, L_{day} = 55 to 72 dB(A)) (Kempen et al., 2002). But, only one study (Knipschild, 1977a) was considered in the meta-analysis. With respect to road traffic noise and hypertension a pooled estimate of 0.95 per 5 dB(A) (95% CI: 0.84-1.08, L_{day} = <55 to 80 dB(A)) was calculated (Kempen et al., 2002). Two cross-sectional studies (Knipschild and Sallé, 1979; Knipschild et al., 1984) were considered in this calculation. The highest degree of evidence was for the association between community noise and ischaemic heart disease. Across the studies, there was not much indication of an increased risk for subjects who lived in areas with a daytime average sound pressure level of less than 60 dB(A). For higher noise categories, however, higher risks were relatively consistently found amongst the studies (Babisch, 2004a). Statistical significance was rarely achieved. Some studies permit reflections on dose-response relationships. These mostly prospective studies suggest an increase in risk for outdoor noise levels above 65-70 dB(A) during the daytime, the relative risks ranging from 1.1 to 1.5. Noise effects were larger when mediating factors like years in residence, room orientation and window opening habits were considered in the analyses. In a meta-analysis it was concluded that the risk of ischaemic heart disease increased by 1.09 per 5 dB(A) of the road traffic noise level (95% CI: 1.05-1.13, L_{day} = 51-70 dB(A)) (Kempen et al., 2002), when two cross-sectional studies (Babisch et al., 1993a) were considered. However, the pooled estimate of two prospective studies (Babisch et al., 1999) was calculated to be 0.97 per 5 dB(A) (95% CI: 0.90-1.04, L_{day} = 51-70 dB(A)) (Kempen et al., 2002). When the diagnosis of ischaemic heart disease was limited to myocardial infarction, three studies (Babisch et al., 1999; Babisch et al., 1994) were considered in this

meta analysis. Then the linear effect estimate was 1.03 per 5 dB(A) increase in road traffic noise level (95% CI: 0.99-1.09, L_{day} = 51-80 dB(A)). New studies have appeared in the meantime, which are included in the present updated review (Matsui et al., 2001; Matsui et al., 2004; Bluhm et al., 2001; Rosenlund et al., 2001; Goto and Kaneko, 2002; Lercher et al., 2002; Evans et al., 2001; Belojevic and Saric-Tanaskovic, 2002; Maschke, 2003; Niemann and Maschke, 2004; Franssen et al., 2004; Babisch et al., 2005). Others are on their way or have not yet been finalized and published, e.g. the pan-European projects "Hyena" (Jarup et al., 2003).

5.4 Updated review of epidemiological studies

Sixty epidemiological studies were recognized as having either objectively or subjectively assessed the relationship between transportation noise and cardiovascular endpoints. The identification of studies was based on the author's expert-knowledge of the topic and respective literature. Details are given in the major report (Babisch, 2006). Information particularly on night-time exposure (L_{night}: 22-6 hr or 23-7 hr) was seldom available. Newer studies used non-weighted or weighted averages of the 24 hr exposure (L_{eq}, L_{dn}, L_{den}). Some aircraft noise studies used national calculation methods (e.g. Dutch Kosten Units). For comparisons of study results and the pooling of data (meta analysis), sound levels were converted on the basis of best guess approximations to L_{day} (Passchier-Vermeer, 1993; Franssen et al., 2004; Matschat and Müller, 1984; Bite and Bite, 2004). It should be noted in this context that doubling/halving of road traffic volume results in a 3 dB(A) higher/lower average sound pressure level. Not all studies allowed dose-response reflections because some of them considered very broad exposure categories. Besides objective noise measurements, subjective measurements of exposure have been used in some epidemiological noise studies, which is in accordance with the noise-stress model. Type of road (e.g. busy street, side street etc.), disturbances and annoyance were rated by the study subjects from given scales.

5.5 Mean blood pressure

Table A2 of the major report (Babisch, 2006) lists the major findings of epidemiological traffic noise studies in which mean blood pressure was considered as the outcome (http://www.umweltdaten.de/publikationen/fpdf-l/2997.pdf). It indicates mean systolic and diastolic blood pressure differences as obtained from extreme group comparisons of noise exposure. The effects in children and in adults are discussed separately. The findings in children are difficult to interpret with regard to possible health risks in their later life. The effect may be of a temporary nature and may not be relevant to permanent health damage. There is evidence during childhood (Gillman et al., 1992), adolescence (Yong et al., 1993) and adulthood (Tate et al., 1995) that the blood pressure level at an early age is an important predictor of the blood pressure level at a later age. Studies over the full age range are missing (tracking). Growth and body weight are important factors on blood pressure development. The impact of body size was not adequately considered in some of the studies. A crude hint regarding reversible effects on blood pressure came from one study (Morrell et al., 2000). Results of the Munich intervention study on the effects of a reduction of aircraft noise have only been reported regarding cognitive performance but not with respect to change of blood pressure (Hygge et al., 2002). It was concluded from the available data on the length of exposure that children do not seem to adapt to high levels of road traffic noise but to some extent to aircraft noise (Bistrup et al., 2001; Passchier-Vermeer, 2000). However, the data base appears to be too poor to draw final conclusions. Aircraft noise studies focussed on the exposure at school, while road traffic noise studies mostly considered the noise exposure at home. The conclusions given by Evans and Lepore seem still to hold true (Evans and Lepore,

1993): “We know essentially nothing about the long-term consequences of early noise exposure on developing cardiovascular systems. The degree of blood pressure elevations is small. The clinical significance of such changes in childhood blood pressure is difficult to determine. The ranges of blood pressure among noise-exposed children are within the normal levels and do not suggest hypertension. The extent of BP elevations found from chronic exposure are probably not significant for children during their youth, but could portend elevations later in life that might be health damaging." Regarding mean blood pressure, no consistent findings in the relationship between traffic noise level and mean systolic or diastolic blood pressure can be seen in adults across the studies. In longitudinal studies, problems arose from migration of subjects, which had a considerable impact on sample size. The latter problem also applies to cross-sectional studies, in general. Sensitive subjects may tend to move out of the polluted areas, which dilutes the effect of interest. Medication due to high blood pressure may affect the blood pressure readings. However, the exclusion of subjects with hypertension or hypertension treatment, dilutes the true effect on blood pressure differences, if the hypothesis (noise causes high blood pressure) is true. In principle, hypotension - a fall in blood pressure - can also be a stress reaction. All this makes it more reasonable to look at manifest hypertension (defined by a cut off criterion) as a clinical outcome rather than at mean blood pressure readings (Ising, 1983; Winkleby et al., 1988). To date, there is no evidence from epidemiological data, that community noise increases mean blood pressure readings in the adult population. However, this does not discard the noise hypothesis as such. Studies suffered from insufficient power, narrow exposure range or other difficulties in the study design.

5.6 Hypertension

Table A3 of the major report (Babisch, 2006) gives the results of epidemiological traffic noise studies on the relationship between community noise level and the prevalence or incidence of hypertension (http://www.umweltdaten.de/publikationen/fpdf-l/2997.pdf). Hypertension in these studies was either defined by WHO criteria (Guideline Subcommittee, 1999), or similar criteria based on measurements of systolic and diastolic blood pressure, or from information which was obtained from a clinical interview, or a social survey questionnaire about doctor diagnosed hypertension. Most studies refer to road traffic noise. However, in recent years some new aircraft noise studies entered the database. The subjects studied were the adult male and female population, sometimes restricted to certain age ranges. With regard to the association between community noise and hypertension, the picture is heterogeneous. With respect to aircraft noise and hypertension studies consistently show higher risks in higher exposed areas. The evidence has improved since a previous review (Babisch, 2000). The relative risks found in four significantly positive studies range between 1.4 and 2.1 for subjects who live in high exposed areas, with approximate daytime average sound pressure level in the range of 60-70 dB(A) or more. Swedish studies found a relative risk of 1.6 at even lower levels >55 dB(A). With respect to road traffic noise, the picture remains unclear. New studies, more likely than older studies, tend to suggest a higher risk of hypertension in subjects exposed to high levels of road traffic noise, showing relative risks between 1.5 and 3.0. However, the earlier studies cannot be neglected in the overall judgement process. Across all studies no consistent pattern of the relationship between community noise and prevalence of hypertension can be seen. Dose-response relationships were considered in new studies. Subjective ratings of noise or disturbances due to traffic noise seem to consistently show a positive association with prevalence of hypertension. The relative risks found here range from 0.8 to 2.3. These studies, however, are of lower validity due to principal methodological issues regarding over-reporting (Babisch et al., 2003).

5.7 Ischaemic heart disease

Table A5 of the major report (Babisch, 2006) gives the results of cross-sectional epidemiological traffic noise studies on the relationship between noise level and prevalence of ischaemic heart disease (IHD); *Table A6* of the major report gives the results of case-control and cohort studies on the association between noise level and incidence of IHD (http://www.umweltdaten.de/publikationen/fpdf-l/2997.pdf). In cross-sectional studies, IHD prevalence was assessed by clinical symptoms of angina pectoris, myocardial infarction (MI), ECG abnormalities as defined by WHO criteria (Rose and Blackburn, 1968), or from self-reported questionnaires regarding doctor-diagnosed heart attack. In longitudinal studies, IHD incidence was assessed by clinical myocardial infarction as obtained from hospital records, ECG measurements or clinical interviews. The majority of studies refer to road traffic noise. With regard to ischaemic heart disease (IHD), the evidence of an association between community noise and IHD risk has increased since a previous review (Babisch, 2000). There is not much indication of a higher IHD risk for subjects who live in areas with a daytime average sound pressure level of less than 60 dB(A) across the studies. For higher noise categories, a higher IHD risk was relatively consistently found amongst the studies. Statistical significance was rarely achieved. Some studies permit reflections on dose-response relationships. These mostly prospective studies suggest an increase in IHD risk at noise levels above 65-70 dB(A), the relative risks ranging from 1.1 to 1.5 when the higher exposure categories were grouped together. Noise effects were larger when mediating factors like residence time, room orientation and window opening habits were considered in the analyses. This accounts for induction period (Rose, 2005) and improves exposure assessment. The results appear as consistent when subjective responses of disturbances and annoyance are considered, showing relative risks ranging from 0.8 to 2.7 in highly annoyed/disturbed/affected subjects. However, these findings may be of lower validity due to methodological issues.

5.8 Medication and drug consumption

Table A8 of the major report (Babisch, 2006) gives the results of studies on the relationship between drug consumption and community noise (http://www.umweltdaten.de/publikationen/fpdf-l/2997.pdf). Medication was primarily investigated with respect to aircraft noise. A significant prevalence ratio for medication with cardiovascular drugs of 1.4 was found in the sample of the Amsterdam airport (Knipschild, 1977a). The results of the "drug survey", where the annual data of the pharmacies regarding the purchase of cardio-vascular drugs were analysed (repeated cross-sectional survey) supported this finding. An increase in drug purchase over time in the exposed areas and not in the less exposed was found. This refers to the purchase of cardiovascular and antihypertensive drugs, as well as the purchase of hypnotics, sedatives and antacids (Knipschild, 1977c). Furthermore a dependency with changes in night-flight regulations was found (decrease after reduction of night-flights). A large recent study around Amsterdam airport found only a slightly higher risk of self-reported medication with cardiovascular drugs, including antihypertensive drugs, (relative risk 1.2) in aircraft noise exposed subjects where the noise level L_{den} exceeded 50 dB(A) (Franssen et al., 2004). Dose-response relationships across noise levels (L_{den} = <50 to 65 dB(A)) with respect to prescribed and non-prescribed sedatives/sleeping pills were found (relative risk 1.5 and 2.0, respectively) in the highest noise category of L_{den} = 61-65 dB(A). The preliminary results of an ongoing aircraft noise study from Sweden carried out around Stockholm's airport are in line with the Dutch studies (Bluhm et al., 2004a). A significant relative risk of 1.6 for the use of antihypertensive drugs was found in male subjects, where the noise level according to the Swedish calculation

standard exceeded FBM = 55 dB(A). The road traffic noise studies, where medication/purchase of drugs was investigated also tend to show a higher use in higher exposed subjects (Eiff and Neus, 1980; Schulze et al., 1983; Lercher, 1992). The relative risk for cardiovascular drugs was 1.3 in the Bonn study and 5.0 in the Erfurt study. The results for other drugs including sleeping pills, sedatives, tranquillizer and hypnotics ranged between 1.2 and 3.8 in these studies. All in all, the studies on the relationship between the use of medication or purchase of drugs and community noise support the general hypothesis of an increase in sleep disturbance and cardiovascular risk in noise-exposed subjects.

5.9 Evaluation of studies

This section refers only to studies where the prevalence or the incidence of manifest cardiovascular diseases was considered as a potential health outcome of chronic exposure to environmental noise. The focus here is on a quantitative risk assessment with respect to manifest diseases. Furthermore, studies on the effects of low-altitude jet-fighter noise are also excluded, because this type of noise includes other dimensions of stress (e.g. fear). Thirty-seven studies had assessed the prevalence or incidence of manifest diseases, including hypertension and ischaemic heart diseases (angina pectoris, myocardial infarction, ECG abnormalities).

5.9.1 Criteria

Epidemiological reasoning is largely based on the magnitude of effect estimates, dose-response relationships, consistency of finding, biological plausibility of the effects and exclusion of possible bias. Internal (the role of chance) and external validity (absence of bias and confounding) are important issues in the evaluation of studies (Bradford Hill, 1965). Analytic studies (e. g. cohort or cases-control studies) are usually considered as having a higher validity and credibility than descriptive studies (e. g. cross-sectional or ecological studies) (Hennekens and Buring, 1987), although many of the reservations against cross-sectional studies seem to be of minor importance when considering noise. For example, it does not appear to be very likely that diseased subjects tend to move differentially more often into exposed areas. Rather the opposite may be true, if noise stress is recognised as a potential cause of the individual's health problem. Thus, a cross-sectional study design may act conservatively on the results. The presence of a dose-response relationship is not a necessary criterion of causality. Non-linear relationships, including "u-" or "j-" shaped, saturation and threshold effects may reflect true associations (Calabrese and Baldwin, 2003; Rockhill, 2005). With respect to the derivation of guideline values in public health policy, the assessment of a dose-response relationship enables a quantitative risk assessment on the basis of continuous or semi-continuous (e.g. 5 dB(A) categories) exposure data. Dichotomous exposure data - on the other hand - that refer to a cut off criterion which splits the entire exposure range into two halves, can be used to evaluate the hypothesis of an association (qualitative interpretation), but not a quantitative assessment. The objective or subjective assessment of exposure and/or health outcomes is an important issue when judging the validity of a study (Malmström et al., 1999; Hatfield et al., 2001; Cartwright and Flindell, 2000). The objective prevalence of hypertension was found to be higher in a population sample than the subjective prevalence of hypertension (Schulte and Otten, 1993b). In a telephone survey more than half of the hypertensives classified themselves as normotensive (sensitivity 40% for men and 46% for women) (Bowlin et al., 1993). In a representative health survey, the validity of the self-reported assessment of morbidity (subjective morbidity) was found to be "low" with respect to hypercholesterolaemia, "intermediate" with respect to angina pectoris, hypertension and stroke and "high" with respect to myocardial infarction (Bormann et al., 1990). Myocardial

infarction is a very definite and severe health outcome which subjects would clearly know if they had experienced it. Its assessment by questionnaire tends to be more credible than that regarding hypertension. Test-retest reliability was found to be good with respect to “harder” outcomes, including high blood pressure and heart attack (Lundberg and Manderbacka, 1996; Lipworth et al., 2001). Over-reporting, on the other hand, may be a source of potential bias, particularly, when both, exposure and outcome, are assessed on a subjective basis (Winkleby et al., 1988; Babisch et al., 2003). The subjects may be more prone to blame their environment for their health problems, or may even tend to exaggerate adverse effects or exposure in order to influence noise policy. Therefore, a higher credibility and ranking was given to studies where exposure and outcome were assessed objectively (e.g. sound level versus subjective ratings, and measurement of blood pressure or a clinical interview versus self-reported hypertension in a self-administered questionnaire). This means that the sound level must have been measured or calculated on the basis of the traffic counts, and clinical interviews or measurements must have been carried out by medically trained personnel (no self-administered questionnaire data) to give a study a high ranking. Studies which have been adequately (e.g. stratification, model adjustment (regression), matching) controlled for a reasonable set of confounding variables in the statistical analyses, besides age and sex, were given a high ranking.

5.9.2 Assessment

The evaluation about the epidemiological studies was made with respect to the identification of good quality studies that can be feasibly considered for the derivation of guideline values. These studies can either be used for a statistical meta-analysis, for a combined interpretation (synthesis), or for singular interpretations. All the studies were evaluated with respect to the following criteria for the inclusion or exclusion in the synthesis process. Necessary criteria were: (1) peer-reviewed in the international literature, (2) reasonable control of possible confounding, (3) objective assessment of exposure and (4) objective assessment of outcome, (5) type of study, and (6) dose-response assessment. All six criteria were fulfilled by the two prospective cohort studies carried out in Caerphilly and Speedwell (Babisch et al., 1999; Babisch et al., 2003), the two prospective case-control studies carried out in the western part of Berlin ("Berlin I" and "Berlin II") (Babisch et al., 1992; Babisch et al., 1994), and the new prospective case-control study carried out in entire Berlin ("NaRoMI" = "Berlin III") (Babisch et al., 2005; Babisch, 2004b). The studies refer to road traffic noise and the incidence of myocardial infarction. They were also the only ones considered in an earlier meta-analysis on this issue (Kempen et al., 2002), with the exception of the “NaRoMI” study, which was not available at that time. All these studies are observational analytic studies (Hennekens and Buring, 1987). If descriptive studies on individuals – namely cross-sectional studies - are allowed, another two studies from Caerphilly and Speedwell on the association between road traffic noise and the prevalence of ischaemic heart diseases (IHD), myocardial infarction (MI) and angina pectoris (AP) can be taken into account (Babisch et al., 1988; Babisch et al., 1993b; Babisch et al., 1993a). These studies were also considered in the meta-analysis by v. Kempen et al. (Kempen et al., 2002). However, the results of the Berlin study on MI prevalence (Babisch et al., 1994) - which was also considered in that meta-analysis - are not considered here, because the outcome was assessed subjectively with a self-administered questionnaire (an exclusion criterion). All the studies suggest an increase of ischaemic heart diseases, in particular, myocardial infarction. These studies are used for a new meta-analysis (section 5.10).

Regarding aircraft noise, the cross-sectional Okinawa study (Matsui et al., 2004; Matsui et al.,

2001) on the association between aircraft noise and hypertension fulfils the inclusion criteria. When studies are included that did not assess dose-response relationships but only compared dichotomous categories of exposure in the analyses, two more studies appear on the list. The studies were carried out in the vicinity of the Amsterdam airport. They suggest a higher risk of cardiovascular diseases in general (Knipschild, 1977b), and – specifically - for hypertension and ischaemic heart diseases (angina pectoris, ECG-abnormalities, heart trouble) (Knipschild, 1977a) in subjects from areas exposed to high aircraft noise. These studies were considered in the meta-analysis by Kempen et al. (Kempen et al., 2002). However, they do not fulfil the strict criteria set here. Finally, if the inclusion criteria are widened to include peer-reviewed studies that assessed dose-response relationships between objective indicators of exposure and the subjective (self-reported) prevalence of diseases, a further two studies can be considered. These are the cross-sectional study carried out in Stockholm regarding the association between aircraft noise and hypertension (Rosenlund et al., 2001), and the cross-sectional part of the study in Berlin regarding the association between road traffic noise and myocardial infarction (Babisch et al., 1994). *Figure 17* shows the results of the three aircraft noise studies carried out in Amsterdam, Okinawa and Stockholm (Knipschild, 1977a; Matsui et al., 2004; Rosenlund et al., 2001). The graph clearly indicates that the results are too heterogeneous to derive a pooled dose-response curve. However, all three studies show an increase in risk with increasing noise level.
Studies that are not given a high ranking according to the above mentioned criteria, however, may serve as additional sources of information to support the evidence of the conclusions being made on the basis of this review. This is illustrated in *Figure 18*. The entries are relative risks (centre of the bars) with 95% confidence intervals (the bars) for dichotomous comparisons of noise exposure (extreme groups or high vs. low). A relative consistent shift of the bars to relative risks greater than 1 can be seen. The dark-shaded bars in the diagram refer to studies where the noise exposure was determined objectively (noise levels), the light-shaded bars where it was determined subjectively (annoyance). Road traffic and aircraft noise studies are here viewed together. No corresponding results are available for rail traffic studies. If different subgroups of the population (males/females) or different health endpoints were taken into account, specific studies appear more than once in the illustration.

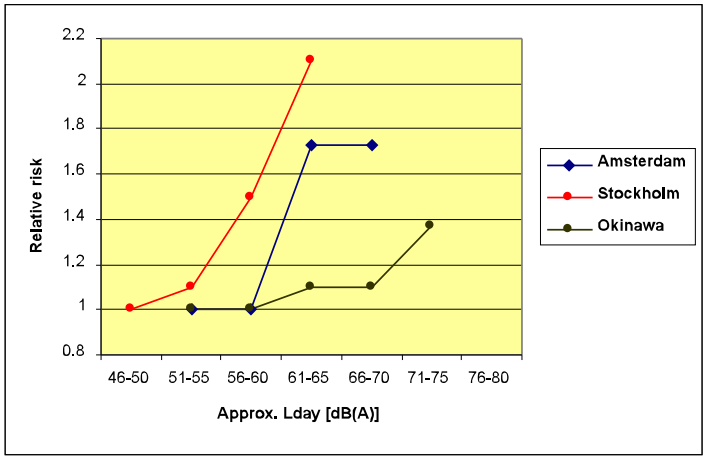
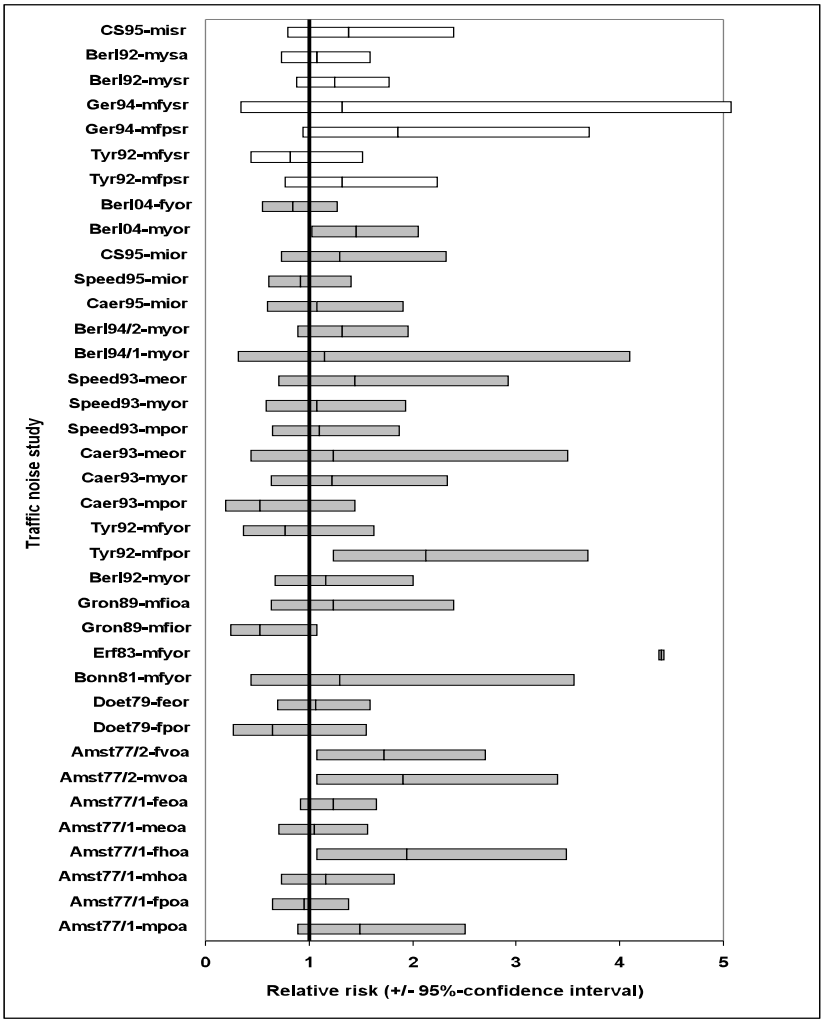


Figure 17. Association between aircraft noise level and the prevalence of hypertension



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 signs, h- heart complaints, i -ischemic heart disease, p- Angina pectoris, v- cardiovascular complaints in general, y- heart attack

Figure 18. Results of epidemiological studies on the association between traffic noise and ischaemic heart disease (original figure (Babisch, 2002) has been modified according to the results of (Babisch, 2004b))

5.10 Dose – response curve: meta analysis

For a quantitative risk assessment and the derivation of guidelines for public health noise policy a common dose-response curve is required. The risk estimates obtained from different noise studies can be summarized using the statistical approach of a meta-analysis. Based on the judgement criteria discussed in section 3.9.2, five analytic and two descriptive studies emerged that can be used to derive a common dose-response curve for the association between road traffic noise and the risk of myocardial infarction. Two separate meta analyses were made by considering the analytic studies that were carried out in Caerphilly and Speedwell (cohort studies) and Berlin (case-control studies) on the one hand, and the descriptive studies that were carried out in Caerphilly and Speedwell (cross-sectional studies) on the other hand. It turned out as a result of the evaluation, that all these studies referred to the road traffic noise during the day (L_{day}: 6-22 h) and the incidence or prevalence of myocardial infarction as the outcome. Study subjects were men. In all analytic studies the orientation of rooms was considered for the exposure assessment (facing the street or not). With respect to the Caerphilly and Speedwell studies, the 6 yrs pooled follow-up data provided the respective information. In all descriptive studies the traffic noise level referred to the facades that were facing the street and did not consider the orientation of rooms/windows. All individual effect estimates were adjusted for the covariates considered in each of the studies. Different sets of covariates were considered in each study. However, this pragmatic approach accounts best for possible confounding in each study and provides the most reliable effect estimates derived from each study. The concept of meta-analysis was used to aggregate and summarize the findings of the different studies (Olkin, 1995; Blettner et al., 1999). The program "meta" was downloaded from the "Stata" website for use in the statistical package STATA (version 8.0), and for calculating the pooled random effect estimates. *Table IV.1* shows individual and pooled effect estimates with confidence intervals (rounded brackets), statistical weights (square brackets) for the individual studies, and the Q-test of heterogeneity between studies. According to the Q-test, the nil-hypothesis of non-heterogeneity was never discarded. *Figures 19 and 20* show odds ratios of individual studies and the pooled estimates for the descriptive and analytic studies.

Table IV.1. Single and pooled (meta analysis) effect estimates (odds ratios and 95% confidence intervals) of descriptive and analytic studies on the relationship between road traffic noise level (L_{day}) and the incidence/prevalence of myocardial infarction

	Road traffic noise level - L_{day} - [dB(A)]					
Descriptive studies	**51-55**	**56-60**	**61-65**	**66-70**		**N**
Caerphilly	1.00	1.00 (0.58-1.71), [13.29]	0.90 (0.56-1.44), [17.23]	1.22 (0.63-2.35), [8.98]		2512
Speedwell	1.00	1.02 (0.57-1.83), [11.19]	1.22 (0.70-2.12), [12.62]	1.07 (0.59-1.94), [10.94]		2348
Pooled	1.00	1.01 (0.68-1.50)	1.02 (0.72-1.47)	1.14 (0.73-1.76)		
Q-Test		p = 0.96	p = 0.41	p = 0.77		
Analytic studies	**<=60**	**61-65**	**66-70**	**71-75**	**76-80**	**N**
Caerphilly + Speedwell	1.00	0.65 (0.27-1.57), [4.95]	1.18 (0.74-1.89), [17.48]	---	---	3950
Berlin I	1.00	1.48 (0.57-3.85), [4.21]	1.19 (0.49-2.87), [4.94]	1.25 (0.41-3.81), [3.09]	1.76 (0.11-28.5), [0.50]	243
Berlin II	1.00	1.16 (0.82-1.65), [31.43]	0.94 (0.62-1.42), [22.76]	1.07 (0.68-1.68), [18.92]	1.46 (0.77-2.78), [9.27]	4035
Berlin III	1.00	1.01 (0.77-1.32), [54.42]	1.13 (0.86-1.49), [50.87]	1.27 (0.88-1.84), [28.24]	---	4115
Pooled	1.00	1.05 (0.86-1.29)	1.09 (0.90-1.34)	1.19 (0.90-1.57)	1.47 (0.79-2.76)	
Q-Test		p = 0.57	p = 0.87	p = 0.84	p = 0.90	

Numbers are odds ratios, 95% confidence intervals are given in round brackets (), weights are given in square brackets []; Pooled = pooled estimates (meta analysis), p = probability of the Q-Test for heterogeneity, N = sample size

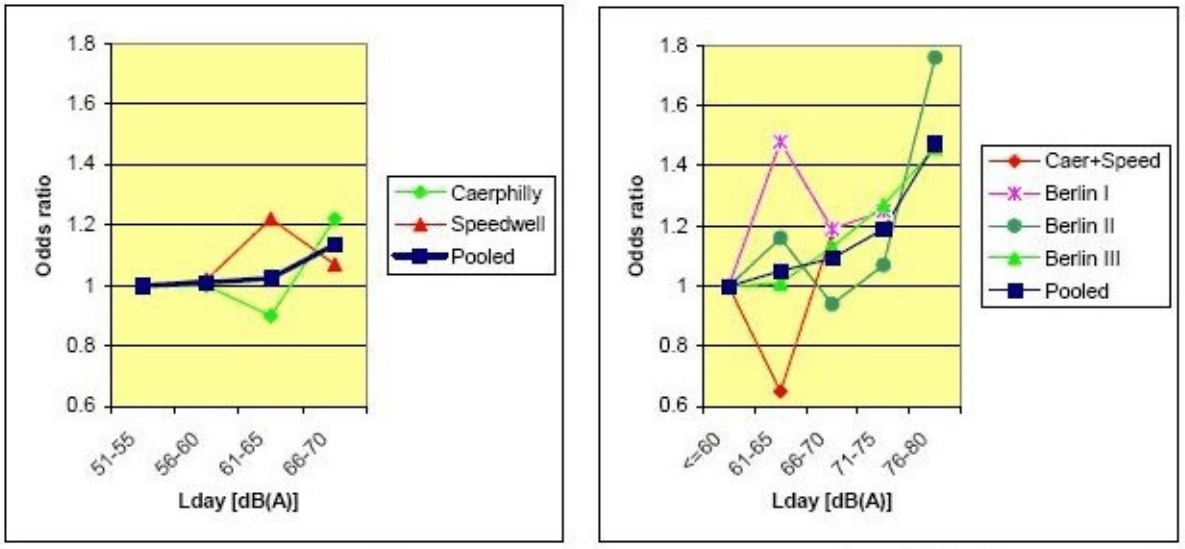


Figure 20 and 20. Single and pooled effect estimates (odds ratios) for the descriptive and analytic studies of the association between road traffic noise level and the prevalence (left graph) and incidence (right graph), respectively, of myocardial infarction.

5.11 Effect modification

Support for any noise effect relationship may come from subgroup analyses that are in line with the noise hypothesis. This refers to effect modification with respect to residence time, window opening behaviour and other determinants that affect the noise exposure and cumulative noise dose. In the Amsterdam aircraft noise studies, a steady increase in the purchase of cardiovascular and antihypertensive drugs at local pharmacies was found over the period of 8 years in a community newly exposed to aircraft noise. No such increase was found in a control community that was not exposed to aircraft noise (Knipschild, 1977c). Positive associations between the prevalence of cardiovascular diseases and residence time in exposed areas (but not in unexposed) were also found in the road traffic noise studies carried out in Bonn with respect to hypertension (Eiff and Neus, 1980; Neus et al., 1983) and in Caerphilly and Speedwell with respect to the ischaemic heart disease (Babisch et al., 1999; Babisch et al., 2003). When the analyses of the road traffic noise studies carried out in Berlin, Caerphilly and Speedwell were restricted to subjects who had not moved within a retrospective period of 10 to 15 years, the effect estimates turned out to be larger than for the total samples of each study (Babisch et al., 1994; Babisch et al., 1999; Babisch et al., 2005). Similarly, a larger effect was found in the study in Sollentuna with respect to hypertension (Bluhm et al., 2001). No such an effect was found in the Luebeck study (Herbold et al., 1989; Hense et al., 1989). The cross-sectional data of the study carried out in Los Angeles on children regarding mean blood pressure, indicated some habituation to aircraft noise (Cohen et al., 1980). The longer the children were enrolled in the school, the smaller was the difference in blood pressure between exposed and non-exposed children. However, the follow-up study suggested that this may also be an effect of attrition (Cohen et al., 1981). The longer the families experienced the noise, the more likely that they moved away from the exposed areas (selection bias). In contradiction to this, blood pressure differences between children exposed and not-exposed to road traffic noise increased with school-grade (Karsdorf and Klappach,

1968). Intervention studies were conducted with respect to changes in blood pressure and changes in air traffic operation (e.g. opening/closing of airports or runways). In the Munich study, a larger increase in blood pressure was found in children from a noisy area (Evans et al., 1998). Other Studies suggested reversible effects on blood pressure when the exposure was lowered (Morrell et al., 2000; Morrell et al., 1998; Wölke et al., 1990). In the Tyrol study, significantly lower blood pressure readings were found in subjects who kept the windows closed throughout the night (Lercher and Kofler, 1996; Lercher and Kofler, 1993). When the subjects lived close to the highway (within a distance of approx. 500 m), the prevalence of hypertension was higher in subjects whose bedroom was facing the main road than in those, whose bedroom was not facing the main road. The orientation of rooms and window opening was also found to be an effect modifier of the association between road traffic noise and ischaemic heart disease in the Caerphilly and Speedwell studies (Babisch et al., 1999). The relative risk with respect to the noise level was slightly higher in subjects with rooms facing the street and subjects keeping the windows usually open when spending time in the room. A much greater relative risk of hypertension was found in subjects who slept with open bedroom windows in the Spandau Health Survey (Maschke, 2003; Maschke et al., 2003a). Hearing impairment was found to be an effect modifier on the association between aircraft noise and hypertension (Rosenlund et al., 2001). Amongst the exposed subjects, a higher risk associated with the noise was only found in subjects without hearing loss.

5.12 Exposure during the night

Unfortunately, epidemiological noise research provides nearly no information regarding the particular impact of noise exposure during the night on cardiovascular health outcomes. The Spandau Health Survey explicitly distinguished between the exposure of the living room (during the day) and the exposure of the bedroom (during the night). There, a slightly higher relative risk of hypertension was found with respect to the traffic noise level during the night (relative risk 1.9 vs. 1.5) compared with the noise level during the day (Maschke, 2003; Maschke et al., 2003a). Furthermore, sleeping with open bedroom windows was associated with a large increase in risk. However due to the small sample size, the confidence intervals were very large. In the drug survey of the Amsterdam aircraft noise studies, a steady increase in purchase of hypnotics (sleeping pills) and sedatives was found (Knipschild, 1977c). This trend decreased considerably when night flights were largely banned. Such decrease was not found regarding cardiovascular drugs for which the purchase also increased over time. However, this may partly be due to the fact that atherosclerotic manifestations of high blood pressure were less reversible (in contrast to vasoconstriction, which is more related to acute or semi-acute effects, e.g. in children). It was mentioned in the previous section that closing the windows had a protective effect on blood pressure readings in the Tyrol study (Lercher and Kofler, 1993). This was only found regarding closing the windows during the night and not during the day. Furthermore, subjects who had switched the bedroom and the living room because of the noise, had a significantly lower blood pressure than those who did not so. The findings are discussed in a broader context of coping strategies (Lercher, 1996). When subjective responses to community noise were considered, higher relative risks of cardiovascular diseases were found for noise-related disturbances of sleep and relaxation, rather than for other disturbances or subjective descriptors of noise exposure, which did not refer to the night-time. This was found in the Caerphilly and Speedwell studies (Babisch et al., 2003), the "NaRoMI" study (Babisch et al., 2005), the Spandau Health Survey (Maschke et al., 2003a) and a general population sample of Germany (Bellach et al., 1995). The "LARES" study (Niemann and Maschke, 2004), in which noise-induced sleep disturbance was assessed, did not show a higher relative risk compared with the general annoyance.

5.13 Risk groups

Most epidemiological noise studies looked at cardiovascular effects of community noise in men. This may simply be due to the fact that the prevalence of cardiovascular diseases in middle-aged subjects is higher in men than in women. Statistical power is an important issue for the design of a study. Furthermore, in noise experiments, physiological reactions controlled by the autonomic nervous system were less pronounced in females than in males (Neus et al., 1980; Ising and Braun, 2000). Improper control for possible differential effects of the intake of sex hormones including contraceptives, which may protect or promote adverse (noise-) stress effects, may act conservatively on the results (Eiff, 1993; Farley et al., 1998; Cairns et al., 1985). In the studies carried out in Luebeck (Hense et al., 1989; Herbold et al., 1989), Pancevo (Belojevic and Saric-Tanaskovic, 2002), Berlin (Babisch et al., 2005), Stockholm (Rosenlund et al., 2001), a German population sample (Bellach et al., 1995), Bonn (residence time) (Eiff and Neus, 1980; Eiff et al., 1981b), and in Amsterdam (angina pectoris) (Knipschild, 1977a) higher prevalences of hypertension, ischaemic heart diseases and the use of cardiovascular drugs were found in noise exposed men than in women. The opposite was found in the studies carried out in Bonn (sound level) (Eiff and Neus, 1980; Eiff et al., 1981b), Sollentuna [#46], and in Amsterdam (heart trouble] (Knipschild, 1977a; Bluhm et al., 2001). In the studies carried out in the Soviet Union, it was reported that noise effects on the cardiovascular system were more pronounced in young and middle-aged subjects (Karagodina et al., 1969). Similar results were found in Swedish noise studies (Bluhm et al., 2004b; Bluhm et al., 2001) and the "LARES" study (Niemann and Maschke, 2004). The opposite (larger effects in elderly subjects) was reported from the Amsterdam study (Knipschild, 1977a) and the Stockholm study (Rosenlund et al., 2001). The available database on cardiovascular effects of noise in children is poor. No data are available that refer, in particular, to noise and sleep. The quantitative impact of transportation noise on the cardiovascular system is still a matter of research. A quantitative health risk assessment for children cannot be made at the moment. Based on the available information from noise studies, it must be concluded that children do not appear to be a particular risk group with respect to cardiovascular outcomes, especially blood pressure. This does not mean that the literature does not suggest higher blood pressure readings in children. It only means, that the effect in children does not appear to be different than that in adults. However, children may be longer exposed to noise throughout their lifetime than the adults that have been studied. No long-term follow-up studies are known that focus on noise exposure. Most studies on children considered noise in schools rather than noise at home, which implies different mechanisms about how noise could contribute to a rise in blood pressure (raised effort in learning/speech perception vs. disturbed relaxation/sleep). The prospective part of the Caerphilly and Speedwell studies gave a small hint that health status could be a modifying factor. In subjects with prevalent chronic diseases, road traffic noise was associated with a slightly larger increase in the incidence (new cases) of ischaemic heart diseases than in subjects without prevalent diseases – when the objective noise level was considered (Babisch et al., 2003). Surprisingly, when annoyance and disturbances due to traffic noise were considered for exposure, the opposite was found. Noise effects were only seen in subjects without prevalent diseases. This was discussed with respect to reporting bias.

5.14 Risk evaluation

The process of risk assessment (risk evaluation) comprises hazard identification ("Which health outcome is relevant for the exposure?"), exposure assessment ("How many are affected") and dose-response assessment ("Threshold of effect?"). This information is summarized in "risk characterization" ("health hazard characterization"). It involves the

interpretation of the available evidence from the available data and other scientific disciplines, and is subject to discussion of the uncertainties. These include chance, bias and validity of studies as well as transparency, replicability and comprehensiveness of reviews. As a result of the risk evaluation process, a quantitative estimate about the likelihood that the hazard will affect exposed people will be derived. Usually attributable risk percentages are calculated (Walter, 1998). This will serve as key information for any kind of risk management including regulatory options (Jasanoff, 1993). The term “adverse” is essential in this context of environmental standard setting. Risk management should ensure that “adverse” health effects do not occur. The fact that an organism responds to noise does not have to be per se “adverse”. The severity of a health outcome is an important determinant of the adversity of an effect and implies variable action levels for public health policy (Babisch, 2002; Babisch, 2004a; Griefahn et al., 2002; Health Council of The Netherlands, 2003). Since considerable parts of the population are exposed to high noise levels (EEA, 2004), noise policy can have a significant impact on public health (Neus and Boikat, 2000). Due to the increasing number of people affected with the decreasing severity of the effect, even small individual risks and less severe health outcomes can be relevant for public health and decision-making. It has been shown, that moderate noise exposures implying a small individual risk, may cause more noise-induced cases of health impaired subjects than higher noise exposures. It was pointed out that the number of people suffering from poor health due to aircraft noise is dominated by the larger number of people that is exposed to relatively moderate to low noise levels and not by those exposed to high noise levels (Franssen et al., 2004). This means that more emphasis should be put on the reduction of noise in moderately exposed areas. However, public health policy cannot only consider population attributable risks (risk percentages), but must also consider individual risks (lifetime risk). In practice, it seems to be reasonable that noise policy should reduce noise beginning with the highest exposures and ending with the lowest ones. Decision-making will have to find common standards of acceptable risks, which may vary according to the cost-benefit considerations within and between communities and countries. Such practical standards may though vary due to the economic development and abilities, the cost-benefit considerations and priority settings of a community or country. Health quality targets derived from scientific research are usually intended to minimize risks; decision making in the political process is only partly scientifically based due to economic limitations and concurring interests (Nijland et al., 2003). Different health-outcomes or indicators of well-being and quality of life imply different action levels. Environment and health policy must determine acceptable noise standards that consider the whole spectrum from subjective well-being to somatic health (e.g. annoyance, physiological arousal, health risk). The evidence for a causal relationship between community or transportation noise and cardiovascular risk appears to have increased throughout the recent years due to new studies that accomplish the data base. Compared with earlier conclusions (see section 3.3) this refers to hypertension and ischaemic heart diseases, in particular:

Biochemical effects:	limited evidence
Hypertension:	limited or sufficient evidence
Ischaemic heart disease:	sufficient evidence

5.15 Conclusions

The evaluation process, used in this paper considered the "necessary" criteria: peer-reviewed publication in an international journal, reasonable quantitative control of possible confounding, objective assessment of exposure and outcome, type of study (analytic vs. descriptive), and dose-response assessment (not only dichotomous "high" vs. "low"). The

approach differs from that of an earlier meta-analysis (Kempen et al., 2002) in that there regression coefficients were calculated for the entire dose response curve within a single study (e.g., the increase in risk per 5 dB(A)), which were then pooled between studies. Since higher exposure categories usually consist of smaller numbers of subjects than the lower categories, regression coefficients across noise levels tend to be influenced by the lower categories. This may lead to an underestimation of the risk in higher noise categories. The approach presented here pooled the effect estimates of single studies within each noise category, thus giving more weight to the higher noise categories and accounting for possible non-linear associations.

Figures 21 and 22 show the two risk curves for descriptive and analytic studies (Hennekens and Buring, 1987). The graphs show the pooled effect estimates (odds ratios) and the 95% confidence intervals for each noise category. Whereas the cross-sectional studies (*Figure 22*) cover the sound level range of L_{day} from >50 to 70 dB(A), the cohort and case-control studies (*Figure 21*) cover the range from 60 to 80 dB(A). Both curves together can serve as a basis for a quantitative risk assessment. From *Figure 21* it can be seen that below 60 dB(A) for L_{day} no noticeable increase in MI risk is to be detected. Therefore for the time-being, L_{day} = 60 dB(A) can be seen as NOAEL ("no observed adverse effect level") for the relationship between road traffic noise and myocardial infarction (Babisch, 2002). For noise levels greater than 60 dB(A), the MI risk increases continuously, and is greater than 1.2 for noise levels of 70 dB(A). This can be seen in *Figure 22*. It should be mentioned that the risk estimates, in general, were found to be higher in subjects that had lived in the exposed areas for a longer time (Babisch et al., 1994; Babisch et al., 1999; Babisch et al., 2005). This is in accordance with the noise hypothesis and the effects of chronic noise stress (Lercher and Kofler, 1996; Thompson, 1997). However, for the calculation of population attributable risks the figures for the whole population are relevant due to unknown information about residence time.

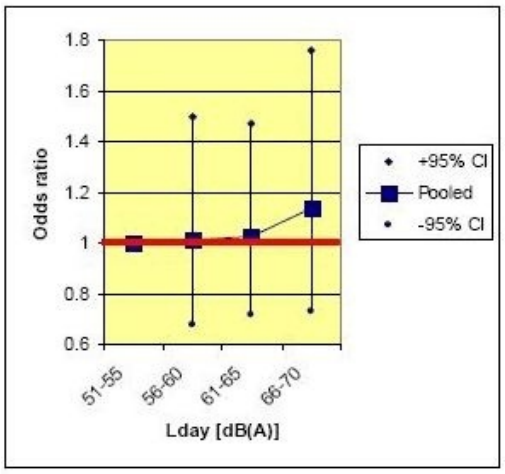


Figure 21

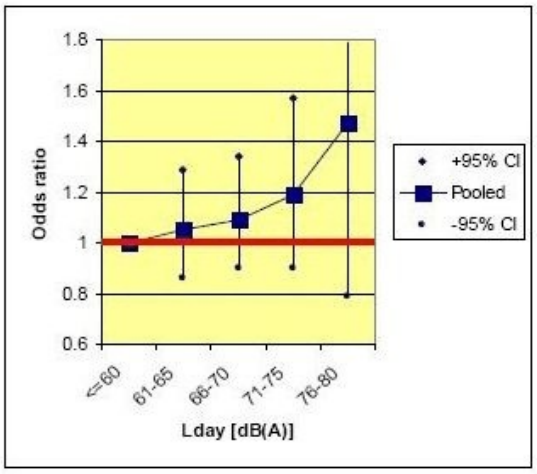


Figure 22

Figures 21 and 22 show pooled effect estimates (meta analysis) of descriptive and analytic noise studies of the association between road traffic noise level and the prevalence (left graph) and incidence (right graph), respectively, of myocardial infarction (odds ratio 95% confidence interval).

No particular risk groups could be identified on the basis of epidemiological research on cardiovascular effects of community noise. The assessment of dose-effect relationships

sometimes suggested a cut-off level, above which the risk tends to increase. From a biological point of view, one would expect a continuous increase in risk with increasing noise level. However, adaption, habituation and coping may be reasons for an empirical threshold of effect. Decisions with respect to guidelines values usually refer to a quantitative risk assessment of populations (e.g. population attributable risk percent). However, prevention strategies – for ethical reasons - should not ignore the individual risks of highly exposed subjects, even if their number may be small.

With respect to night noise exposure, nearly no information is available from epidemiological studies on the cardiovascular effects of long-term noise exposure of the bedroom during the night. Only one study distinguished between the exposures of the bedroom and the living room in the statistical analyses (Maschke et al., 2003b). The results suggested slightly higher effect estimates for the prevalence of hypertension with respect to the noise exposure of the bedroom (during the night) compared with the exposure of the living room (during the day). However, the difference was small (odds ration 1.9 vs. 1.5), which means that it still remains an open question whether the night exposure or the overall exposure throughout the whole day is the driving force. The study has some methodological limitations that were addressed in the summary of the major technical report and in a recent advisory report of the Dutch Health Council (Health Council of the Netherlands, 2004). They are mainly concerned with the fact that the study population consisted of a selected, predominantly older and health conscious group of persons that might have already suffered from regular health problems (risk group). A few studies that looked at the association between subjective responses to community noise and cardiovascular outcomes suggest a closer relationship with sleep-related annoyance /disturbance reaction rather than with non-sleep related annoyance/disturbance (Babisch et al., 1999; Babisch et al., 2005; Bellach et al., 1995; Maschke et al., 2003a; Niemann and Maschke, 2004). Closing the bedroom window or, vice versa, sleeping with the bedroom window open, was associated with a lower or higher risk, respectively (Lercher, 1996). The same was found with respect to changing the bedroom with the living room because of noise. These findings may indicate that night-time noise may be more a determinant of noise-induced cardiovascular effects than the daytime exposure. However, daytime activity patterns and expectations of the individuals are much more inhomogeneous than during the night, which tends to dilute the statistical association of true effects with the day-noise exposure.

Given the situation that only a few data are available from epidemiological studies with respect to effects on sleep (exposure of the bedroom during the night), there does not seem to be any other way of reasoning, than inferring night noise recommendations or guidelines from the results of studies that refer to the noise exposure during the daytime period (L_{day}) or the whole day (L_{dn}, L_{24h}). L_{den}, in this context, appears to be a useful noise indicator for decision-making and regulatory purposes. Penalties of 5 dB(A) and 10 dB(A) are usually given to the evening period and the night period, respectively. It can be used for noise mapping and refers normally to the most exposed facade, which incorporates a certain degree of exposure misclassification regarding cause-effect relationships. This weighted indicator was introduced to assess the relationship between sound level and noise annoyance (Directive 2002/49/EC, 2002). However, it may not be adequate for (somatic) health-related noise effects' research. Non-weighted separate exposure indicators, such as L_{day}, $L_{evening}$ or L_{night}, may be more appropriate when assessing physiological responses to the noise. In urban settings, night-time average noise levels (22-6 h) for road traffic tend to be approx. 7-10 dB(A) lower than daytime average noise levels - relatively independent (no freeways) of the traffic volume of

the street (Utley, 1985; Ullrich, 1998; Evans et al., 2001). In such cases, L_{den} is approx. 2 to 3 dB(A) higher than L_{day} (Bite and Bite, 2004). Therefore, in epidemiological studies in which the relative effects of road traffic noise is studied, the sound emission during the daytime can as well be viewed as an approximate relative measure of the overall sound emission including the night. This seems to be further justified because existing noise regulations usually consider a 10 dB(A) difference between the day and the night. The NOAEL of 60 dB(A) for L_{day} corresponds, in this respect, with 50 dB(A) for L_{night}. This approximation can only be made with respect to road traffic noise.

Aircraft noise has been less intensively studied in noise epidemiology. The studies focused on high blood pressure. Dose-response curves were hardly considered. A large European study on the association between aircraft noise and road traffic noise on blood pressure is currently being conducted (Jarup et al., 2003). Regarding aircraft noise - and particularly the ongoing debate on night-flight restrictions in the vicinity of busy airports - no other alternative exists at present than to take the MI risk curves derived from road traffic noise studies as an approximate for aircraft noise. 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 (Babisch, 2004a; Ortscheid and Wende, 2000). 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 (Miedema and Vos, 1998). More research is needed regarding the association between aircraft noise and cardiovascular endpoints.

This section is clearly focussed on ill-health as an outcome of the adverse effect of noise. A common dose-effect curve for the relationship between road traffic noise (outdoors) and the risk of myocardial infarction was developed. This curve can be used for a quantitative risk assessment and the calculation of attributable cases in a community. However, decisions regarding limit values have to be made within the spectrum between discomfort (annoyance) and ill-health (disease) (Babisch, 2002; Lindström, 1992). Whereas quality targets at the lower end of the effects scale may be more flexible, quality targets at the upper end may be more obligatory. For example, for ethical reasons (equality principle) it does not seem to be justified if (ill-)health-based limit values are varied according to the type of living area as expressed in land development plans (e.g. residential, mixed or commercial).

6 Insomnia (*Health Council of the Netherlands, 2004, pg64*)

A group of Japanese researchers carried out a questionnaire-based survey of 3600 adult Japanese women (aged between twenty and eighty) to gather information about the factors that contribute to insomnia (Kageyama, 1997). Some 11 per cent of subjects were found to be affected by insomnia (as defined on the basis of the WHO's ICD10 classification system, see Ch.II.1). Analysis of the survey data took account of various distorting variables, such as age, number of (small) children in the family, social status, receipt of medical treatment, regularity of bedtimes, apnoea-like problems and serious unpleasant experiences in the six months prior to completing the questionnaire. When the percentage of insomniacs in each of the three areas with the highest exposures was compared with the percentage in the low-exposure areas, the ratios worked out at, respectively, 1.4 (2100 vehicles per hour, L_{night} of around 65 dB(A)), 2.1 (2400 vehicles per hour, L_{night} of around 67 dB(A)) and 2.8 (6000 vehicles per hour, L_{night} of around 70 dB(A)). The

most frequently reported problem was difficulty getting to sleep.
Research into the effects of exposure to air and road traffic noise has shown that increases in night-time noise exposure or in noise exposure during the sleep latency period have a statistically significant adverse impact on subjects' ability to get off to sleep and on sleep inception periods.

7 Effects on performance *(Hygge)*

7.1 Cognition and slow-wave sleep

Jan Born and co-workers at University of Lübeck (Benedict et al., 2004; Born & Wagner, 2004; Drosopoulos, Wagner & Born, 2005; Gais & Born, 2004; Wagner, Gais & Born, 2001) have reported interesting research and put forward intriguing hypotheses on the relation between noise exposure, sleep loss and subsequent cognitive performance. They conclude that declarative memory benefits mainly from sleep periods dominated by slow-wave sleep (SWS), while there is no consistent benefit of this memory from periods rich in rapid eye movement sleep (REM). This points to the importance of SWS for declarative memory.
Since sleep in the early night is dominated by SWS, in contrast to late night when REM sleep dominates, this would imply that noise in the early night, e.g. aircraft noise before midnight, would be particularly damaging to memory and related cognitive functions. However, this implication has not yet been explicitly tested. That is, there seem to be a certain risk for impoverished memory due to noise in the early night, but there is as yet no graded quantification about whether ordinary before-midnight noise levels around large airports are sufficient to make a difference to SWS. We also lack graded quantification about the relationship between impoverished SWS and the resulting effect on different aspects of declarative memory.
That is, in terms of Figure 1 we have evidence for the arrow marked b, but we do not have enough information to say whether the strength of arrow a is sufficient to cause reduced SWS in field settings.
Further, since children's memory systems pass through developmental changes and are not structured in the same way as for adults, it would be interesting to know to what extent the Born-group results are also valid for children, and whether the depth of children's sleep counteract or enhance the SWS dominance in the early night.

7.2 Comparing day- and nighttime noise exposure

As implied by Figure 1, the relation between noise exposure and resulting effects on cognition should be analysed somewhat differently depending on whether the noise exposure takes time during the day or night. Analysing the cognitive effects of daytime noise exposure is fairly straightforward. For night time noise exposure, however, any effects on cognition can either be a more or less direct effect of the noise exposure, or an indirect effect mediated by reduced sleep or sleep quality.
Also, comparing e.g. memory and learning functions when exposed to night time noise, in contrast to daytime noise, shifts the focus of analysis away from encoding (in memory) or acquisition (in learning) while in noise, to a focus on storing the material to be remembered or learnt while asleep (compare to day-time noise effects on cognition as reported by Hygge Evans & Bullinger, 2002; Stansfeld et al., in press). That is, assuming that people mainly are asleep a night, all cognitive work that relies on the intake of information, listening or reading is not relevant.
In all, this suggests that studies of daytime noise levels cannot be pushed much to give rough

estimates of the effects of night time exposure.

7.3 Comparing children and adults

How far can daytime noise levels effects on children be generalized to give a rough estimate of the effects on adults? Are children more sensitive? Judging from earlier daytime studies of children and adults doing the same cognitive tasks while exposed to noise, children are not more sensitive than adults to noise (Boman, Enmarker & Hygge, 2004), but they perform at a lower level than the adults both in noise and in quiet. Thus, it could be said that children are not more vulnerable to (daytime) noise in relation to cognitive performance, but since so much more of cognitive work is expected from children while in school, their learning environment and their cognitive tasks can be said to more noise vulnerable than corresponding environments for adults.

7.4 Noise and aftereffects

An argument can be made for noise as a stressor inducing reduced motivation (Glass & Singer, 1972), which in turn may act as a mediator of impaired cognitive performance. Along this line of reasoning, nighttime noise may be more potent in inducing reduced motivation than daytime noise is, but for the time being this is only a conjecture and has not been tested.

7.4.1 The role of restoration

Noise can be viewed both as a source of stressful demands and as a constraint on restoration. Noise levels and noise sources that are not by themselves particularly demanding during the awake hours of the day, may never the less be quite effective in blocking and constraining when they appear in periods meant to be restorative, like sleep (Hartig, 2004). To what extent this idea is applicable to nighttime noise exposure has not yet been explored.

7.4.2 Noise and communication

Some of the difficulties with children's response to noise are related to problems in speech perception. A metric that weights night time exposure more heavily is, in fact, backwards since children's auditory processing with parents and teachers is obviously more critical during waking hours.

8 Effects on psychic disorders (*Stansfeld & Skalski)*

Noise exposure at night may be more disturbing than daytime noise because it interferes with rest and sleep at a time when people want to relax. It seems plausible that night time noise might have a particular effect on mental health. However, there is little direct research on night time noise and mental health and it is first necessary to consider the evidence for environmental noise and mental health in general. The association between noise and mental health has been examined using a variety of outcomes including (at the simplest level), individual symptoms, as well as psychiatric hospital admission rates, use of health services and psychotropic medication, and community surveys.

8.1 Transportation Noise and Mental health

Sources of transportation noise that have been studied in relation to mental health include

road traffic noise and aircraft noise. Studies relating to each type of noise will be considered in turn.

8.1.1 Road Traffic Noise

The association between road traffic noise exposure and psychological distress, has been studied in the small town of Caerphilly, South Wales. In the cross-sectional results, no association was found between the initial level of road traffic noise based on traffic noise maps, in terms of Leq referring to the period 6 a.m. to 10p.m., and minor psychological distress, measured by the General Health Questionnaire (GHQ), a screening questionnaire for depression and anxiety, even after adjustment for socio-demographic factors (Stansfeld et al, 1993). In longitudinal analyses in the Caerphilly Study, no association was found between road traffic noise and psychological distress, even after adjustment for socio-demographic factors and baseline psychological distress, although there was a small non-linear association of noise with increased anxiety scores (Stansfeld et al, 1996).

The disadvantage of the Caerphilly Study is that it relied on one location with not very high levels of traffic noise. In a secondary analysis of a large British road traffic noise study, which took into account multiple noise exposure sites, the noise level in dB(A) exceeded for 10% of the time was weakly associated with a 5-item mental health symptoms scale adjusting for age, sex, income and length of residence (Halpern, 1995). Weaker associations between traffic density and the mental health symptoms scale may relate to the skewed distribution of this traffic density variable. It seemed that traffic noise was more important than traffic flow. The scale used included some clear mental health items but also some that were less obviously related to mental health. It may be questioned whether the reported association between noise level and mental health symptoms was actually due to noise exposure; adjustment for the amount of 'noise heard' reduced the association very little, suggesting no causal association with noise, but it is likely that there was a good deal of error in the measurement of this variable, reducing its validity.

It may be that the peak noise level is a better indicator of environmental noise heard indoors than noise measures averaged over time and that peak levels are a crucial indicator for mental health. Furthermore, in a road traffic noise study in Belgrade, 253 residents exposed to road traffic noise levels of > 65dB(A), with high levels both day and night (Leq 76.5 in the day, 69.5 at night in the noise exposed area), experienced significantly more fatigue, depression, nervousness and headaches, compared to residents exposed to < 55dB(A) (Belojevic & Jakovlevic, 1997). Sleep quality was also found to be worse among the inhabitants of noisy streets, compared to inhabitants of quiet streets and those living in noisy streets had more difficulties falling asleep, more night awakenings and more pronounced tiredness after sleep. However, there were no differences in time to fall asleep, going back to sleep, duration of sleep or consumption of sleeping pills between noise exposed and non-exposed residents. A great methodological advantage of this study was that the high and low noise exposure areas were homogenous for age, sex, employment and subjective noise sensitivity. A community study in 366 Japanese women suggests that road traffic noise only has effects on depression, fatigue and irritability above a threshold of 70 dB(A) (Yoshida et al, 1997). However, it is difficult to be confident of the results of these analyses as they were unadjusted for age or social deprivation.

Milder psychological states such as health functioning and wellbeing have also been

examined in the first stage of an intervention study on the effect of introducing a by-pass to relieve traffic congestion in a small town in North Wales (Stansfeld et al, 2000). Health functioning was measured by the SF-36 General Health Survey (Ware & Sherbourne, 1992), including dimensions of general health status, physical functioning, general mental health and social functioning. Ninety-eight respondents were studied who lived on a busy high street with traffic noise levels varying between 72 and 75 dBA outdoor Leq. These respondents were compared with 239 control subjects living in adjacent quieter streets (noise level 55-63 dB(A) outdoor Leq). Although subjects were well-matched on age, sex, housing insulation, car ownership and employment status, they were not so well-matched on proportion of manual workers, household crowding, deprivation and home ownership. There was no evidence that respondents exposed to higher levels of road traffic noise had worse health functioning than those exposed to lower levels of the noise, adjusting for levels of deprivation.

Another method of assessing mental health effects related to noise exposure is to use an indirect indicator such as medication use. In five rural Austrian communities exposed to road traffic noise, noise levels above 55 dB(A), including increasing night time exposure to noise from trucks , were associated with increased risk of taking sleeping tablets (OR = 2.22 [CI 1.13- 4.38]) and overall prescriptions (OR = 3.65 [CI 2.13-6.26]) relative to road traffic noise exposure less than 55 dB(A) (Lercher,1996). This suggested effects at fairly low noise levels. In this case mental ill-health may be secondary to sleep disturbance, which is likely to occur at lower nocturnal noise levels than mental health symptoms resulting from daytime noise exposure. As this occurred in a rural setting where road traffic was the predominant source of noise it would be interesting to replicate these findings in other settings.

8.1.2 Road traffic noise and mental health in children

Noise exposure and mental health has also been studied in children where child self-reported mental health on a standard scale and teacher ratings of classroom adjustment in response to motorway, road and rail noise were measured in a large sample of 8-11 year-old Austrian primary school children and in a second-stage sample of extreme noise-exposed groups. Noise exposure was significantly associated with classroom adjustment scores but, intriguingly, child self-reported mental ill-health was only impaired in noisy settings for children of low birth weight and preterm birth (Lercher et al, 2002).

8.1.3 Aircraft Noise

Community surveys have found that high percentages of people reported 'headaches', 'restless nights,' and 'being tense and edgy' in high aircraft noise areas (Kokokusha 1973; Finke et al 1974; Öhrström 1989). An explicit link between aircraft noise and symptoms emerging in such studies raises the possibility of a bias toward over-reporting of symptoms (Barker and Tarnopolsky 1978). Notably, a study around three Swiss airports (Grandjean et al 1973), did not mention that it was related to aircraft noise and did not find any association between the level of exposure to aircraft noise and symptoms. In the West London Survey, 'tinnitus', 'burns, cuts and minor accidents', 'ear problems' and 'skin troubles' were all more common in areas of high-noise exposure (Tarnopolsky et al 1980). Acute symptoms like 'depression', 'irritability', 'difficulty getting off to sleep', 'night waking', 'skin troubles', 'swollen ankles', and 'burns, cuts and minor accidents' - were particularly common in high-noise areas. However, apart from 'ear problems' and 'tinnitus', 20 out of 23 chronic symptoms were more common in

low noise environments. Symptoms did not increase with increasing levels of noise. This is possibly related both to more social disadvantage and associated ill-health among residents in low aircraft noise exposure areas and the possible unwillingness of chronically unhealthy individuals to move into potentially stressful high-noise exposure areas. Nevertheless, it would not exclude an effect of noise in causing some acute psychological symptoms. As the majority of aircraft noise exposure is during the day, day time exposure is likely to have greater effects than night time exposure. Many of the effects of noise in industrial and teaching settings may be related primarily to disturbances in communication.

8.2 Noise Exposure and Mental Hospital Admission Rates

Much of the concern with the possible effects of noise on mental health began with the study of admissions to psychiatric hospitals from noisy areas. Early studies found associations between the level of aircraft noise and psychiatric hospital admissions, both in London (Abey Wickrama et al 1969) and Los Angeles (Meecham and Smith 1977). These results have been criticised on methodological grounds (Chowns 1970; Frerichs et al 1980) and a replication study by Gattoni and Tarnopolsky (1973) failed to confirm these findings. Jenkins et al (1979) found that age-standardized admission rates to a London psychiatric hospital over four years were higher as the level of noise of an area decreased, but lower noise areas were also central urban districts, where high admission rates would be expected. In a further extensive study of three hospitals (Jenkins et al 1981), high aircraft noise was associated with higher admission rates in two hospitals, but in all three of them, admission rates seemed to follow non-noise factors more closely; the effect of noise, if any, could only be moderating that of other causal variables but not overriding them. Kryter (1990), in a re-analysis of the data, found "a more consistently positive relation between level of exposure to aircraft noise and admissions rates." Undoubtedly, the route to hospital admission is influenced by many psychosocial variables that are more potent than exposure to noise. Therefore, whether or not noise causes psychiatric disorder is more suitably answered by studying a community sample.

8.3 Noise Exposure and Psychiatric Morbidity in the Community

In a community pilot study carried out in West London, Tarnopolsky et al (1978) found no association between aircraft noise exposure and either General Health Questionnaire (GHQ) scores (Goldberg 1972), (dichotomized 4/5, Low scorers/High scorers) or estimated psychiatric cases (Goldberg et al 1970). This was the case even when exposure to road traffic noise was controlled, except in three subgroups: persons "aged 15-44 of high education" (41%, 14% $p<0.05$), "women aged 15-44' (30%, 13% n.s.), and those in "professional or managerial occupations." The authors expressed the guarded opinion that noise might have an effect in causing morbidity within certain vulnerable subgroups.

In the subsequent West London Survey of Psychiatric Morbidity (Tarnopolsky and Morton-Williams 1980), 5885 adults were randomly selected from within four aircraft noise zones, according to the Noise and Number Index. No overall relationship was found between aircraft noise and the prevalence of psychiatric morbidity either for GHQ scores or for estimated numbers of psychiatric cases, using various indices of noise exposure. However, there was an association between noise and psychiatric morbidity in two subgroups: 'finished full time education at age 19 years +', and 'professionals'. These two categories, which had a strong association with each other, were combined and then showed a significant association between noise and psychiatric morbidity ($X2 = 8.18$, df 3 $p<0.05$), but only for the proportion

of high GHQ scorers. Tarnopolsky and Morton-Williams (1980) concluded that their results "show so far that noise per se in the community at large, does not seem to be a frequent, severe, pathogenic factor in causing mental illness but that it is associated with symptomatic response in selected subgroups of the population."

More recent studies have examined the effects of higher levels of military aircraft noise. Exposure to higher levels of military aircraft noise around the busy Kadena military airport in Japan was related in an exposure-effect association to depressiveness and nervousness measured by questionnaire, using the Todai health Index, based on the Cornell Medical Index (Ito et al, 1994; Hiramatsu et al, 1997). Mental health subscales included in this study measured depressiveness, nervousness, neurosis, and mental instability. Noise level was expressed as WECPNL (the power average of the maximum perceived noise exposure level in dB(A)) from 75-79, 80-84, 85-89, 90-94 and over 95). In unadjusted analyses, statistically significant differences were found in scores of depressiveness, nervousness, and neurosis between the non-noise exposed control group and the pooled group exposed to 75-95 WECPNL. In multivariate analysis adjusting for age, sex marital status, type of house and length of residence, noise exposure greater than 95 WECPNL was associated with higher scores on depressiveness and neurosis (Hiramatsu et al, 1997). Clear exposure effect relationships were not found between scale scores and noise exposure, as expressed in five unit steps. However, using more broadly defined groups, an exposure effect association was evident. This highlighted differences between the highest noise exposure group and lower exposure groups and indicated a threshold effect rather than a linear relationship - that mental health effects are more likely to be found at higher noise levels. In general, psychological rather than somatic symptoms were more related to noise in this study. Further analyses of the Japanese studies, suggest that high levels of military aircraft noise may have effects on mental health; in a cross sectional study of 5,963 inhabitants around two air bases in Okinawa, those exposed to noise levels of Ldn 70 or above had higher rates of 'mental instability' and depressiveness (Hiramatsu, 2000). Those who were more annoyed showed a higher risk of mental or somatic symptoms. A further survey using similar methodology on 6,486 respondents found exposure-effect associations between aircraft noise exposure, nervousness and mental health (Miyakita et al, 1998). These are important studies because of the opportunity to examine the effect of high noise exposure levels and the probability that out-migration of vulnerable persons from noisy areas biasing the sample was small.

The use of health services has also been taken as a measure of the relationship between noise and psychiatric disorder. Grandjean (1973) reported that the proportion of the Swiss population taking drugs was higher in areas with high levels of aircraft noise and Knipshild and Oudshoorn (1977) found that the purchase of sleeping pills, antacids, sedatives and antihypertensive drugs all increased in a village newly exposed to aircraft noise, but not in a 'control' village where the noise level remained unchanged. In both studies, there was also an association between the rate of contact with general practitioners and level of noise exposure. In the Heathrow study (Watkins et al, 1981), various health care indicators were used - use of drugs, particularly psychiatric or self?prescribed, visits to the general practitioner, attendance at hospital, and contact with various community services - but none of these showed any clear trend in relation to levels of noise. A recent study found that the use of sleeping tablets and sedatives was elevated with increasing night time noise exposure, especially in the elderly (Passchier-Vermeer, 2002). This has been judged to be 'sufficient' evidence of a noise effect (Health Council of the Netherlands, 2004).

8.4 Aircraft noise exposure and mental health in children

Poustka et al (1992) studied the psychiatric and psychosomatic health of 1636 children aged 4 to 16 in two geographical regions that differed according to the noise made by jet fighters frequently exercising at low altitude. Psychological and neurological outcomes were not related to noise exposure. They found that associations between noise exposure and depression and anxiety could be demonstrated, but only beneath the threshold of clinical significance. These results are less convincing because the areas differed socio-economically and the results were not adjusted for these factors and also because of lack of precision of the measures of noise exposure. However, in Munich, children living in areas exposed to high aircraft noise had lower levels of psychological well-being than children living in quieter environments (Evans et al, 1995). The longitudinal data from around Munich showed that after the inauguration of the new airport, the newly noise-exposed communities demonstrated a significant decline in self-reported quality of life measured on the Kindl scale, after being exposed to the increased aircraft noise for 18 months (third wave of testing), compared with a control sample (Evans et al, 1998). Impairment of 'quality of life' is a less severe disturbance than impairment of mental ill-health. Further studies have examined the effects of noise on child psychiatric disorder.

Chronic aircraft noise exposure was not associated with anxiety and depression (measured with psychometrically valid scales), after adjustment for socioeconomic factors, in the Schools Health & Environment Study around Heathrow airport (Haines et al, 2001a). In a further larger study of children's health around Heathrow Airport - the West London Schools Study (Haines et al, 2001b) - an association was found between aircraft noise exposure level and increased hyperactivity scores on the hyperactivity subscale of the Strength & Difficulties Questionnaire (Goodman, 1997). These studies suggest that noise influences child mental health, in terms of hyperactivity and that it may affect child stress responses and sense of well-being.

8.5 Neighbourhood noise and mental health

Noise from neighbours is the commonest source of noise complaints to local authorities in the UK (Chartered Institute of Environmental Health, 1999). Noise which is continuous and apparently indefinite, of uncertain cause or source, which is emotive or frightening, or apparently due to thoughtlessness or lack of consideration is most likely to elicit an adverse reaction (Grimwood, 1993). In the 1991 BRE survey, people most objected to barking dogs, banging doors, noise from radio, television, or hi-fi and human voices (Grimwood, 1993). In this survey, two types of emotional response to noise were observed: outwardly directed aggression, characterized by feelings of annoyance, aggravation, bitterness and anger towards the source of the noise, and a more emotional response of tension, anxiety and feelings of pressure. These responses are reminiscent of the distinction between internalising and externalising disorders. Whether noise from neighbours can induce psychiatric disorder has been little studied in community research, but this is area that deserves further study (Stansfeld et al 2000).

Undoubtedly, prolonged exposure to noise can be very upsetting, intrusive and interfering for sleep and everyday activities. In poorly built dwellings, especially apartments, even low-intensity noises may be clearly audible through walls, floors, or ceilings (Raw & Oseland, 1991). In this situation, noise is destructive of privacy, especially for those living alone, and

may be associated with perceptions of threat or increase a sense of isolation. This may be especially the case among people who are chronically anxious and likely to complain of sensitivity to noise; prolonged noise exposure may make them more anxious and unhappy. Often, this leads to arguments with neighbours, leading to a breakdown of neighbourly relationships and further isolation which may well in itself have a bad effect on mental health. Occasionally, this may be a sign of feelings of persecution associated with psychotic illness in which noise exposure is just an external trigger of an internally generated condition.

8.6 Mechanisms for casual links between noise and mental health

What might the mechanism be for the effects of noise on mental health? One way to approach this is through the effects of noise on cognitive performance where the laboratory evidence of effects is fairly robust (Smith and Broadbent, 1992). Effects of noise on mental health might be expected, because there is evidence that noise impairs other aspects of human functioning such as performance (Loeb 1986) and sleep, that are important in maintaining normal functioning and that noise causes adverse emotional reactions such as annoyance. In general, it seems that noise exposure increases arousal, and decreases attention through distraction (Broadbent 1953), increases the need for focusing attention to cut out irrelevant stimuli (Cohen and Spacapan 1978), as well as altering choice of task strategy (Smith and Broadbent 1981). Even relatively low levels of noise may have subtle ill-effects, and in this respect, the state of the person at the time of performance may be as important as the noise itself (Broadbent 1983). Individuals' perception of their degree of control over noise may also influence whether it impairs memory (Willner and Neiva 1987) while perception of lack of control over environmental conditions may be an important mediator of health effects.

Additionally, noise may also affect social performance as: (1) a stressor causing unwanted aversive changes in affective state; (2) by masking speech and impairing communication; and 3) by distracting attention from relevant cues in the immediate social environment (Jones et al 1981). It may be that people whose performance strategies are already limited for other reasons (e.g. through high anxiety) and who are faced with multiple tasks may be more vulnerable to the masking and distracting effects of noise.

The mechanism for the effects of noise on health is generally conceptualised as fitting the stress- diathesis model, in which noise exposure increases arousal, and chronic exposure leads to chronic physiological change and subsequent health effects. It is not clear though whether this model is appropriate for mental health effects. A more sophisticated model (Biesiot, 1989; Passchier-Vermeer, 1993) incorporates the interaction between the person and their environment. In this model, the person readjusts their behaviour in noisy conditions to reduce exposure. An important addition is the inclusion of the appraisal of noise (in terms of danger, loss of quality, meaning of the noise, challenges for environmental control, etc) and coping (the ability to alter behaviour to deal with the stressor). This model emphasises that dealing with noise is an active not a passive process.

8.7 Habituation to noise and mental health

It is likely that mental health effects arise from persistent exposure to noise over a long period of time. But do people habituate or adapt to noise over time? In some studies people do seem to adapt to noise and no longer notice noise that they are frequently exposed to. On the other hand in some studies of annoyance there seems to be little evidence of adaptation (Weinstein

et al, 1981). It may be that, as in physiological studies, a failure of adaptation occurs if the stimulus is novel, salient or implies threat. The development of mental health symptoms implies a failure to habituate to noise, or at least to adapt to noise. In some studies control over noise or active coping with noise rather than passive emotion-focussed coping is related to lower levels of symptoms (van Kamp, 1989). Habituation has not been formally studied in relation to noise and mental health.

8.8 Risk groups for mental health effects of noise

One way to look at susceptibility to noise is to think about groups in the population who may be more susceptible to noise- for instance people with existing physical or mental illness tend to be more highly annoyed by noise and potentially could be vulnerable to mental health effects. Similarly, people with hearing impairment may be vulnerable to communication difficulties in noisy environments that could increase the risk of mental health symptoms. People who report that they are sensitive to noise tend to be more prone to noise annoyance and may be more at risk for common mental disorders (Stansfeld et al, 2002).

8.9 Population groups at risk for mental health effects from noise

There is some evidence that children are more vulnerable to the mental health effects of noise than adults in terms of prematurity, low birth weight and through scoring higher on hyperactivity. There is no consistent evidence of age, social class, ethnic or gender differences in susceptibility to mental health effects from environmental noise.

8.10 Noise Sensitivity

Noise sensitivity, based on attitudes to noise in general (Anderson 1971; Stansfeld 1992), is an intervening variable which explains much of the variance between exposure and individual annoyance responses (Weinstein 1978; Langdon et al 1981; Fields 1994). Individuals who are noise-sensitive are also likely to be sensitive to other aspects of the environment (Broadbent 1972; Weinstein 1978; Thomas and Jones 1982; Stansfeld et al 1985a). This raises the question as to whether noise-sensitive individuals are simply those who complain more about their environment. Certainly, there is an association between noise sensitivity and neuroticism, (Thomas and Jones 1982; Öhrström, et al 1988b; Jelinkova 1988; Belojevic & Jakovlevic, 1997; Smith 2003), although it has not been found in all studies (Broadbent 1972). On the other hand, Weinstein (1980) hypothesized that noise sensitivity is part of a critical/uncritical dimension, showing the same association as noise sensitivity to measures of noise, privacy, air pollution, and neighbourhood reactions. He suggested that the most critical subjects, among whom noise-sensitive people would be grouped, are not uniformly negative about their environment, but more discriminating than the uncritical group, who comment uniformly on their environment.

Noise sensitivity has also been related to current psychiatric disorder (Bennett 1945; Tarnopolsky and Morton-Williams 1980; Iwata 1984). Stansfeld et al (1985a) found that high noise sensitivity was particularly associated with phobic disorders and neurotic depression, measured by the Present State Examination (Wing 1974). Similar to this association with phobic symptoms, noise sensitivity has also been linked to a coping style based on avoidance, which may have adverse health consequences (Pulles et al 1988) and a tendency to report health complaints rather than take a more active coping approach to noise (Lercher & Kofler, 1996). Noise sensitivity may be partly secondary to psychiatric disorder: depressed patients

followed over four months became less noise-sensitive as they recovered (Stansfeld 1992). These 'subjective' psychological measurements were complemented by an 'objective' psychophysiological laboratory investigation of reactions to noise in a sub-sample of depressed patients. Noise-sensitive people tended to have higher levels of tonic physiological arousal, more phobic and defence/startle responses and slower habituation to noise (Stansfeld 1992). Thus, noise-sensitive people attend more to noises, discriminate more between noises, find noises more threatening and out of their control, and adapt to noises more slowly than people who are less sensitive. Through its association with greater perception of environmental threat and its links with negative affectivity and physiological arousal, noise sensitivity may be an indicator of vulnerability to minor psychiatric disorder, although not necessarily psychiatric disorder caused by noise (Stansfeld 1992).

In analysis of a subset of noise-sensitive women, compared to less sensitive women in the West London survey, there was no evidence that aircraft noise exposure predicted psychiatric disorder in the sensitive women (Stansfeld et al, 1985a). In the Caerphilly study, noise sensitivity predicted psychological distress at follow-up after adjusting for baseline psychological distress, but did not interact with the noise level, suggesting that noise sensitivity does not specifically moderate the effect of noise on psychological distress (Stansfeld et al 1993). However, in further analyses, a statistically significant association between road traffic noise exposure and psychological distress, measured by the General Health Questionnaire, was found in noise-sensitive men that was not found in men of low noise sensitivity (Stansfeld et al, 2002). In the original analyses, after adjusting for trait anxiety at baseline, the effect of noise sensitivity was no longer statistically significant. This suggests that much of the association between noise sensitivity and psychological distress may be accounted for by the confounding association with trait anxiety. Constitutionally anxious people may be both more aware of threatening aspects of their environment and more prone to future psychiatric disorder. It seems possible that these traits might be linked.

In a UK community study, associations were examined between noise exposure, noise sensitivity, subjective symptoms, and sleep disturbance in a random sample of 543 adults (Smith et al, 2000). Perceived noise exposure was related to subjective health, but this association became non-significant after adjustment for negative affectivity. In a similar way, adjustment for negative affectivity eliminated the association between noise sensitivity and subjective health. Thus, it was suggested that noise sensitivity was merely a proxy measure of negative affectivity or neuroticism. However, although this means that noise sensitivity is not specific to noise, the more recent analyses suggest that high levels of trait anxiety or neuroticism may be an indicator of vulnerability to noise effects and could put people at risk of adverse psychological effects from noise, even if they do not increase the risk of physical ill-health.

8.11 Mental health consequences of insomnia

Transient insomnia is usually accompanied by reports of daytime sleepiness and performance impairment the next day. Chronic insomnia is generally associated with poorer emotional and physical health. Several large-scale epidemiological studies of the general adult population have shown that between one third and one half of people who complain of chronic insomnia are also diagnosable with primary psychiatric disorders, mostly anxiety and mood disorders. Mellinger et al. (1985) found that 17% of adults reported "a lot" of trouble falling asleep or staying asleep over past year; 47% of them had high levels of psychological distress, with

symptom complexes suggestive of depression and anxiety disorders. In contrast, only 11% of individuals with no history of insomnia showed elevated levels of psychiatric symptoms. In survey of almost 8000 individuals, Ford and Kamerow reported that 10% suffered from significant insomnia for at least a 2-week period during the previous 6 months; 40% of the insomniacs met criteria for psychiatric disorders, with the majority being anxiety disorders and depression; only 16% of those with no sleep complaints had psychiatric illness.

Breslau et al. (1996) found a strong correlation between lifetime prevalence of sleep problems and psychiatric disorders, with anxiety, depression, and substance abuse disorders being the most common. Similar results have been found by Vollrath et al. (1989), Chang et al. (1997), Dryman and Eaton (1991). In a large-scale European population-based study (Ohayon, Roth 2003), it was found that insomnia more often precedes rather than follows incident cases of a mood disorders.

Insomniacs not only have higher rates of psychiatric disorders, but they also have increased rates of various kinds of psychological symptoms: patients with insomnia reported increased psychological stress and/or decreased ability to cope with stress according to surveys of the American (Roth and Ancoli-Israel 1999) and Japanese (Kim et al. 2000) population. Almost 80% of insomniacs had significant increase on one or more clinical scales on the MMPI (Kalogjera-Sackellares, Cartwright 1997). Even people whose insomnia was due to identified medical factors showed elevation on the MMPI, suggesting a possible causal relationship or specific association between insomnia and psychiatric symptomatology. Compared to good sleepers, severe insomniacs reported more medical problems, had more physician-office visits, were hospitalized twice as often, and used more medication. Severe insomniacs had a higher rate of absenteeism, missing work twice as often as did good sleepers. They also had more problems at work (including decreased concentration, difficulty performing duties, and more work-related accidents) (Leger et al, 2002).

8.12 Insomnia as a mental health symptom

Insomnia is a symptom of many psychiatric disorders, especially depression and anxiety. In studies of depressed patients compared to control subjects, there was prolonged latency to sleep, increased wakefulness during sleep, early morning wakening, decreased sleep efficiency and reduced total sleep time. There is also evidence that insomnia may be a risk factor for developing depression (Riemann et al, 2001; Roberts et al, 2002). This raises the question whether prolonged noise exposure leading to insomnia provokes the onset of depression in susceptible people? This seems theoretically possible, but there is little evidence to support it. In a longitudinal study of adolescents, it was the other way round - that depressive symptoms preceded the onset of insomnia (Patten et al, 2000). Delayed sleep latency in children has been linked to increased externalising symptoms including aggressive behaviour, and impaired attention and social problems (Aronen et al, 2000). In this cross-sectional study, the direction of association was uncertain, but it seems most plausible that the sleep disturbance is a feature of the behavioural disturbance rather, than a cause of it. Three criteria have been suggested for sleep disturbance to be environmentally determined: (1) the sleep problem is temporally associated with the introduction of a physically measurable stimulus or definable set of environmental circumstances; (2) the physical rather than the psychological properties of the environmental factors are the critical causative elements; and (3) removal of the responsible factors results in an immediate or gradual return to normal sleep and wakefulness (Kraenz et al, 2004). Most studies do not fulfil these criteria. In a German school-based study of 5-6 year-old children, sleep disturbance by noise, largely

from road traffic, was reported 'sometimes' in 10% by parents of children and 2% 'often'. Children's reports were slightly higher: 'sometimes' in 12% and 3% 'often' (Kraenz et al, 2004). Further longitudinal research is needed to ascertain whether noise-induced insomnia leads on to overt psychiatric disorder.

In summary, population as well as clinic-based studies have demonstrated a high rate of psychiatric morbidities in patients with chronic insomnia. It has traditionally been assumed that insomnia is secondary to the psychiatric disorders; however it is possible that in some cases the insomnia preceded the psychiatric disorder.

8.13 Depressive episode and Anxiety Disorders

A mild depressive episode is diagnosed by clinical interview. The criteria for mild depressive episode include two or more symptoms of depressed mood, loss of interest or fatigue lasting at least two weeks, with two or three symptoms such as reduced concentration, reduced self-esteem, ideas of guilt, pessimism about future, suicidal ideas or acts, disturbed sleep, diminished appetite and social impairment and fewer than four symptoms including lack of normal pleasure/interest, loss of normal emotional reactivity, waking =>2 hours early, loss of libido, diurnal variation in mood, diminished appetite, loss of =>5% body weight, psychomotor agitation or psychomotor retardation.

Anxiety disorders are similarly diagnosed by clinical interview. The criteria for Generalised Anxiety Disorder include duration of at least 6 months of free-floating anxiety and autonomic overactivity.

8.14 Associations between insomnia and psychiatric disorders

At the present time, exposure-effect associations have not been established between parameters of sleep disturbance (number of behavioural awakenings, body movements or EEG awakenings) and the onset of depressive and anxiety disorders although there is some evidence that insomnia is a risk factor for developing depression (Riemann et al, 2001; Roberts et al, 2002). A number of longitudinal prospective studies in different age groups have found associations between self reports of insomnia and the subsequent onset of psychiatric disorder, in particular major depression. A selection of the most important studies and their findings are outlined in table IV.7.1 below.

Table IV.7.1 Insomnia as a predictor of psychiatric disorder

Study	Sample size	Sample	Follow up interval	Depression measure	Results
Ford & Kamerow, 1989	7954	Community sample	1 year	Diagnostic Interview Schedule	Risk of developing new depression for insomnia on 2 occasions:OR=39.8, 95% CI 19.8-80.0

Breslau et al, 1996	1200	21-30 years members of health maintenance organisation	3 years	Diagnostic Interview Schedule	RR for new onset major depression associated with baseline insomniaRR=4.0, 95%CI 1.5-5.6
Chang et al, 1997	1,053	Male medical students	34 years (median)	Clinical Depression	RR for clinical depression for those who reported insomnia at medical schoolRR= 2.0, 95%CI 1.2-3.3
Roberts et al, 2002	3,136	11-17 years from managed care rosters	1 year	Diagnostic Interview Schedule for Children Major Depression module	Fully adjusted OR for insomnia in waves 1 & 2 for depression at follow upOR=1.92, 95% CI 1.30-2.82

8.15 Conclusions: associations between noise and psychiatric disorders

The effects of noise are strongest for those outcomes that, like annoyance, can be classified under 'quality of life' rather than illness. What they lack in severity is made up for in numbers of people affected, as these responses are very widespread. Current evidence does seem to suggest that environmental noise exposure, especially at higher levels, is related to mental health symptoms and possibly raised anxiety and consumption of sedative medication, but there is little evidence that it has more serious effects. Further research is needed on mental health effects at very high noise levels. Existing studies may be confounded either by prior selection of subjects out of (or into) noisy areas as a result of noise exposure, or by confounding between noise exposure, socio-economic deprivation, and psychiatric disorder. It is also possible that people underestimate or minimise the effects of noise on health through optimism bias (Hatfield & Job, 2001) and that this is particularly protective for mental health.
The evidence is not strong for the association between noise exposure and mental ill-health. What evidence there is suggests that noise exposure may be responsible for psychological symptoms above 70 dBA Leq. Almost all studies have only examined the effects of day time noise on mental health, but it is possible that night time noise, during sleep time, may have effects on mental health at lower levels than day time noise.

The most powerful evidence of noise on mental health comes from studies of military aircraft noise. There is also some evidence that intense road traffic noise may lead to psychological symptoms. There is no evidence of any effects of railway noise on mental health.

9 The severity of self-reported sleep disturbance *(Mueller-Wenk)*

9.1 Introduction

In section II.1.2 of this report, it is stated that sleep disturbance caused by noise may either be diagnosed (Environmental Sleep Disorder: ICSD 780-52-6) or self-reported. Although self-reported sleep disturbance is subjective by definition, its observed occurrence correlates with noise levels as well as with important diagnostic criteria for ICSD 780-52-6. It appears justified to consider self-reported sleep disturbance as an impairment of health, especially if indicated by representative population samples in social surveys. Further, section IV.1 of this report gives a quantitative relationship between noise level L_{night} and the percentage of population that reports a disturbed sleep of high, medium or low disturbance intensity.
But an open question concerns severity: Even if night-time noise causes large percentages of population to declare themselves as highly sleep-disturbed, this could nevertheless represent an almost negligible loss of health, if the mean severity of self-reported sleep disturbance were negligible in comparison with commonly accepted diseases. Attempts have been made to give an answer to this important question, using WHO's concept of disability weights (Murray 1996) as a basis for severity comparisons.

9.2 An assessment of disability weights

A Swiss study (Müller-Wenk 2002) aimed at determining a disability weight for sleep disturbance due to road traffic noise. For this purpose, a description of road-noise-related sleep disturbance was set up: Essentially, this state of health was assumed to be present if a person indicated that, due to traffic noise, he or she had almost every night problems with falling asleep or with continuing sleep during the night or with early or non-restorative waking up in the morning. In addition, a list was established with already available disability weights (Murray 1996; Stouthard 1997) for a selection of 28 diseases of various types, covering a range from very light severity to high severity (Müller-Wenk 2002:65-66). All 64 members of the medical staff of the Swiss Accident Insurance Institute (SUVA) were then asked in a written questionnaire to determine the hitherto unknown disability weight of sleep disturbance by interpolation, i.e. by inserting sleep disturbance at the appropriate place between the presented 28 diseases that were sorted according to ascending disability weight. These participants were chosen because the physicians of the SUVA, besides of being medical doctors, have a particularly high professional know-how in comparing the severity of different types of disability. 42 questionnaire were completed, whereof 41 were usable.

From these questionnaires, an arithmetical mean of 0.055 of the disability weight for sleep disturbance could be calculated, with a 95% confidence limit of 0.039 at the low end and 0.071 at the high end. This result can be illustrated by mentioning diseases from the catalogues of Murray (1996) or Stouthard (1997) with the same disability weight: Hence the disability weight of the road-noise-related sleep disturbance is roughly the same as the disability weight of 'chronic hepatitis B infection without active viral replication', the latter having a mean disability weight of 0.06 and a 95% confidence interval from 0.034 to 0.087. The low end estimate of 0.039 for sleep disturbance severity would correspond to the mean disability weight of 'Benign prostatic hypertrophy (symptomatic cases)', whilst the high estimate of 0.071 would correspond to the mean disability weight of 'uncomplicated diabetes mellitus'. The conclusion is that the mean disability weight of road-noise-related sleep disturbance is not smaller (= less severe) than the disability weight of health impairments commonly recognised as diseases, and there is a strong overlapping amongst the probability

distributions of these disability weights. On the basis of the chosen disabilty weight 0.055 for self-reported sleep disturbance, and taking into account the current traffic noise levels during night in many European states, it is justified to consider noise-related sleep disturbance as a substantial loss of public health.

9.3 Comparison between insomnia and self-reported sleep disturbance

The original list of disability weights (Murray 1996) did not contain any kind of non-normal sleep. In the meantime, WHO has published an extended list (Mathers et al 2004, Annex table 5a), containing a disability weight of 0.100 for insomnia (diagnostic code 307.42). This has opened a way to recheck the disability weight of 0.055 (Müller-Wenk 2002), by asking a panel of medical professionals to compare, on the basis of disability weights, the mean severity of self-declared sleep disturbance due to road noise at night with the mean severity of insomnia. It may be debated whether it is more straightforward to compare two types of sleep anomalies with similar symptoms, or to compare self-declared sleep disturbance with various types of completely different diseases. But it anyway makes sense to use the comparison with insomnia as a second approach for determining the disability weight of self-reported sleep disturbance.

This severity comparison between different sleep anomalies was made in year 2005 by structured oral interviews, executed by a medical staff member of the sleep clinic of Kantonsspital St.Gallen (Switzerland), with 14 general practitioners selected at random from all general practitioners who had admitted patients to the sleep clinic during the 9 precedent months. These patients were mainly suffering from OSAS (Obstructive Sleep Apnea Syndrome). The question was as follows:
"Could you please give us your opinion on the relative severity of three different cases of insomnia:

1. (Primary) Insomnia, in our region usually called psycho-physiological insomnia
2. Obstructive Sleep Apnea Syndrome (OSAS)
3. Traffic noise related sleep disturbance, that may occur with persons who are forced to sleep along through roads with nocturnal motor traffic

Your opinion should be based on the patients you have seen in your office lately, or on other persons of your social environment. When comparing the severity of the health impairment, the focus should be above all on the person's condition during the day after the sleep-disturbed night. The absolute value of the severity is less important for the current study than the relative severity amongst the 3 cases of insomnia. The opinion of the severity may be expressed on a linear scale from 0 (no impairment at all) to 10 (impairment almost unsupportable). On the scale from 0 to 10, you may give us your mean value of the severity, or you may give us a span from a low to a high for the severity."

All of the interviewed general practicioners gave their opinions, and the result is presented in table IV. below.

Table IV.: Severity ratings (10= almost unsupportably disturbing, 0= not in the least disturbing) by 14 general practitioners selected at random

Discussion: It is clearly visible that the severity judgements vary widely between the participating general practitioners. Apart from the differences in personal judgement, this variation is certainly influenced by the mix of patients visiting a particular general practitioner: For instance, general practitioner number 15 could have encountered one or two

	Primary insomnia				OSAS (sleep apnea)				Sleep disturb.(noise)				ratio noise/	Noise/
No	Max	Min	Mean	Rank	Max	Min	Mean	Rank	Max	Min	Mean	Rank	prim insomnia	OSAS
10	6	4	5	3	8	6	7	1	8	6	7	1	1.40	1.00
11	5	3	4	3	9	7	8	1	8	4	6	2	1.50	0.75
12			5	3			10	1	7	8	7.5	2	1.50	0.75
13	2	3	2.5	2	4	5	4.5	1	1	2	1.5	3	0.60	0.33
14			3	2			6	1	1	2	1.5	3	0.50	0.25
15			8	2			9	1			6	3	0.75	0.67
16			8	1			7	2			4	3	0.50	0.57
17			5	1			5	1			3	3	0.60	0.60
18	2	3	2.5	2			6	1	1	2	1.5	3	0.60	0.25
19			8	1			3	2			2	3	0.25	0.67
20			6	2			7	1			4	3	0.67	0.57
21			7	2			8	1			0	3	0.00	0.00
22			4	3			5	2			6	1	1.50	1.20
23			4	3	6	7	6.5	2	8	9	8.5	1	2.13	1.31
Mean			**5.143**	**2.143**			**6.57**	**1.286**			**4.18**	**2.429**	**0.89**	0.64
Sigma													**0.60**	
Median													**0.63**	
Upper value 95% C.I.for mean													**1.20**	
Lower value 95% C.I. for mean													**0.58**	

very serious cases of OSAS, whilst his experience with noise-related sleep disturbance might refer to persons that were only moderately disturbed by night-time noise in their bedroom. On the other hand, number 22 could have had experience with persons suffering very much from sleep disturbance due to high traffic noise exposure, whilst his OSAS or primary insomnia patients happened to be light cases. We have to accept that even general practitioners do have a limited experience with the whole range of cases of each of the three types of insomnia, so that their opinion on the mean severity of noise-related sleep disturbance, compared to the mean severity of OSAS or insomnia, is influenced by the randomness of their patients mix.

Nevertheless, the table supports the following statements:

1. With respect to the severity, the majority of general practitioners is ranking noise-related sleep disturbance lower than insomnia and OSAS, whilst three of them put noise-related sleep disturbance in the first rank. Only one of the participants (number 21) considers noise-related sleep disturbance as a fully negligible disturbance.
2. The severity ratio between noise-related sleep disturbance and insomnia varies between 0 and 2.1. Seven of the 14 general practitioners indicate a severity ratio between 0.5 and 0.75, that is to say that half the participants are of the opinion that the severity of noise-related sleep disturbance amounts to 50-75% of the severity of insomnia.
3. The mean of this severity ratio is 0.89, with a standard deviation (sigma) of 0.60. The confidence interval CI for the mean goes from 0.58 to 1.20. The median of the severity ratio is 0.63. The distribution is skewed to the right.

The severity ratio developed above can be used as a proportionality factor between the known disability weight for insomnia and the required disability weight for self-reported sleep disturbance. Bearing in mind that the already existing WHO disability weight for insomnia is 0.10, a best guess for the mean disability weight for self-reported sleep disturbance due to road traffic noise at night is therefore 0.089, with a CI from 0.058 to 0.12.

9.4 Conclusions

1. According to the two groups of interviewed medical professionals, persons that declare themselves to be chronically deprived of normal sleep by road traffic noise, have a health state whose mean disability weight is comparable to 'chronic hepatitis B infection without active viral replication', or higher. Irrespective of the question whether self-reported sleep disturbance is formally recognised as a disease or not, its severity is comparable to commonly accepted diseases.
2. The best estimate for a mean disability weight for self-reported sleep disturbance due to road traffic noise was 0.055 (CI 0.039; 0.071) according to (Müller-Wenk 2002), whilst our recheck based on a comparison with insomnia resulted in a disability weight of 0.09 (CI 0.06; 0.12). The higher disability weight according to the second approach might be caused by the fact that in this second approach, there was a stronger focus on " the person's condition during the day after the sleep-disturbed night".
3. The above figures compare reasonably with a study published by Van Kempen 1998, cited in Knol&Staatsen (2005:46), where a severity weight of 0.10 for severe sleep disturbance was found, basing on the judgement of 13 medical experts enquired according to the protocol of Stouthard (1997).
   In conclusion, we propose a mean disability weight of 0.07 for self-reported sleep disturbance due to road noise or similar ambient noise. This disability weight can be used in connection with the equations of section IV.1 for highly sleep disturbed persons.

10 Discussion: Can chronic short-term effects cause long-term effects on health?

EEG modifications, cardiovascular responses, body movements and awakenings due to noise occur within a few seconds after the stimulus. In addition to the instantaneous effects related to single events, large field studies on aircraft (Passchier-Vermeer et al., 2002) and road traffic noise exposure during night-time (Griefahn et al., 2000, Passchier-Vermeer et al., 2004) show that also sleep latency and average motility during the sleep period increased monotonously as a function of the noise exposure level. The increase in average motility was substantially higher than would be expected on the basis of the instantaneous extra motility at the times of the noise events (Passchier-Vermeer et al., 2002) suggesting persistent arousal during the sleep related to aircraft noise. Furthermore, an international field study (Jurriëns et al., 1983) found slightly reduced REM-sleep, increased time being awake according to the EEG, increased average heart rate, and reduced performance on a reaction time test in people when exposed during the night to higher road traffic noise levels.
The relationship between instantaneous effects and more global modifications of one night sleep as well as chronic changes, is not simple as illustrated by the findings concerning motility. An increase in average motility that is substantially higher than would be expected on the basis of the instantaneous extra motility at the times of the noise events (Passchier-Vermeer et al., 2002) suggests a persistent arousal during sleep related in a

dose-dependent way to the aircraft noise.
Since EEG arousal and instantaneous motility are correlated, this finding suggests that also the number of (micro-) arousals may increase during noise exposure more than by the sum of the instantaneous (micro-)arousals that occur contingent upon a noise event.
For overall motility during sleep, clear indications have been found of associations with further effects, although the causal direction is not in all cases clear. Mean (onset of) motility during sleep is associated with the following variables based on questionnaires and diaries (Passchier- Vermeer et al., 2002):
• frequency of conscious awakening during the sleep period. The increase is 0.8 conscious awakenings per night, if motility increases from low to high;
• frequency of awakening remembered next morning. The increase is 0.5 remembered awakenings per night, if motility increases from low to high;
• long-term frequency of awakening attributed to specific noise sources assessed with a questionnaire;
• sleep quality reported in a morning diary;
• long-term sleep quality assessed with a questionnaire;
• number of sleep complaints assessed with a questionnaire;
• number of general health complaints assessed with a questionnaire.
The associations of mean motility with these variables are stronger than the corresponding associations of mean onset of motility.
For evaluating the adverseness of the instantaneous effects, it is important to consider whether they bring the body in a more persistent state of higher arousal or not, although this is not the only criterion. Those effects which are progressively disappearing with the repetition of the stimulus may be less harmful than those which do persist over long exposure time, provided that the suppression of the effects do not require costs in another form. For example, short-term cardiovascular effects that appear to not to habituate could lead to permanent cardiovascular system impairment (Carter, 1996; Carter, 1998).
The relations presented for motility and conscious awakening imply that motility is sensitive to noise and has a relatively low threshold, while conscious awakening, the strongest instantaneous interference of noise with sleep, has the highest threshold of the instantaneous effects considered.
In one of the most sophisticated field studies (Passchier-Vermeer et al (2002)), increased probability of instantaneous motility was found for events with a maximum sound level LAmax > 32 dB(A), while in a meta-analysis conscious awakening was found for events with L_{Amax} > 42 dB(A) (Passchier-Vermeer, 2003). Above their threshold, these effects were found to increase monotonously as a function of the maximum sound level during a noise event (aircraft noise). It is important to note that in another recent sophisticated field study (Basner et al., 2004), the threshold found for EEG awakening was LAmax = 35 dB(A), i.e., only a little higher than the 32 dB(A) found for noise-induced awakenings. This strengthens the evidence that noise starts to induce arousals at LAmax values in the range 30 to 35 dB(A). Given the night-time noise levels to which people are exposed, these results imply that instantaneous effects are common. Although most studies concerned aircraft noise, the instantaneous effects can be assumed to occur at similar levels for different types of transportation.
The above observations can be used as a basis for setting limits with respect to night-time transportation noise. For transparency, it is useful to distinguish two steps in choosing actual limits: the first step is the derivation of a health-based limit, the second step is the derivation of a actual limit that takes into account the health based limit as well as feasibility arguments. Here we are concerned with the first step.

When deriving a health based limit, two points need to be considered: the dose-dependent effects of a single noise event, and the number of events. With respect to the dose-dependent effects of a single event, adverse effects can be distinguished from effects that by themselves need not be adverse but can contribute to an adverse state. We propose to classify conscious awakenings as an adverse effect. Conscious awakenings have been estimated to occur at a baseline rate of 1.8 awakening per night. A substantial increment of conscious awakenings over this baseline is thought to be adverse. Since in general falling asleep after conscious awakening takes some time, and this latency is longer after noise induced conscious awakening that will often also induce an emotional reaction (anger, fear), it will also reduce the time asleep and may affect mood and functioning next day. Although additional, more sophisticated analyses could be performed to refine this estimate, we propose LAmax = 42 dB(A) as the currently best estimate of the threshold for conscious awakening by transportation noise. This would mean that the No Observed Effect Level (NOELAmax) for transportation noise events is at most 42 dB(A).

The most sensitive instantaneous effect that has been studied extensively in field studies is motility. A single interval with (onset of) noise induced motility by itself cannot be considered to be adverse. However, noise induced motility is a sign of arousal, and frequent (micro-)arousal and accompanying sleep fragmentation can affect mood and functioning next day and lead to a lower rating of the sleep quality. Therefore, motility is relevant for adverse health effects, but more than a few intervals with noise induced motility are needed for inducing such effects. Although additional, more sophisticated analyses could be performed to refine this estimate, we propose LAmax = 32 dB(A) as the currently best estimate of the threshold for motility induced by transportation noise. The threshold found for EEG awakening was LAmax = 35 dB(A), i.e., only a little higher than the 32 dB(A) found for noise-induced awakenings. This would mean that the No Observed Effect Level (NOELAmax) for transportation noise events is most likely at most 32 dB(A), and definitely not higher than 35 dB(A).

It is important to note that the above given NOELAmax ~32 dB(A) and NOELAmax ~ 42 dB(A) are indoor levels, in the sleeping room. Although events below 32 dB(A) are audible, and, hence, further research may show more sensitive effects than motility, on the basis of the presently available evidence we propose to assume that NOELAmax = 32 dB(A) and set a health based nighttime noise limit that is tolerant for transportation noise events with LAmax ~ 32 dB(A). On the other hand, since adverse health effects need to be prevented by health based limits and even though vulnerable groups may require lower limits, on the basis of the presently available evidence we propose to assume that NOAELAmax = 42 dB(A) and set a health based night-time noise limit that does not tolerate transportation noise events with LAmax > 42 dB(A).

On the basis of the above proposal, it would be possible to derive a night-time noise guideline value in terms of L_{night}. Such a guideline value would indicate the level below which no short term effects are to be expected that would lead to temporary reduced health or chronic disease. Such a guideline value need to be compared with guideline values derived directly with a view to preventing temporary reduced health and chronic diseases. In particular, for self-reported sleep disturbance, which is an expression of reduced well being and may be an indication of effects that could contribute to cardiovascular disease, exposure-effect relationships have been derived on the basis of an extensive set of original data from studies from various countries (Miedema et al., 2003; Miedema, 2004). The percentage of people reporting high noise induced sleep disturbance (%HS) levels off at 45 dB(A) but at a nonzero effect level. The remaining effect may be caused by events not incorporated in the exposure assessment and it appears that if all noise contributions would be incorporated in the exposure

metric, high noise induced sleep disturbance would vanish between 40 - 45 dB(A), say at 42 dB(A). Since values found for other temporary reduced health effects or chronic diseases, in particular cardiovascular diseases, will be higher, and considering self-reported sleep disturbance as an adverse effect, this would suggest L_{night} = 42 dB(A) as the NOAEL to be compared with the value derived from the short term effects. Note that this is an outdoor level, which would, assuming partly opened windows and an actual insulation of 15 dB(A), correspond to an indoor equivalent night-time sound level of 27 dB(A).

The above discussion is based on motility, EEG awakenings, and conscious awakening. In addition, EEG micro/minor-arousals, and autonomic reactions have been discussed above. Furthermore, there are potential instantaneous effects, such as effects on memory consolidation or restoration of the immune system, for which the information on a possible relation with noise exposure is so limited that they were not considered here. In order to acquire more insight in these effects, more field research is needed. Field research is needed because earlier studies have shown that estimates of effects on the basis of laboratory studies are much higher than estimates from field studies. Methodological differences between the different approaches certainly cannot be the only possible explanation. Research allowing the introduction of some specific but light laboratory technique into the sleepers own bedroom, should be encouraged as for example used in the Swiss Noise Study 2000 (Brink, M., Müller, C. H., & Schierz, C. (2006b). The key to better insight in effects of night-time noise, leading to mechanistic models describing the relationships between noise exposure, instantaneous effects, effects at the level of a 24 h period and chronic effects, appears to be epidemiological studies at home with well designed instrumentation.

The relationships between noise exposure, instantaneous effects, effects at the level of a single 24 h period, and chronic effects is complex because the effects at a smaller time scale do not simply add up to effects at a larger time scale. For example, the noise-related increase in night-time average motility was substantially higher than would be expected on the basis of the instantaneous extra motility at the times of the noise events (Passchier-Vermeer et al., 2002), suggesting persistent arousal during the sleep related to aircraft noise. It is likely that such shifts in the basic state are more important for the development of chronic effects than the instantaneous effects per se. A further complication is that some effects habituate. Habituation in some effect parameters can occur in a few days or weeks, but the habituation is not always complete. The measured modifications of the cardiovascular functions remain unchanged over long periods of exposure time (Muzet and Ehrhart, 1980; Vallet et al., 1983). Most striking is that none of the cardiovascular responses show habituation to noise after a prolonged exposure, while subjective habituation occurs within a few days. It appears plausible that in particular non habituating effects lead to the development of chronic effects, but also the disappearance of effects with continuing exposure may come at cost associated with suppressing the effects. A third complication is that daytime noise exposure may contribute to the effects found in relation to night-time noise. Large epidemiological studies are needed that compare populations exposed to similar daytime noise and differ in their night-time noise exposure only. A specific challenge for mechanistic models on the effects of noise on sleep is the identification of factors that make subjects vulnerable to night-time noise. The following groups may be hypothesized to be more vulnerable to noise during sleep: old people, ill people, people with chronic insomnia, shift workers and people resting during daytime, people with tendency to depression, light sleepers, pregnant women, people with high anxiety and high stress levels. Furthermore, children need attention because of their relatively high exposure during sleep, and because they are in a phase of neurocognitive development for which undisturbed sleep may be particularly important.

CHAPTER V. GUIDELINES AND RECOMMENDATIONS

Blessed are those drowsy ones: for they shall soon nod to sleep.–
Thus spoke Zarathustra

1 Assessment

In Chapter I the need for a guideline document for night time exposure was defended on the basis of the lack of guidance, the signs that a substantial part of the population could be exposed to levels that might risk their health and well being and the EU-activities that force the public and authorities to take position when noise maps showing L_{night} levels are made public.
If direct evidence on the effects of night noise on health could not sufficiently be collected, it was proposed to look at indirect evidence: the effects of noise on sleep(quality) and the relations between sleep and health.
In Chapter II the evidence is presented that sleep is a biological necessity, and disturbed sleep is associated with a number of health outcomes. Studies of sleep disturbance in children and in shift workers show clearly the adverse effects. Unravelling the relations between sleep and health (figure 9) show that sleep is an essential feature of the organism, so that simple direct relations can hardly be expected.
In chapter III it is shown beyond doubt that noise disturbs sleep by a number of direct and indirect pathways. Already at very low levels physiological reactions (heart rate, body movement, arousals) can be reliably measured. Also it was shown that awakening reactions are relatively rare, occurring at a much higher level.
Chapter IV summarizes the known evidence for the direct effects of night noise on health. For a number of effects there was found to be sufficient evidence: self reported sleep disturbance, use of pharmaceuticals, self reported health problems and insomnia. For other effects (hypertension, myocardial infarcts, depressions and others) limited evidence was found: although the studies were few or not conclusive, a biological plausible pathway could be constructed from the evidence presented in Chapter III. It was pointed out that the effects for which there was sufficient evidence present could by itself lead to a considerable burden of environmentally related disease in the population. Myocardial infarcts(MI) are an example of this. Although evidence for this relation between Lday and MI was recently promoted from limited to sufficient, this was not the case for L_{night}, simply because no separate night noise relation was studied. Evidence from other studies however point to the relative higher importance of night exposure; future studies might make this clear.

Overlooking the vast amount of evidence available it can be safely concluded that:
-sleep is a biological necessity, and disturbed sleep is associated with a number of health outcomes
-there is sufficient evidence for biological effects of noise during sleep: increase in heart rate, arousals, sleep stage changes, hormone level changes and awakening
- while this disturbance is viewed as a health problem in itself (environmental noise disturbance) it also leads to further consequences for health and well being.
- there is sufficient evidence that night time noise exposure causes: self reported sleep disturbance, increase in medicine use, increase in body movements, and (environmental) insomnia.
- there is limited evidence that diisturbed sleep causes fatigue, accidents and reduced performance the following day.
- there is limited evidence that noise at night causes clinical symptoms like cardiovascular

illness, depressions and other mental illness. It should be stressed that a plausible biological model is available with sufficient evidence for the separate elements and the direct relation is supported by animal experiments (see appendix III for an extensive report).

In the next section threshold levels are presented for the effects where these can be derived.

2 Thresholds for observed effects

The (no) observed adverse effect level (NOAEL) is a concept from toxicology, and is defined as the greatest concentration which causes no detectable adverse alteration of morphology, functional capacity, growth, development or life span of the target organism (WHO, 2000). For the topic of night noise (where the adversity of effects is not always clear) this concept is less useful. In stead the observed effect thresholds are provided: the level above which an effect starts to occur or shows to be dependant on the dose. This can be as well an adverse effect (like MI), or a potentially dangerous increase in a natural occurring effect like motility.
Threshold levels are important milestones in the process of evaluating the health consequences of environmental exposure. The threshold levels also delimit the study area, which may lead to a better insight into overall consequences. In the table all effects are summarized for which sufficient or limited (see introduction for a definition) evidence exists. For the effects with sufficient evidence the threshold levels are usually well known, and for some also the dose-effect relations over a range of exposures could be established.

Table 5.1: Summary of effects and threshold levels for effects where **sufficien**t evidence is available				
	Effect	Indicator	threshold, dB	Reference (Chapter. section)
Biological effects	Change in cardiovascular activity	*)	*)	Ch.III.1.5
	EEG awakening	$L_{Amax,inside}$	35	Ch.IV.10
	Motility, onset of motility	$L_{Amax,,inside}$	32	Ch.III.1.8 dose-effect relation for aircraft
	Changes in duration of various stages of sleep in sleep structure fragmentation of sleep	$L_{Amax,inside}$	35	CH.III.1
Sleep quality	Waking up in the night and/or too early in the morning	$L_{Amax,inside}$	42	Ch.III.1.7, dose-effect relation for aircraft
	Prolongation of the sleep inception period, difficulty getting to sleep	*)	*)	Ch.III.1
	Sleep fragmentation, reduced sleeping time	*)	*)	Ch.III.1
	Increased average motility when sleeping	$L_{night,outside}$	42	Ch.III,2. dose-effect relation for aircraft
Well being	Self reported sleep disturbance	$L_{night,outside}$	42	Ch.IV.2. Dose-effect relations for aircraft/road/rail
	Use of somnifacient drugs and sedatives	$L_{night,outside}$	40	Ch.IV.5.8
Medical conditions	Environmental Insomnia[1]	$L_{night,outside}$	42	Ch III.1, Ch.IV.1., Ch.IV.2

[1]Note that "Environmental Insomania" is the result of diagnosis by a medical professional whilst "Self reported sleep disturbance" is essentially the same, but reported in the context of a social survey. Number of questions and exact wording may differ.

Table 5.2: Summary of effects and threshold levels for effects where **limited** evidence is available				
	Effect	Indicator	Estimated threshold, dB	Reference
Biological effects	Changes in (stress) hormone levels	*)	*)	Ch.II.5
Well being	Drowsiness/Tiredness during the day and evening	*)	*)	Ch.II.2.3
	Increased daytime irritability	*)	*)	Ch.II.2.3
	Impaired social contacts	*)	*)	Ch.II.2.3
	Complaints	L_{night}	35	Ch.IV.3
	Impaired cognitive performance			Ch.II.2.3
Medical conditions	Insomnia	*)	*)	Ch.IV.6
	Hypertension	L_{night} (Probably depending on day time exposure as well)	50	Ch.II.2.3 Ch. IV.5.6
	Obesity	*)	*)	Ch.II.2.3
	Depression (in women)	*)	*)	Ch.IV.8
	Myocardial infarction	L_{night} (Probably depending on day time exposure as well)	50	Ch.IV.5.15
	Reduction in life expectancy (premature mortality)	*)	*)	Ch. II.2.3 Ch. II.5
	Psychic disorders	L_{night}	60	Ch. IV.8.15
	(Occupational) accidents	*)	*)	Ch II.3.2.3 Ch II.4

*) Although the effect has been shown to occur or a plausible biological pathway could be constructed, indicators or threshold levels couldn't be determined.

Note that as the evidence for the effects in this table is limited, the threshold levels have also a limited weight. In general they are based on expert judgement of the evidence.

3 Relations with L_{night}

Over the next years, the END will require that night exposures are reported in L_{night}. It is therefore interesting to look into the relation between $L_{night,outside}$ and the effects from night time noise. The relation between the effects listed in table 5.1 and 5.2 and $L_{night,outside}$ is however not straightforward. Short term effects are mainly related to maximum levels per event inside the bedroom: $L_{Amax,inside}$. In order to express the (expected) effects in relation to the single EU- indicator, some calculation needs to be done. The calculation for the total number of effects from reaction data on events (arousals, body movements and awakenings) needs a number of assumptions. The first that needs to be made is independence: although there is evidence (Brink, 2006) that the order of events of different loudness influences strongly the reactions , the calculation is nearly impossible to carry out if this is taken into consideration. Secondly the reactions per events are known in relation to levels at the ear of the sleeper, so an assumption for an average insulation value must be made. In this report a value of 21 dB (see Chapter I, sections 3.4 and 3.5) has been selected. This value is however subject to national and cultural differences. One thing that stands out is the desire of a large part of the population to sleep with windows (slightly) opened. The relatively low value of 21 takes this already into account. If noise levels increase, people do indeed close their windows, but obviously reluctantly as then complaints about bad air increase and sleep disturbance remains high. This was already pointed out in the Guideline on community noise (WHO, 2000).

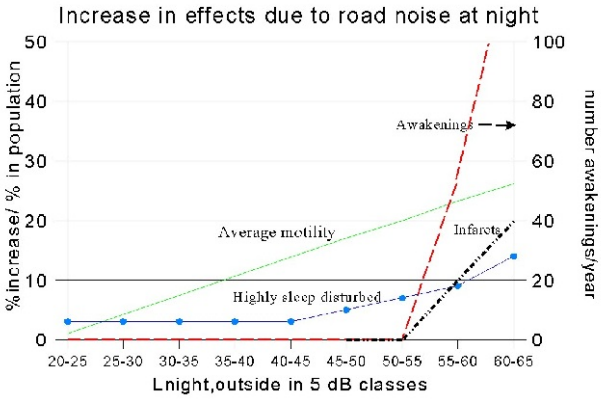


Figure 24. Relation of effects with Lnight for an urban road

From source to source the number of separate events varies considerably. Road traffic noise is characterised by relatively low levels per event and high numbers, while air and rail traffic are characterised by high levels per event and low numbers. For 2 typical situations estimates are made and presented in graphical form. The first is an average urban road (600 motor vehicles per night, roughly 3 million/year, the criteria from the END)) and the second case is for an average air traffic

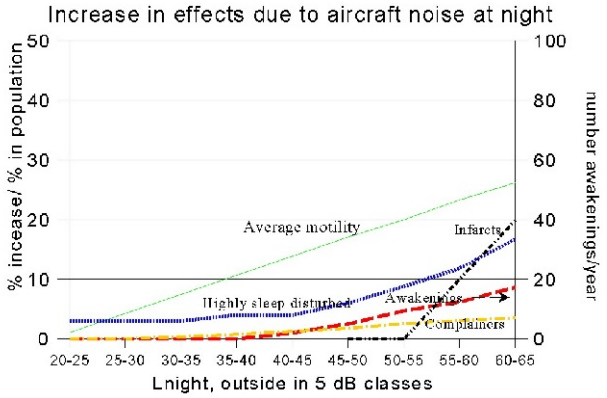


Figure 25. Relation of effects with Lnight for a typical airport situation.

exposure situation (8 flights per night, nearly 3000 per year).

Figure 24 shows how effects increase with an increase of $L_{night,outside}$ values for the typical road traffic situation (urban road). To illustrate this in practical terms: values over 60 dB Lnight occur at less then 5 meters from the centre of the road in this example. In figure 25 the same graph is presented for the typical airport situation.

In these examples the worst case figures can be factors higher: the maximum number of awakenings for an $L_{night,outside}$ of 60-65 dB is around 300/year.

A recent study suggests that high background levels (from motorways) with low number of separate events can cause high levels of average motility (Passchier (2006), to be published).

In table V.3 the full details are summarized.

Table 5.3	Effects (yearly;additional with respect to the normal except odd ratio) 0=below threshold, +=increase)										
$L_{night,outside}$	Arousals		Body movements related to single exposures -15 sec intervals-		Average body movements (without the single exposures)	Awakenings		% sleep disturbed (% highly sleep disturbed)			Myocardial Infarcts
UNIT	number	number	number average air traffic	number average urban road	number	number average air traffic	number average urban road	% of exposed Air	Road	Rail	odd ratio
NORMAL (no exposure)	Children 2555	Adults 3650	21000		21000	600		0			1
20-25	0	0	0	0	200	0	0	0	0	0	1
25-30	0	0	7	0	875	0	0	<3	<3	<2	1
30-35	+	+	22	0	1547	0	0	<3	<3	<2	1
35-40	+	+	37	0	2220	0	0	4	3	2	1
40-45	+	+	58	243	2900	2	0	4	3	2	1
45-50	+	+	85	635	3600	5	0	6	5	2	1
50-55	+	+	111	1145	4200	9	0	9	7	3	1
55-60	+	+	145	1770	4900	12	54	12	9	4	1.1
60-65	+	+	180	2520	5500	17	155	17	14	6	1.2

4 Dealing with situations exceeding the thresholds.

Noie exposure data demonstrate that a large part of the population is over the no-effect levels. It is expected that this will extend into the future for quite some time. This means that circumstances may require that a risk-assessment must be made. It is then recommended to apply the method laid out in Chapter I, using the values given in table 5.1 and 5.2 and dose-effect relations given in Chapter IV. In order to estimate the Burden of Disease the Disability weights provided by WHO (2007, to be published) can be used. The weight for self reported sleep disturbance provided in Ch.IV.9 is an element of the study to be published.
Typical actions requiring risk-assessment are:
- new infrastructure projects (if an environmental impact statement is required, as part of this)
- improvement programs
- policy evaluation
- national or international setting of limit values

In the EU-Position Paper on L_{night} (2002) an overview of national night time noise regulations may be found.

5 Protection measures & control

What is the best strategy to reduce sleep disturbance?
The first thought should always be to reduce the impact, either by reducing the number of events or by reducing the sound levels, or both. For some effects reducing the number of events may seem to be more effective (although that depends on the exact composition), other effects are reduced by lowering overall noise level by either the number of events, the levels per event or by any combination.
In combination with other measures, sound **insulation** of bedroom windows is an option, but care must be taken to avoid negative impact on inside air quality. Even then, many people may want to sleep with their windows open, thereby making the insulation ineffective. Although good instruction may go some way to helping to overcome this, it is still a matter well worth taking into account. In warmer climates in particular insulation is not a serious option for residential purposes and excessive exposure must be avoided either by removing the persons exposed or removing the source if source-related measures fail.
Although air-conditioning of houses (or just bedrooms) is not commonplace in the EU, there are indications that its use is increasing, especially in the warmer parts of the Union. Although this still leaves the possibility that people may sleep with their windows open outside the summer season, it is something to consider when discussing measures.
Exposed areas could be a good choice for uses such as offices where there will be no people at night or where it is a physical impossibility to sleep with the windows open (fully airconditioned buildings, for example hotels and sometimes hospitals).
A simple measure is the **orientation** of noise-sensitive rooms on the quiet side of the dwelling (this applies to road and rail traffic noise).
Zoning is an instrument that may assist planning authorities to keep noise-sensitive land uses away from noisy areas. In the densely populated areas of the EU this solution must often compete with other planning requirements or plain lack of suitable space.

6 Recommendations for health protection

Sleep is an essential part of human functioning and is recognized as a fundamental right under the European Convention[2] on Human Rights. Based on the evidence of the health effects of night noise, an overall summary of the relation between night noise levels and health effects, and stepwise guideline values are presented as shown in Table 5.4 and 5.5, respectively.

Table 5.4 **Effects of different levels of night noise on the population's health**	
$L_{night,outside}$ up to **30 dB**	Although individual sensitivities and circumstances differ, it appears that up to this level no substantial biological effects are observed.
$L_{night,outside}$ of **30 to 40 dB**	a number of effects are observed to increase: body movements, awakening, self reported sleep disturbance, arousals. The intensity of the effect depending on nature of the source and on the number of events, even in the worst cases the effects seem modest. It cannot be ruled out that vulnerable groups (for example children, chronically ill, elderly) are affected to some degree.
$L_{night,outside}$ of **40 to 55 dB**	there is a sharp increase in adverse health effects, and a substantial proportion of the exposed population is now affected and adapt their lives to cope with the noise. Vulnerable groups are now severely affected
Above a $L_{night,outside}$ **of 55 dB**	the situation is considered increasingly dangerous for public health. Adverse health effects occur frequently, a high percentage of the population is highly annoyed, and there is limited evidence that the cardio-vascular system is coming under stress.

Especially in the range between $L_{night,outside}$ from 30 to 55 dB a closer look may be needed into the precise impact as this may depend much on the exact circumstances. Above 55 dB the cardiovascular effects become the dominant effect which is thought to be less dependant on the nature of the noise.

From Table 5.1 it is clear that a number of instantaneous effects are connected to threshold levels expressed in $_{Lamax}$. The health relevance of these effects cannot be easily established. It can be safely assumed, however, that an increase in the number of such events over the base line may constitute a subclinical adverse health effect by itself leading to significant clinical health outcomes.

For the primary prevention of subclinical adverse health effects in the population related to night noise, it is recommended that the population should not be exposed to night noise levels greater than 30 dB of $L_{night,outside}$ during the night when most people are in bed. Therefore,

[2] *Article 8:1. Everyone has the right to respect for his private and family life, his home and his correspondence.* Although in the Case against the UK the Court ruled that the UK-government wasn't guilty of the charges, the right on undisturbed sleep was recognized (the Courts consideration 96).

$L_{night,outside}$ 30 dB is the ultimate target of Night Noise Guideline (NNGL) to protect the public, including the most vulnerable groups such as children, the chronically ill and the elderly, from the adverse health effects of night noise. Two interim targets are proposed for the countries where the NNGL cannot be achieved in a short period for various reasons, and where policy-makers choose to adopt a stepwise approach at the local or national levels (Table 5.5).

Table 5.5. Proposed night noise guidelines and interim targets.	
Interim target I (IT-I)	$L_{night,outside}$ = 55 dB
Interim target II (IT-II)	$L_{night,outside}$ = 40 dB
Night noise guideline	$L_{night,outside}$ = 30 dB

All countries are encouraged to reduce gradually the size of the population exposed to levels over the interim targets, 40 dB of $L_{night,outside}$ (IT-I) and 55 dB of $L_{night,outside}$ (IT-II), as effectively as possible.
It is highly recommended to carry out risk assessment and management activities at national level targeting the exposed population, and aiming at reducing night noise to the level below IT-I and IT-II. IT-II can be used for health impact assessment of new projects (e.g., highways, railways, airports or new residential areas) even before the achievement of IT-I, as well as for the risk assessment of the whole population. In the long run the NNGL would be best achieved by control measures on the sources along with other comprehensive approaches.

7 Relation with the 2000 Community Guidelines

The WHO-Community Guidelines published in 2000 have been quoted a number of times in this paper, so one rightfully could ask what the relation is between the 2000 guideline and the present night noise guideline.
Night-time exposure to noise and sleep disturbance is indeed covered in the 2000 guidelines, and this is the full statement (page 63 of the web-version):

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

It should be borne in mind that the 2000 guidelines is based on studies carried out up to 1995 (and a few meta analysis some years after). Important new studies (Passchier-Vermeer 2002 and Basner 2004) have become available after that, together with new insights on normal and disturbed sleep.

Comparing the above statement with the recommendations, it is clear that new information made more precise statements possible. The thresholds for a number of effects are now known, and much lower than an L_{Amax} of 45. The last 3 sentences still stand: there are good reasons for people to sleep with their windows open, and to prevent sleep disturbances one should consider the equivalent sound pressure level and the number of sound events. The present guidelines allow responsible authorities and stakeholders to do this. Viewed in this way, the present guidelines may be considered an extension to the 2000 guidelines. That also means that the recommendations contained in the sections on noise management and control can be applied to the guideline values of this document.

8 Recommendations for further research

Although the fundamental relations between sleep, health and noise have been laid bare, throughout the report gaps in knowledge have been noted. On the other hand, some research is going on and being planned. Without pretending to be exhaustive, a summary is presented here.

Gaps in knowledge

- The relation between insulation/window behaviour/noise level are important to estimate exposure and sleep related health outcomes. There is however a lack of data on all three elements.
- Only few countries have data about exposure to noise of the population, and even fewer focus on night time exposure.
- There are indications that night time noise contributes more to health problems than day time noise. This may be true for cardiovascular disease and psychiatric disorders, and other disorders as well.
- Medical and environmental concepts of insomnia seem to differ. A study could be set up to bring more unity and thereby improving the type of and the need for different intervention actions.
- That a number of accidents is fatigue related and therefore sleep problems play a role is not very much in doubt. Data to quantify this and the importance of noise during the night time are missing however.
- · There is very limited information on a possible relation between noise exposure and potential instantaneous effects, such as effects on memory consolidation or restoration of the immune system, and for this reason were not considered in this report.
- Animal studies show that noise and sleep deprivation are stressors that both lead to early death. A careful designed animal study may establish if for instance the noise effect is due to sleep deprivation or due to autonomous processes.

Studies on their way and planned.

- Although not directly connected to sleep, the EU-HYENA (Hypertension and exposure to noise near airports) study into the effects of aircraft noise on high blood pressure may shed some light on sleep related disturbances if analysed with night time exposure in mind.
- the German group (Basner, 2004) responsible for the outstanding sleep and aircraft noise study is planning a study into the relation between road traffic noise and sleep.
- the CALM-strategy paper produced in connection with European research into advanced transport noise mitigation (CALM, 2004) recommends to study improved dose-effect relations for L_{night} and methods for valuation undisturbed sleep. The CALM-strategy papers are input for the EU-research program (Frame work Program 7).

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APPENDIX I. Relations between L_{night} and instantaneous effects

Statement 1

Let f be a function of *SEL* that gives the expected number of instantaneous effects caused by a single event. With a given L_{night} and a given number of events *N*, the expected number of times that an effect occurs in the night, *n*, is maximal if all events have equal *SEL*, provided that f∘10lg is increasing but negatively accelerated.

Statement 2

If

$$n_{max} = 10^{(Lnight-SEL+70.2)/10} \cdot f(SEL),$$

has a maximum over *sel* and f is the quadratic function $f(SEL) = a\ SEL^2 + b\ SEL + c$, then the maximum occurs irrespective of L_{night} at

$$SEL_0 = 4.34 - A \pm [(A - 4.34)^2 - (c/a) + 8.68A]^{½},$$

where A = b/(2a). (Only with + at the place of ± the value will come in the realistic range of *SEL*)

Statement 3a

If the shape of the time pattern of the sound level has a block form, then $SEL = L_{Amax} + 10lgT$, where L_{Amax} is the maximum sound level (integrated over 1-s) and *T* is the duration of the noise event in s.

Statement 3b

If the sound level increases with rate *a* (in dB(A)/s) and after time point *t* = 0 decreases with rate *–a*, then $SEL \quad L_{Amax} - 10lga + 9.4$.

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APPENDIX II. The process

In September 2003 the WHO Centre for Environment and Health started the Night Noise Guideline project with a grant from the European Commission (DG-SANCO). The first meeting was held in Bonn, on 7 and 8 June 2004. It was agreed that the partners would produce background papers on a number of topics identified in the meeting.

The second meeting (Geneva, December 2004) concentrated on the technical issues like exposure assessment, metrics, health effects, guideline set-up. The partners presented the first draft papers for the different identified topics and detailed discussion took place for each one of them. The discussions concentrated mainly on central issues like exposure assessment and guideline derivation.

The WHO noise unit organized and prepared the background material and some partners prepared

papers. The discussion was around the papers and on the way forward, especially to address lacks on evidence and what (and how) to consider as health outcome.

The project's third meeting (April 2005) reviewed the final version of the background papers and established the future work. The meeting took place in Lisbon in the premises of the DGS (Direcção Geral da Saúde - Portuguese Directorate General for Health).

This meeting already discussed in detail the final version of the document and how to proceed until the end of the project.

In September 2005, the fourth meeting of the project was convened in Den Hague to discuss the issue of noise indicators and the penalty weight of night noise for Lden.

In December 2006 the final conference took place in Bonn where the final draft was discussed with stakeholders. The text of the conclusions was extensively discussed, amended and approved. On the whole there was general agreement on the approach chosen by the project team. The participants agreed on the key message of the final text to be presented as Chapter V of this document.

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APPENDIX III. Animal studies on stress and mortality (*Ising*)

II. Introduction

Is noise a health risk or does it just annoy? This basic question needs to be carefully answered when establishing night noise guidelines. No one will deny that in the case of high noise levels there is a risk of inner ear damage, but what about the moderate levels of environmental noise? To approach this rather difficult question we have to combine all available methods.

1.) In animal experiments it is possible to assess the complete causal chain from noise exposure via physiological reactions and biological risk factors to morbidity or even mortality. However, a quantitative application of the results to humans is not possible. Instead, the method is useful in studying the patho-mechanisms qualitatively.

2.) Experiments on humans are, for ethical reasons, restricted to the study of reversible physiological reactions. But as long as there is no proof that reactions to chronically repeated noise exposures are increasing the risk of specific diseases, the results of such physiological studies are not considered conclusive.

3.) Epidemiological studies have the advantage of investigating health effects which are particularly caused by chronic noise exposure although there is no possibility to control all influencing factors. Additionally, epidemiological studies have to be based upon biologically evident hypotheses.

Our hypothetical model of noise induced health effects is shown in Fig. 15 In Ch.IV.5.2 on "Cardiovascular effects of noise" by Babisch in this report. This model is based on the results of noise experiments with animals and humans. With animal experiments, the whole causal chain from noise exposure to health outcome can be traced as a direct pathway starting with a chronic high level noise exposure which via endocrine stress reactions leads for example to micro-circulatory defects and to manifest hypertension.

Physiological experiments on humans have shown that noise of a moderate level acts via an indirect pathway and has health outcomes similar to those caused by high noise exposures on the direct pathway. The indirect pathway starts with noise induced disturbances of activities such as communication or sleep. Since we are dealing with night noise guidelines, noise induced sleep disturbances and any following persistent health effects are of primary interest here.

In physiological studies with experimentally changed noise exposure the increase of arousals and of hormone excretion was studied in sleeping persons. If our model is correct then in the cause effect chain the arousal ought to precede the endocrine reactions. This order was derived from the different reaction times of the effects. While arousals appear within one second after a noise stimulus, hormones like catecholamines take several minutes, and cortisol about 10 minutes to be increased. This observation together with the fact that arousals are evoked by equal or lower noise levels than the corresponding endocrine reactions confirms the correctness of our model and leads us to an important conclusion:

Noise exposure, which does not evoke arousals in sleeping persons, will not induce adverse health effects.

This conclusion is essential with regard to night noise guidelines. However, the answers to our basic question whether certain health risks are connected with environmental noise must be clarified by epidemiological studies based on noise experiments on both humans and animals.

IV. Types of animal studies

Noise has often been used as a stressor in animal studies. Even Selye [1953] who introduced the psycho-physiological stress concept used noise stimuli in his animal studies. Most of the modern animal studies testing the pharmacological effects of drugs are carried out with and without various stressors. The typical noise exposure is that to short and very intensive sounds. One such example is the study of Diao et al. [2004] who exposed guinea pigs to 4 kHz octave band noise at 115 dB for 5 hours. But these experiments are of little value regarding the noise exposure types in question.

The same is true for another type of animal studies concerned with the prevention of noise induced health effects in wild and domestic animals [for review of the former kind see Fletcher, 1983]. One example for the latter kind is the study of Geverink et al. [1998] on stress responses of pigs to transport and lairage sounds.

Since the subject of the present paper is noise induced health effects in humans, we will review only those studies in which animals are used as a model for humans.

The animal model for aural effects in humans has been established in great detail, so that even quantitative transference of results from animals to humans is possible. However, inner ear damage generally occurs at much higher noise levels than the environmental levels under discussion in this paper. Therefore we will focus our interest on animal models with respect to extra-aural noise effects.

VI. Limiting aspects of animal models

Other than in studies on aural effects, the animal model does not allow quantitative comparisons in studies of extra-aural noise effects. It may however be used for the qualitative investigation of patho-physiological mechanisms following exposure to acute and very short sounds. But an animal model for long-term noise effects as caused by chronically repeated noise exposures needs careful planning. First it has to be ensured that stress reactions in both humans and animals when activated by noise exposure are qualitatively comparable. Secondly, the stress effects of chronic noise exposure have to be assessed in humans, and the animal models to be designed correspondingly. However, in the animal model influences from cortical interconnections have to be excluded as a factor in these noise experiments. Naturally, we cannot expect to establish an animal model for indirect environmental noise effects which in humans may, for example, disturb activities like verbal communication which in turn may induce stress hormone increases.

VIII. Stress hormones in noise exposed animals

3.1 Habituation

In short-term experiments any kind of loud noise exposure will cause acute increases of stress hormones. Long-term repeated noise exposure, however, will cause a certain habituation in the animal. Periodic repetitions of identical noise bursts lead to almost complete habituation. This was probably the main reason why Borg [1981] found no adverse health effects in rats exposed for their whole lives to periodic noise pulses. Therefore, random series of noise pulses are now applied in most long-term studies.

Selye [1974] had already stated that not all stages of a stress response are noxious, especially in the case of mild or brief exposures. Since environmental noise is a mild stressor, adverse health effects are only to be expected under the condition that repeated noise exposures induce long-term stress hormone changes. According to the Allostatic Load Model [McEwen, 1998], the normal response to an environmental stressor such as noise is the physiological activation of the endocrine system enabling the body to cope with the stressor and, after the stress situation is terminated, to

shut off the allostatic response. Fig.1 shows McEwen´s graph of stress response and development of allostatic load.

X. J.D. Henry's model of behavioural stress effects

On the basis of the available literature on stress effects in animals and humans, Henry [1992] developed a model with regard to different biological effects and health risks associated with different coping styles. He explains that the neuroendocrine response to various challenges and threats varies according to the type and degree of control a mammal can exact over it. This in turn is strongly determined by the animal's previous experience. In general, the sympathetic adrenomedullary system is preferentially activated when the animal displays active responding to escape from or deal with an environmental challenge. This is the fight/flight mode of stress response. The adrenocortical axis is preferentially activated as the subjects become immobile/passive when no control or threat of its loss is experienced. This is the conservation-withdrawal mode of response.

XII. The Noise Stress Model

On the basis of noise effect studies in animals and humans (for review see [Ising and Braun, 2000]) we developed a Noise Stress Model. It describes a differentiation of prevailing "stress hormones" under noise exposure: Predominantly adrenaline - and to a lesser degree noradrenaline - are released from the adrenal medulla as the normal response to novel noise stimuli of moderate intensity. Following long-term noise exposures of moderate intensity habituation will alter the response mode and predominantly noradrenaline is released. As a response to extremely intensive noise, near the inner ear pain threshold, predominantly cortisol is released from the adrenal cortex induced by increased releases of adrenocorticotropic hormone (ACTH), especially in the case of unexpected noise.

The described differentiation will only be observed under special conditions. Unexpected exposure for 3 minutes white noise at 75 dB leads, in awake dogs, to increased adrenal secretion of adrenaline and noradrenaline and - following a delayed increase in plasma ACTH - an increase in cortisol secretion [Engeland et al. 1990].

The cortisol response as described is valid for animals and humans in their active phases. During sleep, however, several studies in humans showed cortisol increases under exposure to traffic noise of moderate levels [Evans et al.2001; Maschke et al. 1995; Ising H. & Ising M. 2002; Ising et al. 2004]. It was hypothesised that noise stimuli signalling a danger, for example the noise of an approaching lorry, will during sleep generally be responded by a defeat reaction, which includes the release of cortisol from the adrenal cortex. Appropriate studies with sleeping animals after conditioning them - for example with a specific noise stimulus followed by pain - should be carried out to test this hypothesis.

Rats were exposed for a period of 12 hours to low-altitude flight noise - reproduced electro-acoustically once per hour on average at stochastically fluctuating intervals (L_{Amax} 125 dB, 10 dB downtime: 1 s, Leq: 89 dB) [Ising et al. 1991; Ising, 1993]. We found a tendency for adrenaline and noradrenaline excretions to decrease, whereas plasma cortisol increased significantly). (Although in rats corticosterone is secreted rather than cortisol, we will simplify this paper by using cortisol for rats all the same. In this experiment as well as in all others of our group, normally 4 rats were kept in one stainless steel cage, which was set on a funnel to collect their urine).

These results show that exposure to noise levels approaching or exceeding the pain threshold of the inner ear leads to endocrine reactions qualitatively different from those induced by less intensive noise.

The different endocrine reactions to acute and chronic noise exposure were studied in rats by Gesi et al. [2002]. They were exposed either to a single (6 h) session of loud (100 dBA) noise, or to the same noise stimulus repeatedly every day for 21 consecutive days. Exposure to noise for 6 h on one day induced parallel increases in dopamine, noradrenaline and adrenaline concentrations in tissue samples of the adrenal medulla. After 21 days of noise exposure, noradrenaline concentration was significantly higher than in controls, and that of adrenaline decreased significantly. Cortisol was not assessed in this study.

In another subchronic noise experiment, rats were exposed to irregular white noise at 90 dB for 3 and 9 h per day during 18 and 8 days respectively [van Raaij et al. 1997]. In rats with 3 h exposure per day the blood concentrations of adrenaline, noradrenaline and cortisol did not differ from controls. Exposure for 9 h per day, however, resulted in significantly increased concentrations of noradrenaline and cortisol. At the end of the experiment all animals were subjected to restraint stress and their endocrine reactions were assessed. The authors thus sum up their findings: These results indicate that chronic noise exposure at mild intensities induces subtle but significant changes in hormonal regulation.

The results of another experiment with different levels of random white noise pulses during 45 min/h, 12 h/day for 8 days demonstrate that cortisol responses to subchronic mild noise exposure do not monotonously increase with the noise levels [Bijlsma et al. 2001]. While in rats exposed to 95 dB pulses plasma cortisol concentrations were raised twofold against controls, the exposure to 105 dB pulses did not increase cortisol significantly.

The time dependency of cortisol increase in the blood of rats under exposure to white noise (100 dB, 6 h/day for 21 days) was examined by Gesi et al. [2001]. The authors found a progressive increase in cortisol which reached a plateau 9 days from the beginning of exposure.

In summing up the results of these studies we conclude:

· Acute exposure to unexpected and novel noise of moderate intensities leads to activation of both the sympathetic adrenal-medullary system with increased secretion of adrenaline and noradrenaline, and the hypothalamopituitary (HPA) axis with increased secretion of ACTH and of cortisol from the adrenal cortex.

· Under chronic exposure to unpredictable noise, adrenaline secretion is reduced to normal or subnormal values while noradrenaline and ACTH/cortisol concentrations remain increased.

· Extremely intensive unpredictable noise near the inner ear pain threshold triggers, in awake mammals, a defeat reaction with increases of ACTH/cortisol while the catecholamines adrenaline and noradrenaline remain normal or are slightly decreased.

· Chronic noise exposure at mild intensities will induce changes in hormonal regulation, if the individual threshold of allostasis is exceeded. A chronic allostatic load leads to subtle but significant changes in hormonal regulation, which are at present not fully understood.

XIV. Effects of prenatal noise exposure on the sensitivity to stress

Pregnant rats were subjected to noise and light stress, three times weekly on an unpredictable basis throughout gestation [Weinstock et al. 1998]. Blood concentrations of adrenaline, noradrenaline and cortisol at rest and after footshock were assessed. At rest cortisol was significantly increased in offspring of stressed rats in comparison to controls while adrenaline and noradrenaline did not differ in

either of the groups. After footshock noradrenaline was significantly higher in offspring of stressed rats, showing that prenatal stress can induce long-term changes in the sensitivity of the sympathicoadrenal system to stress.

Pregnant monkeys were repeatedly exposed to unpredictable noise during days 90-145 post conceptionem [Clarke et al. 1994]. Blood concentration of ACTH and cortisol were measured in offspring of stressed and control monkeys at rest and under four progressively stressful conditions.

Prenatally stressed offspring showed higher ACTH than controls in all four stressful conditions while cortisol did not change under stress. These results indicate that prenatal stress may have long-term effects on the HPA axis regulation.

XVI. Effects of noise exposure on cortisol and the immune system

The effect of acute noise stress on rats was studied by assessing blood concentrations of cortisol and total as well as differential leukocyte count [Archana and Namasivayam, 1999]. A significant increase in cortisol and a significant decrease of total leukocyte counts were found.

Rats were exposed to "rock" music (80dB) for 24 h [McCarthy et al. 1992]. In vitro stimulation of leukocyte sub-populations revealed several noise effects. Neutrophils and macrophages secreted significantly less superoxide anion and interleukin-1. Such effects may be detrimental to wound healing.

Pregnant rats were from gestation day 15 to day 21 daily exposed to the noise of a fire alarm bell (L_{Amax} = 85 - 90 dB) delivered randomly for 1 h [Sobrian et al. 1997]. In developing offspring mitogen-specific alterations in lymphoproliferatic activity and reduced immunoglobulin G levels were found at postnatal day 21.

Aguas et al. [1999] exposed a special breed of mice to low frequency noise - a model of noise - occupational for three months as described below [Castelo Branco et al. 2003]. These mice spontaneously developed an autoimmune disease at 6 months of age. Chronic low frequency noise exposure accelerated the expression of the autoimmune disease and affected the immune system, which was associated with kidney lesions and increased mortality.

XVIII. Embryotoxic effects

Geber [1973] exposed pregnant rats day and night for three weeks to constantly changing sound mixtures between 76 and 94 dB for 6 minutes per hour day and night and demonstrated embryotoxic effects, notably calcification defects in the embryos.

Pregnant rats on a moderately magnesium deficient diet were noise exposed during their active phase from 8 p.m. to 8 a.m. for three weeks (stochastically applied white noise impulses L_{Amax} : 87 dB, Leq: 77 dB, t: 1 s duration) [Günther et al. 1981].

As compared to controls on the same diet, there was no difference in bone mineralization. The only significant effect was an increased fetal resorption rate.

The noise was changing in Geber's experiment but the noise level was comparable to Günther's stochastically applied noise impulses (L_{Amax} = 87 dB). Since Günther's impulses were more frequent, their stress effect was at least as strong as the noise exposure employed by Geber. Therefore the major factor that differentiated the two exposure types in causing a reduced mineralization of the rat skeletons [Geber,1973] must have been the additional noise exposure during sleep.

Castelo Branco et al. [2003] studied Wistar rats born under low frequency noise exposure. The third octave level of the applied broadband noise was > 90 dB for frequencies between 50 and 500 Hz. The broadband level was 109 dB(lin). The exposure schedule was chosen as a model for occupational noise: 8 h/day, 5 days/week, and weekends in silence. Third generation rats born in low frequency noise environments were observed showing teratogenic malformations including loss of segments.

XX. Morphological alterations in the myocardium caused by acute noise

Gesi et al. [2002] reviewed the literature and stated: In experimental animals undergoing noise exposure, subcellular myocardial changes have been reported, especially at mitochondrial level; in particular, after 6 hours of exposure only the atrium exhibited significant mitochondrial alterations, whereas after 12 hours as well as subchronic exposure both atrium and ventricle were

damaged.

Exposure of rats to 100 dB(A) noise for 12 h caused a significant increase of DNA damage accompanied by ultrastructural alterations and increased noradrenaline concentrations in the myocardium [Lenzi et al. 2003]. In another paper this group described an increase in mitochondrial calcium (Ca) influx caused by the same noise exposure. They described Ca accumulation at myocardial subcellular level. Summing up their results they write: Moreover, the present results joined with previous evidence indicate that calcium accumulation is the final common pathway responsible for noise-induced myocardial morphological alterations [Gesi et al. 2000].

XXII. Connective tissue proliferation

Hauss et al. [1971] described a proliferation of connective tissue in the myocardium of rats under acute exposure to noise.

On the basis of these results we carried out a noise exposure experiment of 5 weeks with day and night exposure to stochastically triggered bells (L_{Amax} : 108 dB, t (duration of one signal):1 s, Leq: 91 dB) [Ising et al. 1974]. We confirmed the results of Hauss et al. [1971] using an electron microscope to demonstrate fibrosis in the interstitial tissue of the myocardium. Additionally we observed electron dense areas (visible as black spots) located within bundles of collagen in the myocardium. According to Selye [1962], these dark areas were most probably caused by high concentrations of calcium (Ca) carbonate or calcium phosphate deposits. This suggestion is consistent with the results of Gesi et al. [2000].

After publication of these findings, a reservation was correctly voiced that, as the noise exposure had not left intervals for sleep, it was not certain whether the myocardial damage was provoked by the noise stress as such or by a noise-induced lack of sleep. For this reason, in all subsequent experiments we provided for noise-free intervals of 8 to 12 hours during the rats' inactive phases to enable them to sleep.

Rats were exposed for 28 weeks to a random series of white noise impulses from 4 p.m. to 8 a.m. daily with an 8h rest in their inactive phase [Ising et al. 1979]. The third octave spectrum of the noise was flat between 5 and 25 kHz and had a third octave level of 88 dB (lin) (broadband L_{Amax} =97 dB(lin). Leq=87 dB(lin)). The duration of noise impulses was 4 s and the noise to pause ratio 1:10 on average. We found a small but significant increase in hydroxyproline as indicator of collagen in the rats' left myocardium. Electron micrographs showed, similar to our earlier experiment, collagen bundles in the otherwise empty interstitial space but no indication of calcium deposits.

XXIV. Respiratory effects

Castelo Branco et al. [2003] studied respiratory epithelia in Wistar rats born under low frequency noise exposure and further exposed for up to 5403 h during more than 2 ½ years. The third octave level of the applied broadband noise was > 90 dB for frequencies between 50 and 500 Hz. The broadband level was 109 dB(lin). The exposure schedule was chosen as a model for occupational noise: 8 h/day, 5 days/week, week ends in silence. Rats were gestated and born under the described noise exposure with additional exposures from 145 h to 5304 h. Transmission electron micrographs of the tracheal epithelium of rats exposed for 2438 h revealed a subepithelial layer of hyperplastic collagen bundles, several of them exhibiting a degenerative pattern. The results indicate an increased proliferation as well as degenerative processes of collagen.

Castelo Branco et al. [2003] observed sheared cilia in the respiratory epithelia of Wistar rats born under and further chronically exposed to low frequency noise. As interpretation of their findings they state: Both mechanical and biochemical events may be responsible for this pattern of trauma.

XXVI. Electrolytes: Ca/Mg shift

Acute exposure of rats to the fast rising overflight noise of low flying fighter planes reaching levels of up to 125 dB(A)) [Ising et al. 1991; Ising, 1993] resulted not only in an increase of cortisol but also in a decrease of intracellular magnesium (Mg) and an increase of Mg excretion (see Tab.1).

Acute stress - due to 2 h of noise exposure (95 dB white noise) or to overcrowding in the cage - caused in guinea pigs significant increases of serum Mg and decreases of erythrocyte Mg [Ising et al. 1982].

For chronic noise experiments we had been searching for an additional stress factor which would act synergistically with unwanted noise, since in the above described noise experiment, half a year of exposure led to but relatively mild health effects [Ising et al. 1979]. The justification for using two stressors derives from the fact that man has to cope with a whole range of more or less synergistic stress factors and not with noise alone.

Organic damage as a result of chronic stress is likely to occur only under the condition that the overall exposure to stress exceeds a certain tolerance level during a relatively long period of time [Selye, 1976]. For technical reasons, the two options available to supply a suitable additional stress factor were the cold or a magnesium deficiency. Both factors, like habitual noise, cause an increased noradrenaline secretion. For practical reasons we selected different degrees of a magnesium-deficient diet as an additional stress factor. Noise exposure was provided by electro-acoustically reproduced traffic noise of L_{Amax} : 86 dB, Leq: 69 dB over 12 hours during the rats' active phase. For one group the noise level was slightly increased (Leq: 75 dB). The experiment lasted 16 weeks [Günther et al. 1978]. Tab 2 shows the results. Mg deficiency combined with noise exposure led to dose-dependent increases in adrenaline and noradrenaline, which can be used to quantify the overall stress of the dietary treatment. As stress grew, the hydroxyproline (as an indicator of collagen) and calcium content of the myocardium increased while the magnesium content decreased. Long-term stress therefore resulted in an intracellular Ca/Mg shift.

Altura et al. [1992] studied the relationship between microcirculation (measured several days after termination of noise exposure), hypertension and Ca/Mg shifts in vascular walls of noise stressed rats on Mg deficient diets. Noise exposure during the first 8 weeks was set to an energy equivalent level of 85 dB(A) from 8 p.m. to 8 a.m. Noise impulses were randomly switched on at randomised peak levels of 80, 90 and 100 dB(A). During the final 4 weeks the equivalent noise level was elevated to 95 dB(A) and the daily exposure increased to 16 h with an 8 h rest during the animals' inactive phase. In aortic and port vein smooth muscle the Ca content increased with rising noise exposure, with decreasing Mg uptake, and with the combination of both together, while the Mg content decreased. Parallel to this the reactivity of terminal arterioles to noradrenaline was increased (Fig.4a).

Stress-induced Ca/Mg shifts in smooth muscle cells have the potential to increase the risk of hypertension and myocardial infarction (MI) [Ising et al.1985].

Stress increases the membrane permeability of catecholamine-sensitive cells, which in turn raises Ca influx into cells and liberates intracellular Mg. A depression of catecholamine-induced vasoconstriction by stress-dependent hypermagnesemia (excess serum Mg concentration) has been demonstrated experimentally. However, the benefit from this stress-depressing hypermagnesemia is obtained at the expense of increased renal Mg loss. In the long run, chronic stress combined with suboptimal Mg in diet will reduce the Mg release in acute stress situations, causing an increase of vasoconstriction and raising the risk of hypertension.

Further analysis of our experimental results led to an interaction model between chronic stress and intracellular electrolyte shifts [Ising, 1981; Ising et al., 1986] (Fig.5). Chronic stress caused a loss of extracellular and intracellular Mg and an increase in intracellular Ca [Günther et al. 1978]. A decrease of Mg was correlated with an increased physiological noise sensitivity, i.e. to more

severe noradrenaline releases in animals and humans under noise exposure [Günther et al. 1978; Ising et al. 1986; Ising et al. 1985]. We found a positive feedback mechanism between stress - caused by noise and other stressors - and intracellular Mg/Ca shifts, which may end in a circulus vitiosus and increase cardiovascular risks.

XXVIII. Hypertension

Rothlin et al. [1956] exposed rats for 1½ years, day and night, to 90 dB "audiogenic stress" and observed a raising of systolic blood pressure values from 120 mm Hg to about 150 mm Hg. He used a cross breed of Albino rats and wild Norwegian rats since Albino rats did not develop hypertension under noise exposure. After termination of exposure the blood pressure returned to normal.

Albino rats were noise exposed during their whole life span [for review see Borg, 1981] to periodic noise impulses of 80 and 100 dB. This periodic exposure had no detrimental health effects, which can be understood on the basis of the work of Glass et al. [1969]. Unpredictable noise presentation was shown to cause lasting cortisol increases in rats in contrast to periodic exposure to 100 dB, which led to adaptation [De Boer et al. 1989]. The unpredictability of a noise is a decisive precondition of long-term stress effects.

Exposure of primates to traffic noise for 10 h/day during 9 months led to a significant blood pressure increase, which persisted during 3 weeks after termination of exposure [Peterson et al. 1981]. A replication of this experiment with a different species of primates failed to show an increase of their blood pressure [Turkkan et al. 1983].

In the above mentioned experiment of Altura et al. [1992], exposure to unpredictable noise impulses led within 12 weeks to irreversible changes of micro-circulation and an increase of systolic blood pressure (Fig.4b). The observed rarefication of capillaries in the mesentery can be interpreted as an indicator of accelerated ageing of the circulatory system.

XXX. Ageing and life span

The cortisol response and recovery after immobilisation stress was compared in young and old rats. The results are demonstrated in Fig. 6 together with Sapolsky´s Glucocorticoid Cascade Model [Sapolsky et al. 1986]. The stress response of young and old rats is more or less the same. However, while the young rats recover immediately after termination the old ones recover only in part. Therefore, acute stress leads, in old animals, to considerably prolonged cortisol increases. On the other hand, chronically repeated stress activates the HPA axis and can cause cortisol receptor losses even in younger animals, a process generally developing only in old age. Finally, chronic cortisol hypersecretion may occur along with follow-up health defects.

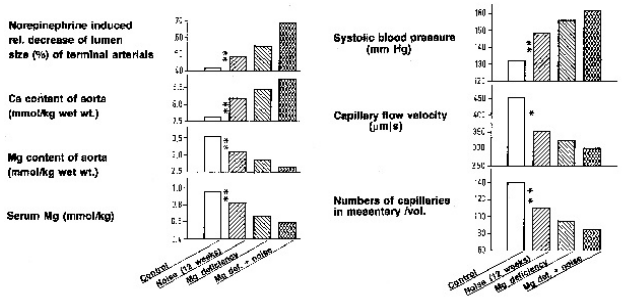

Fig.4 Effects of 12 weeks noise exposure, Mg deficient diet and the combination of both in Wistar rats.
A: Ca/Mg shifts in vascular smooth muscle, Mg concentration in blood and reactivity of arterioles to noradrenaline.
B: Systolic blood pressure, capillary blood flow velocity and numbers of capillaries/volume.

Aguas et al. [1999] exposed a special breed of mice to the above described model of occupational low frequency noise for three months. Chronic low frequency noise

exposure accelerated the expression of the autoimmune disease and was associated with kidney lesions and increased mortality.

Chronic noise exposure of animals on a suboptimal magnesium diet led to increases of connective tissue and calcium and decreases of magnesium in the myocardium [Günther et al. 1978]. These changes were correlated with noradrenaline changes. Since they are also correlated with normal ageing, the noise stress induced changes may be interpreted as accelerated ageing [Ising et al. 1981]. Even the life span was reduced in rats on a Mg deficient diet, and was further dose-dependently reduced in combination with noise exposure.

Table 2

Effects of noise exposure combined with dietary Mg-deficiency in rats.

Adrenaline and noradrenaline excretion was measured during the 4th week of noise exposure; death rate is related to the 4 months' period of Mg treatment; all other parameters were determined at the end of the experiment (mean values ± S.E.).

Treatment		Effect					
4 months	3 months	Urine		Myocardium			Death rate
Mg in diet	Noise	Noradrena-line	Adrenaline	Hydroxy-proline	Ca	Mg	
	Leq/L_{Amax}	[µg/g Cre]		[mg/g dry wt.]	[mg/g d.w.]	[mg/g d.w.]	
control	ambient	18 ± 4	12 ± 2	3.0 ± 0.1	3.0 ± 0.2	37.5 ± 0.8	0
suboptimal	ambient	23 ± 4	18 ± 2	3.0 ± 0.1	3.5 ± 0.5	38.0 ± 1.7	0
suboptimal	69/86 dB	37 ± 11	16 ± 2	3.0 ± 0.1	4.3 ± 0.2	37.9 ± 1.3	0
deficient	ambient	98 ± 17	20 ± 5	3.9 ± 0.1	6.2 ± 0.7	31.2 ± 1.4	38%
deficient	69/86 dB	129 ± 19	41 ± 10	4.6 ± 0.1	6.7 ± 0.6	29.8 ± 1.8	62%
deficient	75/86 dB	172 ± 26	60 ±15	5.6 ± 0.9	8.0 ± 0.9	26.8 ± 0.8	75%

Noise has the potential to cause stress reactions which are enhanced by suboptimal magnesium intake. Chronic noise-induced stress accelerates the ageing of the myocardium and thus increases the risk of myocardial infarction. The involved pathomechanisms include increases of catecholamines and/or cortisol under acute noise exposure and an interaction between endocrine reactions and intracellular Ca/Mg shifts.

XXXII. What can we learn from animal studies about noise effects in humans?

The effects of low frequency noise - the "Vibroacoustic Disease" - was studied primarily in humans (for review see [Castello Branco and Alves-Pereira, 2004].

In this context, the amygdalar contribution to conditioned fear learning, revealed for normal human subjects, has to be mentioned. Longer lasting activation of the HPA-axis, especially abnormally increased or repeatedly elevated cortisol levels may lead to disturbances of the hormonal balance and even severe diseases in man [Spreng, 2000].

Catecholamines induce various detrimental effects on the heart [Ceremuzynski 1981]. Additionally, magnesium deficiency causes alterations of serum lipids [Weglicki 1993], cytokines [Rayssiguier 1990] and prostaglandines [Nigam et al. 1986], in particular an increase of thromboxan, which is released from thrombocytes [Neumann and Lang 1995] and several other cell types and - in turn - thromboxan A2 can aggregate thrombocytes. All these alterations may increase the risk of myocardial infarction (MI).

Beside these cardiovascular stress effects, chronically increased cortisol may induce neuronal degeneration and thus accelerate the ageing also of the brain [Sapolski et al.1986], not only in rats

but in humans as well [Sapolsky, 1994].

The importance of Ca/Mg shifts was confirmed by post mortem studies of hearts from victims of ischaemic heart diseases (IHD) [Elwood et al. 1980]. The tissue samples were taken from areas of the myocardium not affected by the infarction and the results were stable after controlling for several confounders. The results are shown in Table 4. With normal ageing Ca increases and Mg decreases in the myocardium. This process is accelerated in myocardial infarction (MI) patients, which indicates an accelerated ageing of these persons' heart muscle under the pathogenic influences that lead to MI.

Another factor which decreases Mg and increases Ca [Hofecker et al. 1991] and collagen [Caspari et al. 1976, Gibbons et al. 1991, Anversa et al. 1990] in the myocard is normal ageing [Ising et al. 1981]. Therefore, it is plausible that the age-dependent decrease of Mg in hearts of IHD victims was about double of that in age-matched non-IHD deaths. This is therefore an indication that age- and stress-dependent electrolyte changes exist in humans and may be correlated with an increased risk of IHD.

Long-term experiments with Mg-deficient and noise-stressed rats showed that connective tissue and Ca in the myocardium increased with age while Mg decreased. Hence, stress caused by noise or cold is enhanced by sub-optimal Mg intake and accelerates the ageing of the heart and decreases the life span [Ising et al. 1981; Heroux et al. 1977; Guenther, 1991].

Since coronary arteriosclerosis increases strongly with age [Lakatta, 1990] a biologically older heart is at a higher risk of IHD and of MI. The interaction process described seems to be one of the pathomechanisms by which chronic noise stress increases the risk of MI.

Several of the risk factors described in the literature to explain the correlation of work stress with MI have been found to be increased under noise-induced stress as well, i.e. increases of blood pressure and total cholesterol.

APPENDIX IV. Noise and sleep in children (*Kahn*)

1 Factors that modify auditory arousal thresholds in children

By the time that most studies were conducted in infants, it became progressively evident that arousal and awakening thresholds are influenced by a variety of factors. These significantly modify the response to ambient noise by sleeping infants. Some factors inhibit the arousal response, while others enhance the response

1.1 Prenatal and perinatal factors

- Age of gestation.

In 97 healthy infants, auditory awakening thresholds decreased significantly from the 44th to the 60th postconceptional week (Kahn et al. 1986). Awakening thresholds were defined as the infant opening the eyes and/or crying. Mean awakening thresholds dropped from 98.5+/-11 at the 44th postconceptional week to 83 dB(A) by the 60th postconceptional week.

- Cigarette smoke

To evaluate the effects of cigarette smoke on polygraphic arousal thresholds, 26 newborns were studied with polygraphic recordings for one night: 13 were born to mothers who did not smoke, and 13 were born to mothers who smoked (over 9 cigarettes per day) (Franco et al. 1999). Another group of infants with a median postnatal age of 12 weeks were also studied: 21 born to non-smoking mothers and 21 born to smoking mothers. The auditory arousal thresholds of the infants of both age groups were measured with the use of auditory challenges of increasing intensity, administered during REM sleep. More intense auditory stimuli were needed to induce arousals in newborns (p=.002) and infants (p=.044) of smokers than in infants of nonsmokers (mean value of 84+/-11 dB(A) for smokers and 81.6+/-20 for nonsmokers). Behavioral awakening (infants opening the eyes and/or crying) occurred significantly less frequently in the newborns of smokers (p=.002) than of nonsmokers.

It was concluded that newborn and infants born to smoking mothers had higher arousal thresholds to auditory challenges than those born to non-smoking mothers. From the present findings, it appeared that the impact of exposure to cigarette smoke occurred mainly before birth.

1.2 Postnatal factors

The following postnatal factors modify arousal from sleep:

- Sleep stage

In infants, auditory stimuli have generally indicated increased responses during active as compared with quiet sleep (Busby et al. 1994).

- Time of the night

In 31 infants, the arousal thresholds decreased across the night (mean value of 67+/-12.5 dB(A) in the 1st part of the night, for 51+/-3.5 in the 3rd part of the night; p=.017) (Franco et al. 2001). Similar findings had been reported in adult subjects (Rechtschaffen et al. 1966).

- Body position during sleep

To investigate whether prone or supine sleeping was associated with a different response threshold

to environmental stimuli, 25 three-month-old healthy infants with a median age of 9 months were exposed to an auditory challenge while sleeping successively prone or supine (Franco et al. 1998). Three infants were excluded from the study because they awoke while their position was being changed. For the 22 infants included in the analysis, more intense auditory stimuli were needed to arouse the infants in the prone position (median of 70 db(A), range values 50 to more than 100 db(A)) than in the supine position (median of 60 db(A), range values 50 to 90 db(A)) (p=.011). Arousal thresholds were higher in the prone than in the supine position in 15 infants; unchanged in 4 infants; and lower in the prone position in 3 infants (p=.007). It was concluded that infants show higher arousal thresholds to auditory challenges when sleeping in the prone position than when sleeping in the supine position. The findings could not readily be explained. The difference in arousal thresholds could be related to difference in chest wall mechanoreceptor responses, or differences in blood pressure and/or central baroreceptors responses.

- Ambient room temperature

Two groups of healthy infants with a median age of 11 weeks were recorded polygraphically during one night: 31 infants were studied at 24°C and 31 infants at 28°C. To determine their arousal thresholds, the infants were exposed to white noises of increasing intensities during REM and NREM sleep (Franco et al. 2001). The arousal thresholds decreased across the night in the infants sleeping at 24°C (p= .017). The finding was not found for the infants sleeping at 28°C. When analysing the arousal responses according to time of the night, it was found that the auditory thresholds were significantly higher at 28°C (75+/-19 dB(A)) than at 24°C (51+/-3.5 dB(A)) between 03:00 hr and 06:00 hr (p=.003). These findings were only seen in REM sleep.

- Sleeping with the head covered by bedclothes

To evaluate the influence of covering the face of sleeping infants with a bed sheet, 18 healthy infants with a median of 10.5 weeks (range 8 to 15 weeks) were recorded polygraphically for one night (Franco et al. 2002). They slept in their usual supine position. During sleep, a bed sheet was gently placed on their face during 60 minutes. With the face free or covered by the sheet, the infants were exposed to white noises of increasing intensities during REM and NERM sleep. Compared to face free, during the face-covered periods, the infants had increases in pericephalic ambient temperature (p<.001), increases in REM sleep (p=.035) and body movements (p=.011) and a decrease in NREM sleep (p<.001). Respiratory frequency was increased in both REM (p=.001) and NREM (p<.001) sleep. With their face covered, the infants had higher auditory arousal thresholds (mean of 76+/-23 dB(A)) than whith the face free (mean of 58+/-14 dB(A)) (p=.006). The difference was seen in REM sleep only. A positive correlation was found between pericephalic temperature and arousal thresholds in REM sleep (r=.487; p=.003).

- Short sleep deprivation

Following short sleep deprivation, a study reported that in infants there was no measurable change in arousal propensity by auditory stimuli (1 kHz pure tone, delivered in the midline of the cot, from 73 dB and increased in 3 dB steps to 100 dB) during quiet sleep (Thomas et al. 1996). Another study was undertaken to evaluate the influence of a brief period of sleep deprivation on sleep and arousal characteristics of healthy infants (Franco et al. submitted). Thirteen healthy infants with a median age of 8 weeks (range 7 to 18 weeks) were recorded polygraphically during a morning nap and an afternoon nap in a sleep laboratory. They were two hours sleep-deprived, either in the morning or in the afternoon before being allowed to fall asleep. Six infants were sleep-deprived before the morning nap and seven before the afternoon nap. During each nap, the infants were exposed to white noises of increasing intensities in REM sleep to determine their arousal thresholds. Following sleep deprivation, the infants tended to have less gross body

movements during sleep (p = .054). They had a significant increase in obstructive sleep apneas (p = .012). The infants'auditory arousal thresholds were significantly higher following sleep-deprivation (mean of 76+/-13.5 dB(A)) than during normal sleep (mean of 56+/-8.4 dB(A)) (p = .003) during REM sleep. It was concluded that short-term sleep deprivation in infants is associated with the development of obstructive sleep apneas and a significant increase in arousal thresholds.

- Pacifiers and breastfeeding

Fifty-six healthy infants were studied polygraphically during one night: 36 infants used a pacifier regularly during sleep; 20 never used a pacifier (Franco et al. 2000). Thumb users or occasional pacifier users were not included in the study. The infants were recorded at a median age of 10 weeks (range 6-19 weeks). To evaluate their auditory arousal thresholds, the infants were exposed to white noise of increasing intensity during REM sleep. Polygraphic arousals occurred at significantly lower auditory stimuli in pacifier-users than in nonusers (mean of 60+-11.6 with pacifiers, for 71+-15.3 without pacifier; p=.010). Compared to nonusers, pacifier-users were more frequently bottle-fed than breastfed (p=.036).

Among infants sleeping without a pacifier, breast-fed infants had lower auditory thresholds than bottle-fed infants (mean of 67.7+-13.0 breast-fed, for 77.7+-17.5 bottle-fed; p=.049). The question of how a pacifier contributes to protect the sleeping infant might be best explained by the observed loss of the pacifier early after sleep onset. This could contribute to disrupt the infant's sleep and favor arousals.

1.3 Factors that modify auditory arousal thresholds in children: Conclusions

Various factors modify auditory arousal responses from sleep in healthy infants. Some inhibit arousals while others enhance the response. To evaluate the effect and dose-effect relationship on children therefore requires the careful determination of confounders that may bias studies and lead to conflicting results.

Additional confounders should be added to the list of factors that modify arousal thresholds. These include past experience with the stimulus (Rechtschaffen et al. 1966), or the presence of meaning in the noise as both of them are of critical importance in determining the persistence of physical reactions to the noise (McLean and Tarnopolsky 1977). These are the reasons which lead most sleep-wake researchers to use white noises to stimulate the sleeping child.

Knowledge of these variables does little to clarify the physiological determinants of the awakening response, because we have little better idea of how such variables are related to possible physiological determinants than we have for the awakening response itself (Rechtschaffen et al. 1966).

These findings however, underline the significant dose-response relationship between ambient noise and arousal or awakening from sleep in infants.

2 Noise and sleep for different stages of development

2.1 The fetus

The human fetus spends most of its time in a state equivalent to sleep, similar to that recorded in newborn infants. The healthy fetus in utero was shown to react to external noises. This is the result of the development of the human cochlea and peripheral sensory end organs. These complete

their normal development by 24 weeks of gestation. Sound is well transmitted into the uterine environment. Ultrasonographic observations of blink-startle responses to vibroacoustic stimulation are fist elicited at 24 to 25 weeks of gestation, and are consistently present after 28 weeks, indicating maturation of the auditory pathways of the central nervous system (Committee 1997). The fetus reacts to 1 to 4 seconds of 100 to 130 dB of 1220- to 15000-Hz sound. The hearing threshold (the intensity at which one perceives sound) at 27 to 29 weeks of gestation is approximatively 40 dB and decreases to a nearly adult level of 13.5 dB by 42 weeks of gestation, indicating continuing postnatal maturation of these pathways.

Teratogenic effects have been described in animals prenatally exposed to noise (Committee 1997). These were associated with higher levels of cortisol and corticotropin hormones in the exposed animals. No such effects could be demonstrated in humans, in whom studies on the relation between exposure to noises during gestation and shortened gestation or lower birth weights were inconclusive or conflicting. It is possible that in these studies, noise could be a marker of other risk factors (Committee 1997). In conclusions, most studies on the effects of noise on perinatal health have been criticised, as being hampered by serious methodological limitations, both in terms of the measurement of exposure and outcome, and failure to control for other known determinants of the outcomes under investigation. The lack of properly controlled studies makes it difficult to draw conclusions about which effects ambient noise have on perinatal outcomes (Morrell et al. 1997).

2.2 Newborn infants

A large number of investigations have been concerned with the responses of asleep newborn infants to acoustic signals. Many of the studies arise from a large and general interest in child development as well as from a need for hearing tests of infants (Mills 1975).

Infant incubators produce continuous noise levels of between 50 and 86 db (linear) (American 1974). Oxygen inlets produced an additional 2 dB (linear). Slamming of incubator doors and infant crying produced 90 to 100 dB(A) (American 1974). It was shown that inside incubators, background noise level is about 50 dBA and can reach 120 dBA (Committee 1997). Much of the energy is located below 500 Hz, between 31 and 250 Hz (Mills 1975).

Ambient noise appear to influence the quantity and quality of the sleep of newborns. Some newborns appear to be particularly responsive to ambient noises. Sleeping premature, anoxic, or brain-damaged infants detect intruding sounds better than sleeping, healthy, or term babies (Mills 1975).

Newborn infants spend most of their time sleeping. Some studies have documented hearing loss in children cared for in intensive care units (Committee 1997). Noise and some ototoxic drugs act synergistically to produce pathological changes of the inner ears of experimental animals (neomycin, kanamycin, sodium salicylate…). The relationship with the infant's clinical condition and associated treatments has however not yet been clearly defined. Infants exposed to sound levels of incubators are usually premature, on drugs, and in very poor health. Moreover, the exposures are continuous. A weak infant could spend weeks sleeping in such noise level without rest periods away from noise (Mills 1975).

High noise levels may be associated with other types of responses. In young infants, sudden loud noise (of approximately 80 dB) environmental noise induced hypoxemia.

Noise reduction was associated in neonates with increases in sleep time, in particular in quiet sleep (Committee 1997). It also resulted in fewer days of respiratory support and oxygen administration. Premature infants cared with noise reduction had a better maturation of electroencephalograms.

A Committee on Environmental Health of the American Academy of Pediatrics concluded that high ambient noises in the NICU changed the behavioural and physiological responses of infants (Committee 1997). For all the above observations and considerations, sound in infant intensive care units should be maintained under 80 dB(A) (Graven 2000). Among other recommendations, pediatricians were encouraged to monitor sound in the NICU, and within incubators, where a noise level >45 dB is of concern.

2.3 Infants (1 month to 1 year-old).

Some studies of the effect of external noises on the sleep-wake reactions of infants were conducted in their natural home environment. The reactions of babies to aircraft noise were studied by means of electroplethysmography (PLG) and EEG (Ando and Hattori 1977). The recordings were done in the morning, in the infants' sleeping rooms. The infants were exposed to recorded noise of Boeing 727 at take off. The noise was presented at 70, 80 and 90 dB(A) in the peak level at the position of the babies'heads. The subjects, who had not been awakened by exposure to aircraft noise, were exposed to music (Beethoven's 9th Symphony) in levels of 70, 80 and 90 dB(A). The frequency ranged between 100 Hz and 10 kHz. It was found that the babies whose mothers had moved to the area around the Osaka International Airport before conception (Group I; n=33) or during the first five months of pregnancy (Group II; n=17) showed little or no reaction to aircraft noise. In contrast, babies whose mothers had moved closer to the airport during the second half of the pregnancy or after birth (Group III; n=10 or IV; n=3) and the babies whose mothers lived in a quiet living area (Group V; n=8) reacted to the same auditory stimuli. The babies in groups I and II showed differential responses on whether the auditory stimuli were aircraft noise or music. Abnormal PLG and EEG were observed in the majority of babies living in an area where noise levels were over 95 dB(A). It was concluded that the difference in reactivity to aircraft noise may be ascribed to a prenatal difference in time of exposure to aircraft noise. The reactions diminished after the sixth months of life in group I and II, and the ninth month in groups III-V. This phenomenon may be explained as habituation to aircraft noise after birth. However, in all groups, no habituation occurred for a noise level over 95 dB(A) (Ando and Hattori 1977). This study was critized, as the authors did not adjust for several important determinants of birthweight, such as prematurity and the mother's age, weight, smoking status or socioeconomic status (Morrell et al. 1997).

Noise levels may be constantly high in pediatric units. The mean noise levels measured in a center of a surgical recovery room were 57.2 dB(A), while those measured at the patients'heads were 65.6 dB(A) (American 1974). In a medical unit (6-bed wards containing 5 infants between 3 and 17 months) peak sound levels were recorded on the pillow of the cot for 12 min (Keipert 1985). Infant crying produced 75 to 90 dB(A) and a beeper around 76 to 78 bB(A). Peak noise levels recorded at the nurses'station were about 78 dB(A) for telephone, 80 for infant crying, public address system, adult talking, and up to 90 dB(A) for child talking (Keipert 1985).

In a study was conducted on infants exposed to 50 to 80 dB(linear) in the range of 100 to 7,000 Hz (American 1974), a level of 70 to 75 dB (linear) for three minutes led to obvious disturbance or awakening in two thirds of the children. All infants awakened after 75 dB(linear) for 12 minutes.

In other studies conducted on the effects of awakenings and arousals, it was shown that white noise intensity was significantly lower to elicit polygraphically scored arousals than to induce awakenings (Franco et al. 1998).

2.4 Toddlers - Pre-adolescents (8 to 12 years old) - Adolescents (13 to 18 years old)

Developmental variations in auditory arousal thresholds during sleep were investigated in four groups of normal male subjects : children (n=6; 5-7 years old), preadolescents (n=10; 8-12 years old), adolescents (n=10;13-16 year old), and young adults (n=10; 20-24 years old) (Busby et al. 1994). Arousal thresholds were determined during NREM and REM sleep for tones (3-s, 1,500-Hz pure tones delivered in an ascending series of increasing intensity, 5-dB increments beginning at 30 dB SPL ("Sound pressure level") re 0.0002 dynes/cm2 until awakening or maximum intensity of 120 dB) presented via earphone insert on a single night following two adaptations nights of undisturbed sleep. Age-related relationships were observed for both awakening frequency and stimulus intensity required to effect awakening, with awakenings occurring more frequently in response to lower stimulus intensities with increasing age. In children, 43.1% of stimuli induced awakenings, in preadolescents 54.8%, adolescents 72% and adults 100% ($X2=60.37$; $p<.001$). Partial arousals (brief EEG desynchronization and/or EMG activity with the subjects returning to sleep) occurred in 9.8% of children, 4.8% of preadolescents, 12.2% adolescents, 0% adults. Although stimulus intensities required for awakening were high and statistically equivalent across sleep stages in non-adults, higher intensity stimulus were required in Stage 4 relative to Stage 2 and REM sleep. Frequency of awakening increased with age, whereas stimulus intensities required to effect these awakenings decreased with age. These relationships were maintained for individual sleep stages. These results confirm previous observations of marked resistance to awakening during sleep in preadolescent children and suggest that processes underlying awakening from sleep undergo systematic modification during ontogenic development. The observed resistance to elicited awakening from sleep extending up to young adulthood implies the presence of an active, developmentally related process that maintains sleep (Busby et al. 1994).

In another study, 5- to 7-year-old children were shown to be 10-15 dB less sensitive to pure tones than 22- to 30-year-old adults (Mills 1975). Another report on 8-12 year-old male hyperactive and normal children showed that these children were awakened with auditory stimulus intensity levels of up to 123 dB SPL ("Sound pressure level"), much higher than values reported for adults (range of 50-85 dB) (Busby et al. 1994).

In a study on 4 children (2 males), aged 5 to 8 years old on the effects of simulated sonic booms (68 dB(A) near the subjects' ears), 94.1% of the subjects showed no change, 5.9% had shallower sleep, but none aroused or had or behavioural awakening. In general, the frequency of arousal or behavioural awakenings and of sleep stage changes increased with age (up to 75 y) (Lukas 1975).

In a prospective longitudinal investigation, which employed non-exposed control groups, effects of aircraft noise prior to and subsequent to inauguration of a new airport as well as effects of chronic noise and its reduction at an old airport (6 to 18 month post relocation), were studied in 326 children aged 9 to 13 years (Bullinger et al. 1999). The psychological health of children was investigated with a standardized quality of life scale as well as with a motivational measure. In addition, a self-report noise annoyance scale was used. In the children studied at the two airports over three time points, results showed a significant decrease of total quality of life 18 month after aircraft noise exposure as well as a motivational deficits operationalized by fewer attempts to

solve insoluble puzzles in the new airport area. Parallel shifts in children's attributions for failure were also noted. At the old airport parallel impairments were present before the airport relocation but subsided there after (Bullinger et al. 1999).

In a study, the effects of ambient noise on autonomic responses could be demonstrated in children. In 6 to 12 year-old children exposed to intermittent traffic noise during four nights (at a rate of 90 noises per hour; peak intensity of the noise, 45, 55 and 65 dB(A) varied semi-randomly) and two quiet nights: heart rate was affected and relatively higher in noise during REM and Stage 2 than during delta sleep (Muzet et al. 1980, in Abel 1990).

2.5 Conclusions

Several studies on the extra-auditory effects of ambient noises on sleeping children were summarized. In relation to ambient noise, specific changes were reported in both sleep quality and quantity. Some of the effects were shown to have a dose-response relationship.

Several limitations to the present report should be discussed. Firstly, we do not know whether the inference that is often made that the effects of noise might develop with a longer exposure time (Abel 1990) is correct. Serious cardiorespiratory or autonomic changes, such as increases in blood-pressure could only develop following long-time exposure starting from childhood. This, in fact, has never been documented, nor has the extent of intersubject variability, due to difference in susceptibility. Secondly, we have no information to evaluate whether adaptation to ambient noise could limit the effects observed during short-term experiments. Thirdly, as the existing research data are applicable to generally healthy children, we do not know how the reported findings could be applied to ill children, children receiving medical treatments or very young premature infants. Finally, as most studies were conducted in laboratory controlled environments, we do not know the correlation between these studies and the effects of noise in the home. The multifactorial effects of environment on sleep and arousal controls could be much more complex than expected. On might predict that, as for adults, the effects of noise on the child's sleep and health are very complicated and depend upon the spectrum and level of the noise, temporal aspects of the noise, psychological responses to the noise, and the nature of the evaluation technique. The complexity of the conditions related to sleep-wake controls was illustrated by the review of confounding factors affecting auditory arousal thresholds.

Despite these limitations, it can be concluded that, based on the evidence available, the extra-auditory effects of noise could be pervasive, affecting the children's physical and psychological well-being. Changes in sleep quantity and quality together with autonomic reactions are seen when a child is exposed to ambient noise during sleep. Ambient noise exerts a dose-effect relationship on changes of sleep-wake behaviors. These reflect modifications induced within the brain of the sleeping child. It remains, however, to be determine what pervasive effects long-term exposure to ambient noise have on the child's development, health and well-being. Evidence should also be defined to support an enforcement of strategies for noise reduction at the source as suggested by some studies. Noise-induced health effects on children, a clinical and public health concern, should be evaluated by further studies.

AROUSAL AND AWAKENING IN CHILDREN
Review of the literature

N°	dB(A)	% responses	Type of responses	Reference
1	80	70	Nenonates motor response	Steinschneider 1967
2	60	5	Neonates startle response	Gädele 1969
	70	10		
	80	20		
	100	60		
3	60	7	Neonates startle response	Ashton 1967
	65	10		
	70	40		
4	80	70	Child awake	Semczuk 1967
5	100	70	Child awake	Busby 1994
6	100	76	Preadolescent awake	
7	100	86	Adolescent awake	
8	60	100	Adult	
9	120	72	Infant awake	Kahn 1986
10	75	75	Infant awake	Gädeke 1969

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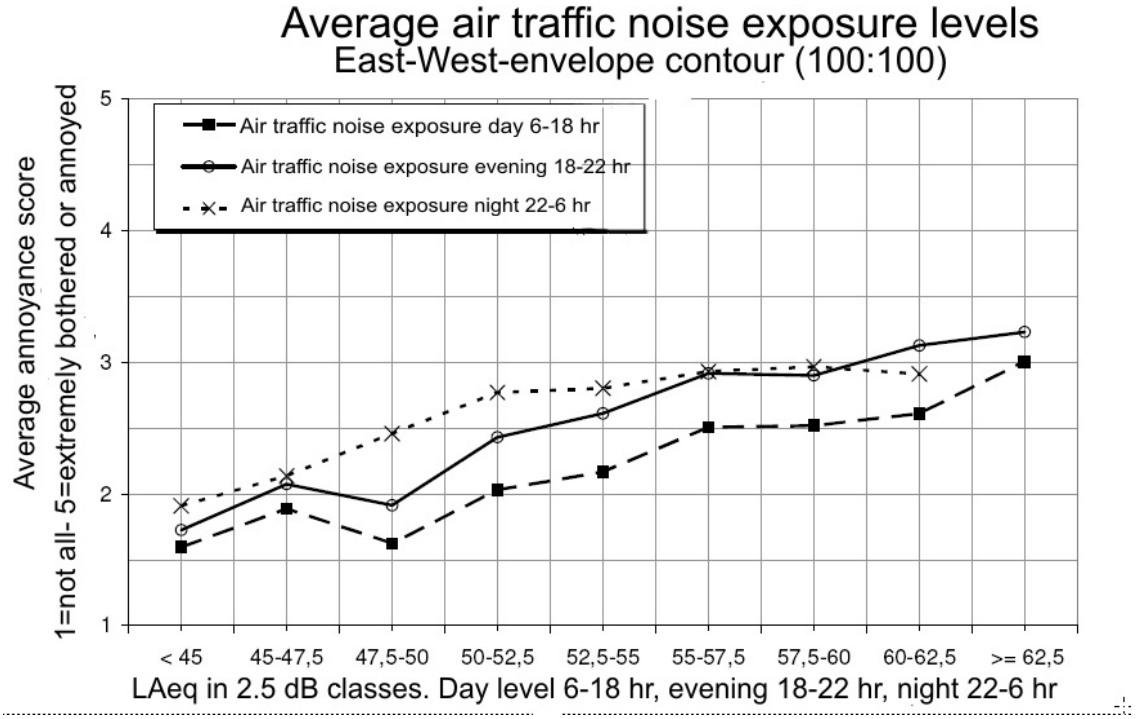
APPENDIX V Glossary of terms and acronyms

Term	Definition
Actimetry	The measurement of accelerations associated with the movement of an actimeter
ADHD	Attention-Deficit Hyperactivity Disorder
Behavioural awakening	Awakening that is registered by the subject by means of a conscious action.
CAP	Cyclic Alternating Patterns
EBD	Environmental Burden of Disease
END	Environmental Noise Directive (EC-2002/49)
EEG	Electro-EncephaloGram, recording of electric activity in the brain
ECG	Electro CardioGram, recording of electric activity of the heart
EEG awakening	Transition from a state of sleep to a state of consciousness, as determined by a sleep EEG.
Heart rate acceleration	A temporary rise in heart rate relative to the average heart rate assessed shortly before a noise event.
ICSD	International Classification of Sleep disorders
Insomnia	Sleeping disorder consistent with an internationally accepted definition (see ICSD), which takes account of difficulty falling or staying asleep, the daytime implications and the duration of the problems
$L_{Aeq,T}$	Exposure to noise for the duration of a given time interval *T* (a twenty-four hour period, a night, a day, an evening) is expressed as an equivalent sound pressure level (measured in dB(A)) over the interval in question.
L_{Amax}	Maximum outdoor sound pressure level associated with an individual noise event.
Lnight	Refers to the EU definition in Directive 2002/49: Equivalent outdoor sound pressure level associated with a particular type of noise source during night time (at least 8 hours), calculated over a period of a year.
Motility onset	The presence of movement in a short time interval, following an interval without movement.
Motility	The presence of movement in a short time interval, as recorded on an actigram.
OSA	Obstructive Sleep Apnea

OTC	Over the counter (medicins sold without prescription)
Polysomnography	The measurement during a subject's time in bed of his or her brain activity by means of EEG, EOG and EMG. The technique involves the use of electrodes to record electrical potentials in the brain..
REM	Rapid Eye Movement (sleep phase)
SEL	(sound exposure level) Equivalent outdoor sound pressure level associated with an individual noise event, with the equivalent level standardised at one second.
Sleep fragmentation	Within a sleep period, the frequency and duration of intervals of wakefulness recorded on a sleep EEG or intervals of motility recorded on an actigram.
Sleep latency	The length of time taken to fall asleep, i.e. the interval between the point at which a person begins trying to go to sleep or allowing him/herself to go to sleep and sleep inception time.
Sleep disturbance	Disturbance of sleep by night-time noise, as perceived by a subject and described in aquestionnaire response or journal entry.
Sleep EEG	Graph created using data from EEG scanning during a subject's time in bed, showing the various stages of sleep as a function of time.
Sleep stage change	Change from a deeper stage of sleep to a less deep stage, as determined by a sleep EEG.
SMR	Standardized Mortality Ratios
SROBD	Sleep Related Obstructive Breathing Disorders
SWS	Slow Wave Sleep (sleep phase)
UARS	Upper Airway Resistance Syndrome

Project result 3: Technical report on the night-weighting factor in L_{den}

The EU-indicators for environmental noise L_{den} and L_{night} were established with the Directive on Assessment and Control of Environmental noise, EC-2002/49. These choices were in turn based on the Position Paper of the Working Group on Indicators. The principle underlying the choice for 2 indicators was that L_{den} was proven to be a good indicator for long term effects (notably annoyance), while the L_{night} was introduced to predict long term effects on sleep.
The weighting factors in the Lden have not been introduced with the perspective of protecting sleep quality, but to fit the indicator for predicting annoyance. It cannot therefore be expected that research in the relation between sleep and health provide more insight in this. This is the reason that this topic is kept separate from the discussion around night time noise guidelines.
The working group studied the presence and the magnitude of the night weighting factor carefully, and concluded that the weighting factors of 5 and 10 dB should be added to evening and night LAeq's, respectively, to calculate a composite indicator.
The basis for this can be derived from a systematic study on the effect of different weigthing factors,[3] and a number of studies showing different sensitivities with respect to exposure at day, evening and night. In studies where only one source is addressed, the various LAeq-indicators are highly correlated. So for technical reasons the advantage of using weighting factors cannot always be demonstrated. The difference in sensitivity however is confirmed by a recent study in Germany (2006).[4]



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[4] Gutachten belästigung durch Fluglärm im Umfeld des Frankfurter Flughafens. Endbericht Regionales Dialogforum Flughafen Frankfurt, 30-6-2006.

The upper line is the average annoyance at night time against the night level, the middle line at evening, and the lower line day time. The differences at the middle of the scale seem not be far from the 5 dB at evening and 10 dB at night . At the high end the differences seem to decrease.

Annex 1: List of partners of NNGL project

• Université Libre de Bruxelles, established in Bruxelles, Belgium
• Queen Mary and Westfield College, University of London, established in London, United Kingdom
• TNO - Netherlands Organisation for Applied Scientific Research - INRO, established in Delft, The Netherlands
• CNRS-CEPA - Centre National de Recherche Scientifique Centre d'Études de Physiologie Appliquée, established in Strasbourg, France
• Univerzita Karlova v Praze 1 - lékařská fakulta, 1st Medical Faculty Charles University, Department of Neurology, established in Prague, Czech Republic
• CESUR/IST - Centro de Sistemas Urbanos e Regionais / Instituto Superior Técnico, established in Lisboa, Portugal
• Centre for Built Environment, University of Gävle, established in Gävle, Sweden
• Umweltbundesamt - Federal Environmental Agency; Department of Environmental Hygiene, established in Berlin, Germany
• Institut National de Recherche sur les Transports et leur Sécurité, LTE - Laboratoire Transports et environnement, established in Bron, France
• Landesgesundheitsamt Baden-Württemberg, established in Stuttgart, Germany
• ARPAT Dipartimento provinciale di Pisa, established in Pisa, Italy
• National Institute of Public Health, established in Copenhagen, Denmark
• RIVM - Rijksinstituut voor Volksgezondheid en Milieu; National Institute of Public Health and the Environment, established in Bilthoven, The Netherlands
• Institute of Hygiene and Social Medicine, University of Innsbruck, established in Innsbruck, Austria
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Annex 3: Report on the first planning meeting on night noise guidelines

Report on the first planning meeting on night noise guidelines

Bonn, Germany, 7- 8 June 2004

European Centre for Environment and Health
(Bonn Office)
World Health Organization Regional Office for Europe

ABSTRACT

This meeting was the first in the European-Commission sponsored project on night time noise guidelines, The project partners, experts and national government officers met to define the work plan and discuss organizational issues. They also allocated responsibilities, looked at the timetable, team coordination, the logistical and finance. The next step will be the production of background papers on the different identified themes and the second meeting is planned for March 2005.

Keywords
NIGHT NOISE
GUIDELINES
ENVIRONMENTAL HEALTH
EUROPEAN UNION
SLEEP

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Background

The project on night time noise guidelines (NNGL)" of the World Health Organization Regional Office for Europe (European Centre for Environmental and Health, Bonn Office) (ECEH), is carried out in partnership with the European Commission (EC) Directorate General Health and Consumer Protection (DG Sanco) and several Member States. Its purpose is to develop guidelines for night time noise exposure. This project will review the evidence of night time noise effects on health, and estimate the magnitude of the associated health risks.

The project will provide expertise and scientific advice to Member States, and propose guideline values for night noise both for short and long-term exposure. It will support the development of future legislation in the area of night noise exposure control and surveillance. The project will follow the WHO methodology for the development of guidelines.

Introduction

Partners of the project, experts and national government officers agreed the methodology to be followed to reach guideline values; the work plans, schedule, responsibilities, and team coordination and the health effects and various end points of night time noise exposure. They also discussed the question of additional contractual partners, and the metrics and exposure assessment.

Five of the project partners, Professor Andre Kahn, Professor Sona Nevsimalova, Dr Andras Muntag, Dr Marie Louise Bistrup and Dr Danny Houtjuis were excused for other professional arrangements.

Summary of the meeting

The meeting was introduced by WHO: Mr Bonnefoy presented the background, the goals of the night noise guidelines project, described the budget, the envisaged role of the partners and possible ways of filling the gaps of expertise in the group; Ms Rodrigues presented the meeting objectives and agenda and Dr Krzyzanowski presented the experience gained by WHO when developing the air quality guidelines and how this experience can help on the development of the night noise guidelines. Mr Bonnefoy gave some examples during the meeting on the experience gained during the development of the drinking water guidelines.

The participants were invited to present the results of their work for the guidelines at the Vilnius housing and health symposium that will take place in October 2004. This symposium could be a good opportunity to have the partners discussing the work progress.

After these general presentations the floor was given to the project partners, followed by comments and questions. The invited representatives for the European Commission were also invited to express their opinion on the project and to give their feedback on how it can help them when developing legislation.

Two working groups were set up to describe the sequence of work and identify who would be responsible for each work package.

Presentations

Dr Michal Krzyzanowski presented the process followed when designing the WHO air quality guidelines and made suggestions for the development of the night noise guidelines. His presentation concentrated on the review of the evidence in order to avoid external criticism. He proposed a scheme for the review for night time noise and for deriving guidelines (figure 1).

The development of the air quality guidelines underwent several steps of analysis and review of scientific data in the field of air pollution toxicology and epidemiology. In-depth reviews of the effects of each pollutant were prepared, followed by a discussion based on this review, and then finally the guideline values were established. A scientific advisory committee was established for reviewing the literature and the background documents.

Dr Krzyzanowski suggested that the first step for developing the night noise guidelines should be to carry a comprehensive literature review according to the approach and method described in the WHO publication “Evaluation and use of epidemiological evidence for environmental health risk assessment”. These reviews should be the background for group discussion and the basis of the guidelines document.

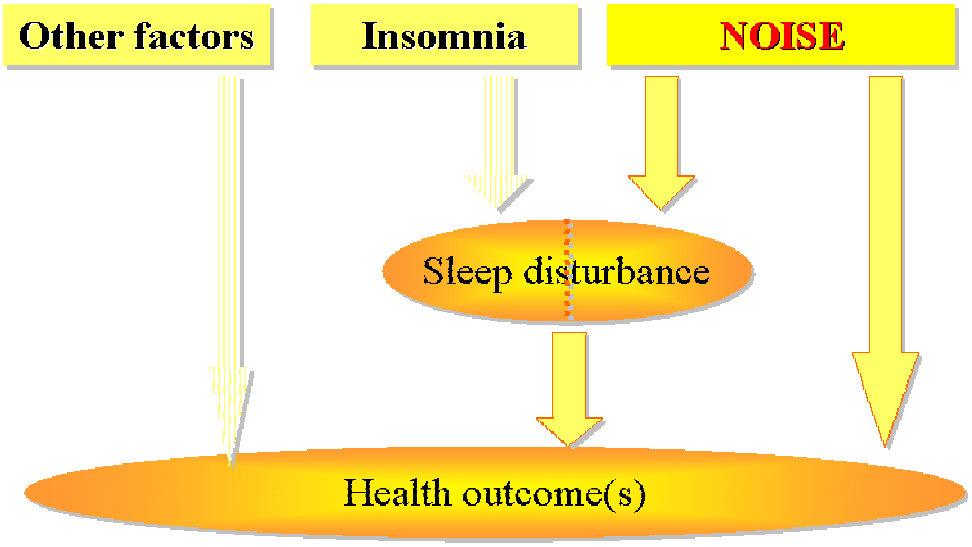

Figure 1 – A possible review of the noise epidemiological evidence on health

Partners' presentations

Dr Gaetano Licitra – Characterization of the different noise levels, sources, metrics

European Union Member States are starting to produce noise maps for their main cities. There is still no clear information on the percentage of population covered by the Directive 2002/49/CE in every Member State. Tt is difficult to outline the extent of the problem of night noise disturbance.

Another difficulty is that noise maps will neither cover neighbourhood noise nor leisure activities, so we may not get a full picture of the problem. Neighbourhood noise is very important because it is often the source of large number of noise events during the night. There is no accurate assessment of the impact of neighbourhood noise in many countries, mainly because it is highly dependent on the normal local behaviour of the population.

Dr Licitra suggested that the members of the NNGL group should be informed of the trends and predictions on the noise levels as well as the action plans carried by some countries for noise reduction. European projects such as CALM or STAIRRS, SOURDINE, IMAGINE, HARMONOISE could provide important input. Cooperation should take place with different stakeholders such as the European Environment Agency, research institutions, European Commission Working Groups and DG Environment.

Dr. Stylianos Kephalopoulos – Human exposure

Dr Kephalopoulos raised the importance of analysing the problem with "human beings" at its centre, and examining the interaction between the different stressors. A concept of total human exposure assessment, similarly to that used in indoor air quality, should be used. When looking at the effects of noise, the effects of other pollutants present at the time of exposure, should also be considered with time activity patterns. There is also uncertainty resulting from the fact that all measures are performed outdoors..

A three-step approach can be considered:

i) Define personal exposure (dose per person)
ii) assess the risk and
iii) evaluate the health effects resulting from the possible combinations.

Knowledge is constantly changing .The guidelines should be able to adapt well to changes in time and developments in science. This is achievable with a consistent framework.

Professor Peter Lercher - exposure assessment; sensitive groups

Professor Lercher acknowledged four major challenges for the assessment and future developments of regulation in the noise field:

i) the problem of sources combination assessment: what we know about today is mostly individual noise source,
ii) mono-sensory assessments are made even if other sensory qualities are present.
iii) the fact that data only exists for "steady" conditions and
iv) the generalization across context. Examples from two very different settings were given (Vienna and Alpine Valley) for dose-effect relationships for annoyance. There are significant differences between the two of them.

There was discussion about the metrics and about which indicators serve better for night-time assessment. Should average level indicators to be used? Some examples were shown of differences in the assessment when using several metrics.

Many factors influence the way in which nose is perceived, pre-dispositions such as noise sensitivity, family history of hypertension, vegetative lability. Some diseases can also make a difference, such as depression and cardiovascular disease. Circumstances are relevant too, such as whether there are combined exposures, whether the noise is low frequency, and the differences between background and peak levels. Among the groups that are susceptible of being predisposed to sleep problems are elderly, pregnant and peri-menopausal women; people with primary insomnia, and co-morbid mental/physical mental diseases; and circumstances such as the environment (temperature, EMF's, etc.) and stress. This complexity will have to be considered when developing simple figures.

Professor Alain Muzet - Identification of relevant adverse health effects

Professor Muzet has classified the possible adverse health effects into three categories: major sleep disturbances which are equivalent to insomnia and include awakenings, moderate sleep disturbances, and other disturbances.

Moderate sleep disturbances include acute responses like sleep stage changes and arousals. The long term effects of these responses are not completely known. What should be the approach to evaluate this impact? What can the guidelines say on this?

A third group of responses occurring during sleep includes all the psychological functions, heart beat rates, body movements, etc. These responses are increasing during sleep, do not habituate to noise, and can lead to damage (evidence exists in laboratory studies).

In summary, Professor Muzet suggested to look at the following effects:

Major sleep disturbances:

1. Lengthening the time to fall asleep;
2. Awakenings from sleep;

3. Too early wake-up time.

Moderate sleep disturbances:
1. Arousals;
2. Sleep stage changes;
3. Reduction in time spent in specific sleep stages:
    - Slow wave sleep
    - REM sleep
4. Sleep quality.

Other disturbances occurring during sleep:
1. Body movements;
2. Autonomic responses;
    - Change in heart rate;
    - Vasoconstriction;
    - Short-lasting increase in blood pressure;
    - Change in respiratory rate.
3. Hormonal responses.

Dr Ivanka Gale

Dr Gale's presentation concentrated in the legal situation concerning environmental laws in the Slovenia. Slovenia became independent in 1991 and since 1996 they have been adapting their legislation to the European Union. At present they have to consider the implementation of harmonized noise exposure indicators, target limit values, dose/effect relationships, modern noise assessment methods and noise mapping.

Dr Staffan Hygge – Effects of noise exposure on different cognitive tasks across age groups

Dr Hygge suggested that separating the different types of memory according to Tulving's memory theory, allows a better assessment of the impact of noise on cognitive functions. Tulving separates the procedural memory, perceptual representational system, semantic memory, primary memory (working memory) and episodic memory. Dr Hygge showed results on semantic memory with quietness, road noise and other people talking. Noise has a strong influence on this type of memory. For day-time noise the ages of 13/14 years are important because that seems to be when changes happen in cognitive development. Most studies on effects on children are for day time noise, Dr Hygge has proposed looking at the night time noise levels from the Munich and RANCH studies, provided the project can identity special funding for this.

Dr Snezana Jovanovic – Sleep disturbances and accidents

Dr Jovanovic proposed carrying out a literature review on sleep and accidents. She will look at insomnia and analyze daytime fatigue, sleepiness and drowsiness as triggers of accidents. Home, road, work, and school accidents will be covered.

Professor Stephen Stansfeld - Sleep and Psychiatric disorder

Professor Stansfeld focused showed data for insomnia and psychiatric disorders. Thirty three per cent of insomnia in community samples is related to a primary psychiatric disorder and seventy five per cent of sleep clinic patients have a primary or secondary psychiatric disorder.

An association between sleep and psychiatric disorders is pertinent because:

i) Structural/biochemical abnormalities in psychiatric disorders also influence sleep regulation
ii) Sleep disturbance is one of the primary symptom of psychiatric disorder
iii) Psychiatric drugs can disturb sleep
iv) Primary sleep disturbances often have associated psychiatric symptoms

Sleep disturbance is an important and common problem in psychiatric disorders. It is an aspect of psychiatric disorder that needs treatment. Investigating this area will be very useful for assessing the impact of noise on mental health.

Dr Michał Skalski – health effects of insomnia

Transient insomnia expresses itself by sleep deprivation and data exists that facilitate the evaluation of the health impacts. Chronic insomnia is different and very little data exists. Sleep disturbance leads to chronic insomnia that, in turn, can lead to some psychiatric disorders.

Transient insomnia has as consequences an increase in daytime sleepiness, impairments of psychomotor function and fatigue.
Chronic insomnia is different from sleep deprivation, it doesn't provoke daytime sleepiness and impairments of psychomotor function but it provokes hyper arousal (autonomic nervous system such as hormone secretion from the hypothalamic-pituitary-adrenal axis – HPA) and leads to psychiatric disorders (depression, anxiety, drug and alcohol abuse).

The risk of accidents and depression are strongly linked with insomnia. The use of this clinical knowledge will be crucial for evaluating the long-term noise effects on sleep.
Insomnia also affects children's cognitive development as they show worse results in learning. The quality of life for insomniacs is decreased and their accident rate is by 4.5 times that of "normal sleepers".

Dr Wolfgang Babisch – Relevant adverse health effects

Dr Babisch advised the group to try to keep decision-makers in mind.. Decision-makers want to know on one hand what is safe and on the other hand they do not want to jeopardize economic development and people's mobility on the other. There are obvious conflicts between quietness and mobility for example.

He presented valuable information on availability of studies, existence of evidence, and scenarii with different metrics and he introduced the few new studies on noise and health. This overview of the existing literature helped guide the group's technical work.

As an example, table 1 provides an overview of the state of the art on the availability of data for the most important effects.

Availability of data		
Effect	**Day**	**Night**
Annoyance / Disturbance	Yes	Yes
Cardiovascular effects	Yes	Infer*
Stress hormones	No	Yes
Electrophysiological sleep	N.R.	Yes
Performance at work	Yes	No

Table 1 – Availability of data for the different effects and noise exposure during day and night time exposure (Babisch 2004)

* - Not enough data is available for night time noise and inferences can be made from studies that considered only daytime measurements of 24h measurements.

For biochemical changes the existing evidence, according to expert's judgments, is "limited", for hypertension and ischaemic heart diseases "limited / sufficient" (using the International Agency for Research on Cancer definitions of „no – insufficient – limited – sufficient" evidence) for night time noise.

Professor Jacques Beaumont, Professor Joao de Quinhones Levy - Noise and health indicators - Evaluating exposure

In the European Union (EU) there are a lot of people exposed to noise, but there is a lack of knowledge about the characteristics of urban noise and the current assessment of noise in the urban context is poor. The key question is therefore how to evaluate this important exposure.

A "good" noise indicator should reflect the energy (Lden), the emergences - temporal and tonal (Lmax,SEL…), the nature of the sources (human activities / mechanical activities). It should be usable depending on time, and geographic scales, classification of the noise situations, acoustical and perceptive characteristics and urban morphology.

A pertinent indicator should consider:

- Perception: being representative of key noise characteristics from the perception point of view;
- Forecasting: suiting real or forecast noise situations;
- Measurability: a standardized measurement method has to be drawn up;
- Computation: to forecast a noise situation, a computation tool must be available;
- Communication: allowing efficient communication between decision makers, infrastructure managers and the public-

Dr H.M.E. Miedema

Dr Miedema identified six points that he considers crucial during the development of guidelines:

1. Which noise sources are we dealing with?
   Will only transportation sources be considered? Construction and demolition noise are increasingly a source of complaints all over Europe.

2. Which health effects are we going to consider?

3. Classification of the evidence (how to use indirect evidence);

4. Identification of sensitive groups;

5. Noise metrics (number of events, sound insulation);

6. Causal chain and subcortical effects (cortex mediated or non-cortex mediated).

Invited Member of Member States and European Commission presentations

A representative of the European Commission (Directorate General of the Environment) and representatives of environment and health Ministries from France, the Netherlands, Germany and Switzerland were invited to present the work on noise on their countries and to express their opinion on how the guidelines could support their legislative work.

EC - DG Environment

The directive 2002/49/EC for environmental noise proposes 2 indicators Lden (day-evening-night level) and Lnight (night level) as indicators for monitoring noise. These two indicators will be used in the strategic noise maps and be the basis for the development of noise reduction and prevention plans in major urban areas as well as around major transport infrastructures. These common indicators will also enable strategic noise maps to be used at national and EU level to show respectively the number of people annoyed (via Lden) and sleep-disturbed (via Lnight). The first maps have to be produced in 2007. Hence, night-time noise guidelines should, as a priority, consider at least these two EU indicators.

Nevertheless, the directive mentions for special cases the use of appropriate supplementary noise indicators. The article 5 of the directive stipulates "Member States may use supplementary noise indicators for special cases such as those listed in annex I(3)"". Maybe this will be the case for the night time guidelines but its use has to be well justified. The annex I(3) of the directive mentions the LAmax and a number of events as adequate supplementary indicators for night time noise peaks, notably from passing trains and aircraft. Given the trends in the growth of night-time volumes of traffic and relative noise exposure around major airports, highways and high speed train lines, these indicators should also be taken into account.

Mr Delcampe suggested that the NNGL group interacts and exchanges information with the Commission working group on health and socio economics aspects and considers the work developed in the position papers which can be downloaded from http://www.europa.eu.int/comm/environment/noise/home.htm , where the reports of the various noise working groups set up by the Commission are available.. In particular he recommended linking with the position paper on EU noise indicators and the one on relationships between transportation noise and annoyance.

France

L'AFSSE, Agence Française de Sécurité Sanitaire Environnemental has recently produced a report on the health effects of noise ("Les effects sanitaires du bruit – rapport d'étape") in French, not yet published. This report would be useful for developing the guidelines.

Mr Grénetier raised the problem of metrics; in his opinion the measurement of an average level is not suitable for assessing night effects. We would like to have more information on short and

long term exposure. The current WHO guidelines on noise don't respond very well to the demands for legislation. For example the number of events is missing.

Important aspects to be considered for a noise indicator should, according to the French representative, include:
- Number of noise events;
- The time they occur during the night;
- Duration of events;

Mr Grenetier advices the group to consider a synthetic indicator that takes into account all these parameters.

The Netherlands

A Dutch committee from the Ministry of Environment produced a document that covers all aspects of night noise exposure for advice to the Dutch Health Council (ready in June 2004). This document introduces the Lnight in the Netherlands, presents the health effects from noise and how they relate to other effects and proposes measures to reduce these effects. It will be public by the end of summer 2004. This document was considered important for the work of the NNGL group, but it is only available in Dutch. Mr Van Den Berg will explore the possibility of translating it into English.

The working group on health and socio-economic aspects established by DG Environment has produced a position paper on Lnight that is now open for comments from the Member States. This document could be an important input for the work of the NNGL group.

Dr Van Den Berg presented data on evidence of noise health effects from laboratory and field studies and some differences between countries on sleeping hours and levels recommended in national legislation.

Germany

The representative of the German ministry of environment would like WHO, and the guidelines document in particular, to provide advice and guidance on the following points:

i) Description of night time noise exposure in terms of the sources of noise and the particular characteristics of the noise sources;
ii) Assessment of the different effects of night noise on sleep;
iii) Development of criteria for a "reasonable" protection of night time sleep.

Dr Brüggemann recommended that WHO doesn't restrict itself solely to the prevalence and incidence of noise-induced illnesses. It should examine the full spectrum of effects produced by night time noise, including the impact on sleep that occurs before the possible onset of health risks, as well as other noise-induced disturbances.

It is generally acknowledged that uninterrupted sleep is of particular importance for a person's health and well-being and for their sustained working capacity. Nonetheless, it is difficult to avoid the impression that the degree of impact that noise has on health frequently tends to be underestimated by the public and by politicians. The creation of the WHO guidelines should contribute to a more intensive discussion and a more realistic perception of the situation.

There are real differences of opinion in the relevant scientific disciplines with regard to the assessment of various sleep disturbances caused by noise. It is therefore little wonder that the informative value of existing studies on the effects of night time noise is judged very differently, not only by the various disciplines, but also by individual scientists.

It does appear, however, that a broad consensus has now emerged, according to which the description of night time noise cannot be based on an average value alone. The noise indices used, however, continue to differ in various aspects. He would therefore very much welcome any moves contained in the guidelines toward greater standardization in this regard as well.

Currently, various legislation being considered in Germany deals with the subject of environmental noise control. Protection against night time noise is an important element in all of these. In terms of air traffic noise, an amendment to the Air Traffic Noise Act is currently being prepared. This will for the first time include limit values for protection against night time air traffic noise. If these values are exceeded, victims will be entitled to the installation of sound insulating windows and ventilation. Issues of night time noise control also feature prominently in the forthcoming implementation of the EU Environmental Noise Directive.

Political and administrative bodies do not, however, deny the financial burden created by noise reduction measures and demand that clear priorities are set. The task is therefore to develop scientific criteria for such an exercise and justifications for the necessary noise abatement measures.

In view of these points, and for the focused future development of noise abatement policy in its entirety, there is a particular need for advice on the following subjects, which should be examined in depth as part of the current project and feature clearly in the guidelines :

i) (Quantified) effects of different doses of noise in terms of sleep disturbance
ii) Assessment of the different types of effects that night noise has on sleep, not only with regard to the acute effects, but also taking account of possible long-term consequences
iii) Development of recommendations for the protection of night time sleep, if possible in the form of control standards.

The primary aim of the project should therefore be to help achieve a consensus regarding the scientific assessment of existing study findings, including the epidemiological evidence of the results. It is time to reach a more unified perspective on this point. The results of WHO's work and its recommendations will then be of the utmost interest for legislators in industrial nations and will potentially have a considerable impact.

Switzerland

The representative of the Swiss Agency for the Environment, Forests and Landscape gave a general overview of the limits for aircraft noise at during night, presented the daytime and night time exposure limits for Switzerland, and discussed the importance of night noise guidelines for policy making.

In Switzerland the night is defined from 22:00 to 06:00 and there is a flight ban from 0:00 to 05:00. The establishment of Swiss limit values of daytime and night time exposure involve identifying three levels of noise: planning level, Impact threshold and alarm value. For the day time they are respectively (for a sensitive level II) 57, 60 and 65 dB; for the night time they are 47, 50 and 60. For aeroplanes they calculated a matrix considering Lmax, Laeq and number of over flights.

Switzerland has a special interest in participating in the project because:

i) The project's scope corresponds with their regular evaluations of the latest studies;
ii) It will allow international harmonization of methods for setting limit values and for noise abatement;
iii) It will help harmonization with the European Union environmental policy. Although Switzerland is not an official Member of the EU, it sits in the heart of Europe and always harmonizes its environmental legislation with the EU.).

General discussion and working groups

After the presentations the group agreed to separate in smaller groups according to expertise and fields of knowledge. Two working groups were created, each with the task of identifying the working papers needed and who should be responsible for each of them. The groups also discussed the topics where not enough expertise exists in the NNGL group for completing the work. The first group concentrated in epidemiology and medicine and the second on physics and physiology (with a big component on exposure assessment and metrics).

Working Group I – Epidemiology and medicine

The following themes were identified for background papers:

1. Methodology for deriving guidelines– Dr Martin Van den Berg
2. Overview of existing epidemiological evidence for cardiovascular effects – Dr Wolfgang Babisch
3. Cognitive development of children and night time noise – Dr Staffan Hygge
4. Accidents – Dr Svena Jovanovic
5. Sleepiness / drowsiness – Dr Ivanka Gale
6. Depression/anxiety
    a. Night noise and mental health – Dr Stephen Stansfeld
    b. Sleep disturbance and mental health – Dr Michal Skalski

7. Animal studies / biological plausibility – Dr Ising (subject to his agreement)
8. Risk groups (children, pregnant women, elderly, stressed people, …) – review of the literature - Dr Willy Passchier-Vermeer (subject to his agreement)
9. Stress hormones - Dr Christian Maschke (subject to his agreement)

The group did not reach an agreement on the importance of producing a new meta-analysis for cardio vascular effects. This project on new studies, mainly in autonomic responses, had been set up after the existing meta-analysis. The opinion of Dr Danny Houthuijs lwould be sought on this matter.

Habituation will be covered in each of the papers.

Working Group II –Physics and physiology

Dr Henk Miedema was the moderator of this group. He asked each of the participants about their understanding of the process and about their willingness to contribute to it.

Professor Muzet presented the health effects that should be considered. He raised the point that the evidence on acute effects is mainly on the general population, consequently the groups that can be at a greater risk are not covered. We should also consider the subjective feelings and the biological measures of acute effects. The date on biological responses is adequate but it comes mainly from laboratory studies that don't include subjective feelings.

There are significant differences between field and laboratory studies that have to be taken into consideration. There is also a shortage of studies on the long term effects (e.g. reduction of sleep time, fatigue, drowsiness, increased accidents, …).
From the literature, threshold values for awakenings can be derived, but it is difficult to have clear figures for the other responses.

Professor Peter Lercher mentioned the temporal variations of noise levels during the night and spatial country differences. How do the existing studies deal with these variations? There are variations between Member States in time pattern; Not only the number of events should be considered but also the time they occur during the night - if there are studies. Differences on sleep habits should be considered: what is the significance of the different definitions of night-time in the different countries? Which index has been used in the studies examining the differences and to ensure the protection of quiet areas and sensitive areas (rural or urban)?

Long-term health damage is a too vague a concept according to Dr Hans Bögli. The "damage" should be accurately defined. What are the causes? To harmonize the evidence a score system could be designed to rate the damage, how important is the damage? This exercise would allow comparing the risks and deciding upon the priorities, as it would bring different effects on a single scale.

The risk groups are important, but they should not be the focus of the guidelines. Having this as main point might sidetrack the work from the scope of the guidelines, it is better to focus on the majority of the population and to consider the special cases in a separate chapter.

Professor Jacques Beaumont focused his presentation on noise metrics, distribution of energy during the night, the importance of measuring peak levels and in the existence of sensitive areas and sensitive periods. Lden is very good for a steady level, but during the night the energy level occurs very often during a short period of time and has a big impact. Methods exist to calculate these levels, but the impact assessment seems to be missing.

France (INRETS) has a lot of studies in airports (e.g. night time Roissy), not very recent, using LAeq, the calculations of peak levels would be possible.
The use of indicators that describe the short high energy events that occur during the night need to be discussed: how can we use them for evaluating the effects?

The most important role of the guidelines should be preventing people being exposed to noise levels that provoke awakenings and adverse effects, according to Mr Nicolas Grenetier. France uses Laeq, 8 hours and this is not representative of the situation, they would like to have additional characteristics on peak levels included in their metrics. Effects as awakenings are important and should not be dropped.

Dr Gaetano Licitra suggested the use of the DPSEEA schema as a frame for producing the guidelines. There is a need for a better metrics, the current legislation only covers noise outdoor and is not well connected with the health effects (indoor levels are more important for describing night time). It's not particularly difficult, but an agreement must be reached.

Dr Stelios Kephatopoulos discussed again the problem of uncertainty in exposure assessment, the sensitivity accuracy in input data, sensitivity to the choice of calculation points and the conversion of this uncertainty in exposure to uncertainty in effects;
in the expert's opinion, the answer to this problem can be outlining a framework, to help to deal with uncertainty in assessing the risk. It will also avoid the document becoming quickly obsolete and ensure that the information it contains will be digestible. A coherent framework can guide the Member States how to make use of the guideline values.

Dr Henk Miedema would like to contribute on the effect side, instantaneous and 24 hours effects with specific attention to mechanisms and possible health effects.

The results of the discussion were to have two bigger themes and two groups producing the background papers:

1. **Noise exposure description** – Professor Lercher, Dr Licitra, Professor Beaumont, Dr Kephalopoulos, Professor Levy
2. **Instantaneous and 24 hour-effects with specific attention to mechanisms** – Dr Miedema, Professor Muzet

Other points discussed – expertise required from experts outside the group

Some points identified as very important and crucial to be considered in the guidelines are not in the field of competence of the project partners. The group suggested they will require input from outside experts The secretariat will produce an outline of the background papers' table of contents and distribute it for discussion.

Neighbourhood noise

A discussion took place on including neighbourhood noise in the guidelines. The project proposal defines the sources as transportation but it could include this source as well.

Even if neighbourhood noise is a very particular noise source and guidelines values will not consider it, this is one of the main sources of people's complaints. The group decided by consensus that even if it will not have guidelines values for it, attention should be paid to this source reflecting the different sources of neighbourhood noise (ventilation, indoor noises, neighbours, etc), the characteristics of housing and how this noise could be mitigated, etc.

The group gave some names for possible authors; the secretariat will contact them and identify the author.

Animal studies

Even though noise effects have a strong component of perception and are strongly related to psychological factors, the studies developed in laboratories with animals could be reviewed in the guidelines.

Conclusions and follow up work

In the plenary session on the last day of the meeting, the group established and agreed on the follow up actions, timetable and key milestones. The main conclusion was that the process would start with the production by the project partners and other experts, of background papers on the different themes identified during this workshop.

These background documents will not be size limited, and should contain as much information as possible on their specific topic. The assigned experts will have the freedom of covering a large range of data, risk assessment, dose-effect relationships and studies. These will be the foundation of the guidelines document therefore no limitations are necessary at this stage. The crucial point is to ensure that the existing knowledge is covered.

Regarding the work already existing in Member States, Dr Van de Berg will explore the possibility of having the "Dutch report" on noise translated to English as well as Mr Grenetier for the French one. If the complete reports cannot be translated, efforts will be made to have the summary available in English.

The production of a meta-analysis will depend on having an expert opinion. Dr Danny Houthuijs will be consulted regarding the importance of having a specific meta-analysis for night noise or/and focusing on children.

Sequence of tasks and time table

The secretariat will distribute the meeting report for comments on the group. Partners should feed back on their attributed background documents. Secretariat will produce the terms of reference for external background papers – **End of June 2004**

The partners should start writing the papers feed backing with the group if necessary. By the **15th October** the secretariat will produce a progress report.
The papers should be ready for internal review by **December 2004 / January 2005.** This review should be finalized by **March 2005.**

The second partners' meeting is planned for **March 2005.** This meeting will have as main result a decision on how the working papers should be summarized, and plan the process of writing the guidelines.

By **December 2005** a first draft of the guidelines will be ready and the third meeting will happen in **January 2006,** this will be a partner consensus meeting, and the process of external review by peers and all stakeholders will be initiated.

In March 2006 the fourth partners' meeting will be held and agreement will be reached on the final version of the document for presenting in **June 2006** to Member States.

Small meetings with partners working in similar subjects could be held between the identified periods.

Annex 1

LIST OF THEMES AND RESPONSIBLES

Themes	Responsible
1. **Setting the scene** Sources, metrics, sensitive areas, number of people exposed, trends, number of events, ,variations during the night, overview of legislation….	Lercher, Licitra, Beaumont, , Levy
2. **Uncertainty in exposure**	Kephalopoulos
3. Instantaneous effects Major sleep disturbances, moderate sleep disturbances, other	Muzet, Miedema,
4. Cardiovascular effects, Hypertension	Babisch
5. Immune system (hormones excretion, decreased glucose assimilation, …)	Maschke (Depending on the expert's agreement)
6. Other health outcomes ,	
a) Physical (fatigue, drowsiness, sleepiness, …)	Gale
b) Cognitive impairment (deterioration of performance, attention and motivation and diminishment of mental concentration and intellectual capacity and, increases the chances of accidents at work and during driving,…)	Hygge
c) Accidents (this point needs special attention although covered in a) and b)	Jovanovic
d) Mental health	Stansfeld, Skalski
e) Sleep pathologies	Nevsimalova (Depending on the expert's agreement)

7. Animal studies	Ising (Depending on the expert's agreement)
8. Scoring the evidence	Boegli
9. Guidelines derivation, methodology aspects	Van den berg
10. Risk groups	Bistrup, Kahn (Depending on expert's agreement) Passchier Veermer
10. Meta-analysis? **The expert will be asked on the relevance of this point**	D. Houthuijs (if of interest)
11. Neighbourhood noise (night)	Colin Grimwood (Depending on expert's agreement)

Annex 2

WORKING DOCUMENTS

Theme 1. Proposed elements of a 1st background paper on night noise exposure assessment

Group members

Jacques Beaumont - Transport and Environment Laboratory, French National Institute for Transport and Safety Research
Stelios Kephalopoulos – EC Joint Research Centre, Institute for Health & Consumer protection, Physical and Chemical Exposure Unit
*Peter Lercher** – Innsbruck Medical University, Institute of Hygiene and Social Medicine
João de Quinhones Levy - Instituto Superior Técnico, University of Lisbon
Gaetano Licitra – ARPAT, Departimento provinciale di Pisa
** Rapporteur*

1. Rationale for tight regulation of night noise exposure
   - Increase in night noise exposure?
   - Spread of night noise to previous quiet and/or sensitive areas?
   - Recent results of sleep research
   - Increase of the pool of susceptibles?
   - The open doors of the END

2. Noise metrics for characterization of night time noise exposure
   - Directive 2002/49/EC
   - Other metrics
   - Qualities needed for best description of night noise exposure with regard to human health effects
   - Noise metrics used in short-term health effect studies
   - Noise metrics used in long-term health effect studies

3. The structure, distribution and measurement of night noise exposure across Europe
   - Types of night noise exposure
   - Continuous noise exposure
   - Intermittent noise exposure
   - Other forms of noise exposure
   - Types of night noise used in short-term human health effect studies
   - Types of night noise used in long-term human health effect studies
   - The uncertainty in estimating the various exposure types

   - Difference between most and least exposed façade
   - Measurement approaches
   - The quiet backyard ?
   - Uncertainty involved in estimating different façade exposures

   - Indoor versus outdoor night noise exposure

Measurement of indoor night exposure
Comparisons across Europe
The uncertainty in estimating the actual night exposure at the ear of the sleeper

Night noise in special areas
Sensitive areas
Definition of sensitive areas
Noise characterization in sensitive areas
Quiet areas
Definition of quiet areas

Overall uncertainty in noise exposure assessment
Sensitivity to accuracy in input data
Sensitivity to choice of calculation points
Sensitivity to choice of noise metrics

Transfer of uncertainty in exposure assessment to uncertainty of effect assessment
Uncertainty due to inaccuracy in measurement data
Uncertainty due to inaccuracy in calculation points
Uncertainty due to the use of inaccurate noise metrics

4. The protection of the night against noise across Europe

The behaviour of populations during night time

Existing guidelines
Description of the variations
The limits of current legislation

Current legislation
Description of the variations across countries
The limits of current legislation

The need for harmonization of protection of nights against noise exposure across Europe

5. Proposal for accurate night noise exposure assessment

In short-term studies
In long-term studies
In noise mapping, SEA and EHIA
In sensitive areas
In quiet areas

6. References

7. Appendix

Theme 2. - “Short-term effects of noise on sleep with specific attention to mechanisms and possible health impact”.

Group members
H. Miedema and A. Muzet

This paper should include three particular aspects:
- On the light of short-term effects, what could be the expected long-term effect of the sleep structure modifications?

- Existence of groups at risk and possible potentialization between noise exposure and general sensitivity to environmental factors.

- Can we explain the differences between “laboratory” and “field” results?

Theme 3. ‘Cardiovascular effects, Hypertension’ - Dr Wolfgang Babisch

The background paper for this theme will include a review of the relevant epidemiological literature on the relationship between transportation noise and cardiovascular endpoints, including the prevalence or incidence of angina pectoris, ECG ischaemia, myocardial infarction and hypertension. Particular emphasis will be given to dose-effect relationships, effect modifiers, noise metrics, gender differences and day/night differences.
A distinction will be made between objective exposure (sound level) and subjective exposure (annoyance..

Issues of evidence will be considered in the background paper using the criteria given in the WHO document “Evaluation and use of epidemiological evidence for environmental health risk assessment”, which was distributed. The noise studies will be discussed with respect to the possible bias. The background paper will refer to reviews that have already been published. Existing reviews will be updated according to new study results.

Since it was decided in the NNGL group meeting not (only) to rely on existing or new meta-analyses to derive dose-effect curves. An attempt will be made to suggest/select a few reliable studies that can be used as a reference for quantitative risk assessments.

The background paper will include results from epidemiological studies on mean blood pressure readings in children. The focus here is not on risk groups, which is covered by theme 9, but on the presence or absence of an association as such.

Theme 5.b. Cognitive impairment – Dr Staffan Hygge

The author will summarize what is reported on children and adults and a number of cognitive functions and will organize the material in the following way:

	Day time noise	Night time noise
Children		
Adults		

Cognitive measures that probably will have a heading of their own

Psychomotor performance
Attention – primary memory
Semantic memory – reading – language development
Episodic memory
Problem solving

Motivation
Accidents

If enough material is available **vulnerable groups** such as children – elderly, the sick and pregnant women.

In a parallel way the author will look at how **sleep deprivation** as an independent variable affects cognitive functions

Theme 5.d. Night Noise and Mental Health

Stephen Stansfeld & Michael Stalski

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Introduction

Transportation Noise and Mental health

Road Traffic Noise

- Acute effects
- Chronic effects

Aircraft Noise

- Acute effects
- Chronic effects

Neighbourhood noise and mental health

Mechanisms for causal links between noise and mental health

Habituation to noise and mental health

Risk groups for mental health effects of noise

- Population groups at risk for mental health effects from noise
- Noise Sensitivity

Sleep disturbance and mental health

- (To be completed by Michael Stalski)

Conclusion

Theme 5.e. Sleep pathologies - Professor Sona Nevsimalova

Subjective criteria of sleep pathologies:

sleep log
sleepiness scales (Epworth or another type)

Objective criteria of sleep pathologies:

1. Screening method – Actigraphy

At least 7 days of monitoring is necessary

2. Gold nocturnal standard – Nocturnal Polysomnography

Parameters to be scored in Nocturnal Polysomnography:

Time in bed
Total sleep time
Sleep latency (NREM sleep and REM sleep)
Sleep efficacy
Sleep architecture (% 1NREM, 2NREM, 3NREM,4NREM and REM sleep)
Cycles NREM-REM (No and continuity)
Stage shifts (Nos)
Pathological events: periodic leg movements (PLMI)
apnea and/or hypopnea (AHI, saturation)
others

3. Excessive daytime sleepiness

MSLT (multiple sleep latency test) and/or
MWT (multiple wakefulness test)

4. Computerized neuropsychological tests – e.g. Vigil

attention is examined:
reaction time and
number of incorrect reactions

Annex 3

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Annex 4: Report on the second meeting on night noise guidelines

Report on the second meeting on night noise guidelines

Geneva, Switzerland, 6- 7 December 2004

European Centre for Environment and Health
(Bonn Office)
World Health Organization Regional Office for Europe

ABSTRACT

This meeting was the second of the night noise guidelines project. Technical discussions have taken place on central issues like exposure assessment, metrics, health effects, guideline set-up.

The partners have presented the first drafts papers for the different identified themes and detailed discussion took place for each one of them. The discussions concentrated mainly on central issues like exposure assessment and guideline derivation. The experts recommend the use of DALY, which is an internationally agreed metric, to assess the severity of health impact and to establish guidelines.

The next meeting is planned for April 2005.

Keywords

NIGHT NOISE
GUIDELINES
ENVIRONMENTAL HEALTH
EUROPEAN UNION
SLEEP

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Background

The WHO - European Centre for Environment and Health - European Centre for Environment and Health, Bonn Office, is coordinating a project aiming at establishing WHO night time noise guidelines (NNGL). 18 partners are associated to the technical part of the work.

WHO convened the first project meeting in Bonn on the 7th and 8th of June 2004. The project partners, experts and national government officers met to define the work plan and discuss the organizational issues. They also allocated responsibilities, looked at the timetable, team coordination, the logistical and finance. It was agreed that the next step would be the production of background papers for the different identified themes.

On the 6th and 7th of December the project's second meeting was held in Geneva. This meeting was supported by the **Swiss Agency for the Environment, Forests and Landscape (SAEFL).**

Introduction

Dr Kerstin Leitner, the WHO Assistant Director-General for Sustainable Development and Healthy Environments opened the meeting. 22 experts and WHO staff attended the meeting (see participants list in Annex 1). Mr. Xavier Bonnefoy (WHO) acted as moderator.

Dr Henk Miedema, Dr. Stylianos Kephalopoulos and Professor Joao Levy have sent their apologies for not being able to attend the meeting due to different constraints. Prof Kahn was remembered and praised by the group.

The technical aspects for deriving guidelines were reviewed in detail. The partners presented the first drafts or their substantiating documents for the different subjects and detailed discussions took place for each one of them.

The discussions concentrated mainly on central issues like exposure assessment and guideline derivation. The experts recommended the use of DALY, which is an internationally agreed metric, to assess the severity of health impact and to establish guidelines.

Summary of the meeting

Statement of Dr Leitner

Dr Kerstin Leitner welcomed the participants and recalled the importance of noise as a major determinant of health, and of the WHO role on protecting human health from environmental factors. WHO has the production of guidelines as a major task and Dr Leitner has invited the group to develop standards and guidelines that can be applied to all WHO Member States (for all regions). She acknowledged that this is a European group but harmonization work should be planned to make the guidelines applicable for all member states.

Member States are requesting advice form WHO, and other international bodies when developing legislation regarding transport and other activities. This group should provide what is,

from a health point a view, the levels permissible and the way to achieve this levels. The guidelines should have according to her, a strong section on noise management and mitigation.

Dr Leitner suggested that the group starts by examining the scientific evidence on thresholds, then establishing the maximum values for each source associating, always when possible, a mitigation technique to reach those values. The guidelines document will be much stronger if it contains recommendations on how to achieve the proposed levels. As examples of success stories of noise reduction she pointed the story of the city of Athens where car horns were banned reducing noise considerably, and she also descried the procedure called “the continuous descent approach” for aircrafts for reducing the noise when landing.

Dr Leitner also recommended a very comprehensive peer review by colleagues from other regions asking them to feedback and input on the document.

Presentations by the project partners

The first partners’ project meeting assigned topics to various teams and asked them to provide a first version of their background paper.

After a small introduction the floor was given to the project partners for the presentation of their technical papers.

The next chapter summarizes the presentations’ main content and the discussions that followed. The full papers will not be annexed to this report, since this was an intermediate project’s meeting and the papers are not yet finalized.

Professor Peter Lercher - Noise management philosophies

For managing noise problems, two different types of noise management approaches are possible: specific acoustical approaches and integrated approaches. The first type includes dose-effect relationships approaches, event based approaches (oriented towards avoiding or stopping the noise sources) and Psychoacoustic approaches. The integrated approaches includes acoustic ecology, soundscape approaches and multi-sectoral environmental health impact assessment approaches.

Establishing guidelines for noise is more complex than for air and water because for these two pollutants maximum concentrations can be defined according to toxicology models. Noise levels on the contrary, can not be established exclusively according to biological effects on human because perception plays an important role (the response changes according to individual sensitivity, source associated perception, among others).

Professor Lercher’s presentation then concentrated on different aspects of noise measurement. There are significant differences between seasons on the same measurement site, and also the annoyance response to the same level of noise depends on the site (depending maybe of geography e.g mountain areas versus flat).

The situation regarding day and night time noise exposure distribution is also changing. The tendency is more and more for not having differences between day and night levels (the exposure during the day has stabilized but it has increased during the night. The increase of

goods transport by railway at night has been one of the main causes of this shift and probably the annoyance attributed to this source has also changed.

On the metrics side, according to the Alpine Valley study, geography and distance to the road correlates very well with sleep disturbance.

Professor Jacques Beaumont – Noise metrics and sleep – a proposal of indicators

Two indicators for noise exposure assessment were proposed (effective perceived noise level (EPNL) and transit exposure level (TEL)) and two case studies for aircraft and tramway noise presented.

EPNL is to be used for aircraft noise. This index considers the background level and the peak levels being expressed in Perceived Noise decibel (PNdB). TEL is to be used for railway noise and considers the speed and length of the train.

In the case studies presented these 2 indicators performed better than Lnight (equivalent sound level for the 8 hours of the night period) in cases of the existence of a high number of noise events. For few events (less than 30 times), Lnight varies more slowly than TEL or EPNL. For large number of events, all the indicators vary in the same order of magnitude. However all two new indexes are rather complex to compute.

Discussion

These two presentations were then discussed. The participants reacted strongly to the second presentation, especially to the proposal of new metrics. It was pointed that existing indicators correlate very well so there is no need to look for and add new indicators. The possibility of including a “distance to the road” parameter was discussed because it correlates well with sleep disturbance.

The group agreed that the number of events is very important and will have to be included, especially for describing awakenings or arousals, but was very much uncertain of the advantages of introducing new indicators.

Professor Alain Muzet - Short-term effects of noise on sleep with specific attention to mechanisms and possible health impact

With his presentation Prof Muzet gave a brief overview of the mechanisms through which noise immediately affects sleep. Noise may cause the following short-term effects on the sleep structure: delay of sleep onset, awakenings, sleep stage changes, electroencephalographic arousals, global modifications in temporal organisation of sleep stages and the total time spent in different sleep stages, and premature final awakening. In addition to these effects, other short-term effects caused by noise are modifications in the autonomic functions (heart rate, blood pressure, vasoconstriction and respiratory rate) and body movements.

It can be considered that chronic short-term effects can provoke more serious and long-term effects on health when they don’t disappear with the repetition of the stimulus over a long time exposure e.g. there is no habituation when of frequent exposure.

Body motility changes with noise stimulus at a relatively low level, while behavioural awakening (the strongest instantaneous interference of noise with sleep) has the highest threshold for occurrence of an instantaneous effect considered. In Passchier-Vermeer et al study increased probability of instantaneous motility was found for events with a maximum sound level (Lmax) of 32 dB(A), while behavioural awakening was found for events with Lmax of 42 dB(A). This study also shown that also sleep latency and average motility during the sleep period increased monotonously as a function of the noise exposure level.

The second part of the presentation focused on possible risk groups to noise. This can be people reporting sensitiveness to noise, shift-workers and night-workers. People sensitive to noise normally complain and protest more against noise.

Several authors have studied individuals reporting noise sensitivity as a factor to evaluate highly sensitive and non sensitive groups and to compare their reactions to noise exposure during daytime and night-time. In the laboratory studies self declared highly sensitive individuals had higher cardiovascular response rates to noise than non sensitive while they were awake, nevertheless there was no difference in sensitivity to noise between the two groups while they were asleep.

The sleep of shift workers is often disturbed by combined influences of ambient factors (noise is one of them) and chronobiological factors (e.g. sleeping at an unusual time of the day), but noise was considered as the first cause of sleep interruptions in a shift worker female group study.

Discussion

The cardiovascular function is a basic reflex function and that is why it does not habituate to noise. Elderly and children are more reactive. During the night it is “observed” more because during the day the information processing through the cortex is different and the individuals can “ignore” the signal. The response also depends on the sleep stage in which the person is.

Professor Sona Nevismalova - Long term noise-induced health risk mediated particularly by sleep

When a person has his/her sleep disturbed by noise the following effects are observed:

- reduction of sleep efficiency,
- increased number of arousals,
- increased stage shifts,
- increased movement time,
- increase of REM sleep,
- decrease of slow wave sleep (NREM 3 + 4),
- and a decrease of total sleep time

These effects, when chronic, can have persistent and permanent effects on mental and physical health of exposed people.

We can observe the first changes on sleep at noise levels of 45 dB(A) Leq. When this level is observed a decrease of REM sleep stages and a decrease of slow wave sleep (NREM 3+4) occur. These changes are more pronounced at 60 dB(A), where a big decreases of REM sleep is observed and we have superficial synchronous sleep (NREM 2) dominating. Several studies demonstrated that intermittent noise has a more disturbing effect on sleep than continuous.

Several environmental stressors including noise can cause insomnia. Insomnia is commonly defined as a difficulty in initiating and/or maintaining sleep. According to a research group of the

U.S. National Center for Sleep Disorders Research (1999), “insomnia is an experience of inadequate or poor quality sleep characterized by one or more of the following: difficulty falling asleep, difficulty maintaining sleep, waking up to early in the morning, non-refreshing sleep“. Insomnia also involves daytime consequences, such as “tiredness, lack of energy, difficulty concentrating, and irritability“.

It is estimated that around 10% of the population have chronic insomnia, when considering insomnia of any duration or severity this number increases to between 30% and 50% of the population. Developing insomnia increases with aging and women are particularly vulnerable to develop insomnia in the course of perimenopausal time.

Risk groups for having their sleep disturbed by noise were indicated by professor Nevismalova, similarly to the previous speakers:

1. sensitive subjects (anxious and with neurotic tendencies);
2. children (because the growth hormone is segregated during SWS sleep and the REM sleep is crucial for memory);
3. women during pregnancy and perimenopausal period
4. shift workers
5. elderly people (their sleep is more superficial)
6. patients at intensive care units,
7. low-birth weight infant units,
8. and residents and disabled persons in nursing homes.

Besides healthy population, standards should be recommended and strictly adhered to in hospitals, particularly at intensive care units.

Dr Leja Dolenc Grošelj - Physical effects resulting from night time noise exposure

Dr Groselj’s presentation covered the “next day effects” after having a disturbed sleep by night time noise exposure. There is solid evidence that noise interferes with sleep, awakenings being the most common consequence. Noise events interrupt the sleeping process for a period of time going from a few seconds to several minutes. The repetition of these sleep modifications throughout the night may lead to chronic sleep loss with consequences such as chronic fatigue, sleepiness during daytime, and a global poor life quality.

Extensive studies dealing with sleep disturbances caused by noise exposure have been published during the last ten years. Most of them are dealing with sleep fragmentation and its consequences, insomnia being the most serious consequence. However, fatigue, drowsiness and excessive daytime sleepiness were significantly less studied. Most of the studies dealing with daytime consequences were subjective. Objective studies with laboratory measurements are lacking.

Discussion

It is extremely difficult to measure fatigue and sleepiness. The only way is by asking people. There is no harmonized way of measuring it. Sleep recordings can be performed but they are rather difficult and expensive. One possible solution would be to adapt the results for insomnia; this is probably the only way to have health data on this subject.

Professor Stansfeld; Dr Michal Skalski - Night noise and mental health

Professor Stansfeld described the associations between noise (transport, neighbours and occupational) and mental health. Limited data exists for night noise, also due to the fact that occupational noise findings can not be used. It is generally accepted that noise exposure at night is more disturbing than daytime noise because it interferes with rest and sleep. Because there is little direct research on night time noise and mental health the evidence for environmental noise and mental health in general was analysed first.

Results of several studies were presented. In these studies the association between noise and mental health has been examined using a variety of outcomes including individual symptoms (at the simplest level), psychiatric hospital admission rates, use of health services, psychotropic medication, and community surveys. Current evidence does seem to suggest that environmental noise exposure, especially at higher levels, is related to mental health symptoms and possibly raised anxiety and consumption of sedative medication, but there is little evidence that there are more serious effects.

Further research is needed on mental health effects originated from exposure to very high noise levels. Existing studies may be confounded either by prior selection of subjects out of noisy areas or by confounding noise exposure, socio-economic deprivation, and psychiatric disorder. It is also possible that people underestimate or minimise the effects of noise on health through "optimism bias" this is a particularly protective factor for mental health. If a person copes well with the noise it is less likely that it has a harmful effect.

Depression is heavily associated with insomnia, but not much is known about the mechanisms linking insomnia to depression. Anxiety and insomnia have similar causes and it is very difficult to distinguish which triggers which.

Population, as well as clinic-based studies, have demonstrated a high rate of psychiatric morbidity in patients with chronic insomnia. It has traditionally been assumed that insomnia is secondary to the psychiatric disorders; however it is possible that in some cases the insomnia preceded the psychiatric disorder.

Discussion

Noise as a stressor affects health in a different way from the other stressors. Noise is a classical stressor involved in the information processing but during daytime, the brain can "ignore" it and a positive reaction towards it will reduce its effects (e.g. when associated with fear, negative attitude regarding the source the effects are much higher).

People that have already mental problems, likewise paranoia, can be extremely affected by noise (all noises are seen as threats). More research is needed to identify night time exposure to noise effects on mental health.

Dr Christian Maschke – Stress mechanisms

Noise triggers a stress reaction, the crucial question is whether noise can actually be regarded as a stressor that induces health damages, mainly in the pathogenesis of cardio-vascular diseases.

Stress is a non-specific reaction of a living organism, which can be triggered by any exogenic or endogenous (including cognitive and emotional) induced stimulation involved in the information processing of the central nervous system. It can cause temporary or permanent functional or structural changes in all body systems. Eustress (positive stress) promotes performance and health; distress (negative stress) is a variety of stress with a pathological appearance.

There is biological plausibility of noise being a psycho social stressor activating the sympathetic and endocrine systems and there is sufficient evidence that noise levels higher than 50 dB (A) disrupt strongly the hormone secretion cycles.

The non-specific reaction by stress is strongly modified by:

- the individual functional condition (activation is not only dependent on the volume of the acoustic stimulus, but is also determined for example by vegetative excitation levels and subjective perceptions of influence)
- habituation – dishabituation (habituation to noise) is only to be expected in cases of acute stimulation or low intensity)
- conditioning of the non-specific reaction (in terms of noise, negative emotions essentially triggered by conditioning include anxiety, fear, helplessness and defencelessness)

Hormones are secreted according to a rhythm. The time a person takes to recover from a stressed situation depends very much upon individual characteristics and of the emotions they experience.

Persons that express hostility have a higher risk of cardio vascular diseases, this is also true to noise. The more a person is hostile to it, the higher is the risk of cardiovascular dysregulation. No-acoustical factors play a big role in this process.

The chronobiological stress caused by night noise occurs during the most important period for recovering of a human-being! It is extremely important to preserve this period to maintain the balance necessary for good health.

Discussion

Hormones during sleep are secreted on a pulsatic way, following a similar pattern, e.g. the growth hormone secretion can be affected by a noise stimuli, however there is a gap on knowledge is on the effects of disturbing these patterns.

Dr Wolfgang Babisch - Health effects related to stress mechanisms - Cardiovascular effects

Dr Babish has carried a very comprehensive literature review. He has selected the studies according to the WHO document “Evaluation and use of epidemiological evidence for risk assessment”. Using all six criteria of the document for considering epidemiological studies, 5 studies emerged that refer to the association between road traffic noise and ischaemic heart disease. The presentation focused on these studies even though the other studies were also mentioned.

All the five studies were analysed and published with respect to clustered data (high vs. low exposed), which raises some concern regarding its application for the derivation of guidelines. One of these studies was considered in the earlier meta-analysis (the only one) for aircraft noise and hypertension. When dose-response studies on self reported prevalence of hypertension are

included, two more peer-reviewed studies appear on the list. These studies were carried out in Berlin (Germany) with respect to hypertension and Stockholm (Sweden) with respect to aircraft noise.

Nearly no information is available from epidemiological studies on the cardiovascular effects of long-term noise exposure, particularly, during the night-time or with respect to the exposure of the bedroom. Only one study suggested slightly higher effect estimates with respect to the exposure of the bedroom (during the night) compared with the exposure of the living room (during the day). However, this study has some methodological limitations that were also addressed in a recent report of the Dutch Health Council. A few studies regarding subjective responses to community noise suggest a closer relationship of cardiovascular diseases with sleep-related annoyance and disturbance reaction rather than with non-sleep related annoyance/disturbance. Closing the bedroom window or, vice versa, sleeping with the bedroom window open, was associated with a, respectively, lower or higher risk. The same was found with respect to swapping bedroom and living room because of noise. These findings may indicate that night-time noise may be more a determinant of noise-induced cardiovascular effects than the daytime exposure. However, during daytime time-activity patterns and expectations of the individuals are much more inhomogeneous than during the night, which dilutes possible effects.

Given the prevailing situation, there does not seem to be any other way of reasoning than inferring night noise recommendations or guidelines from the results of epidemiological studies that refer to the noise exposure during the daytime period (Lday) or the whole day (Ldn, L24h). Lden appears to be a useful noise indicator for formal decision-making and regulatory purposes, but not for noise effects' research. Such global noise indicators refer normally to the most exposed facade, which incorporates a certain degree of exposure misclassification regarding cause-effect relationships.

However, in urban settings, night-time average noise levels from road traffic tend to be approximately 7-10 dB(A) lower than daytime average noise levels. In such cases, Lden is approximately 2 to 3 dB(A) higher than Lday. Therefore, in epidemiological studies in which the relative effects of road traffic noise is studied, the sound emission during the daytime can as well be viewed as an approximate relative measure of the overall sound emission including the night. This seems to be further justified since existing noise regulations usually consider a 10 dB(A) difference between the day and the night, which is also incorporated in the construct of Lden. With respect to railway noise and aircraft noise no such stable relationship can be assumed. The day (night difference depends very much on the cargo traffic during the night and existing night-flight restrictions regarding airports. Assuming these circumstances epidemiological studies can be considered for the development of night noise guidelines that were not focussing explicitly on the exposure during the night. This approximation can, particularly, be made with respect to road traffic noise. 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.

The assessment of dose-effect relationships sometimes suggested a cut-off level, above which the risk tends to increase. From a biological point of view one would expect a continuous increase in risk with increasing noise level. However, adaptation, coping and habituation may be reasons for an empirical threshold of effect. Decisions with respect to guidelines values should

be based on a quantitative risk assessment – not ignoring the individual risks of exposed subjects, if considered as relevant.

Discussion

The expert has considered that sufficient evidence exists for noise exposure and ischemic heart disease even if there aren't a large number of studies. A significant increase is observed for 65 dB(A) during day time and 55 dB(A) for night time.

A new meta-analysis using all the mentioned studies can be performed, but the author would have to be able to consult the original studies (not only the summary and the published results).

Dr Svenaza Yovanovic – Children accidents

Apparently there is only one study on children (Udine hospital, Italy) and the recent LARES survey dealing with accidents and noise. Some data exist for falls, poison, ingestion of toys or other strange objects, heavy injuries and road traffic accidents but no linkage has been done with noisy conditions at the school or home.

One very interesting fact is, as shown by a Swedish study, that twenty five percent of school injuries (in children between 7 to 19 years old) are caused by other pupils. Some of this violence and aggressiveness can be related to noisy conditions.

A German study carried in Cologne, with 5000 children, has shown associations between bad sleep conditions and aggressiveness. Leger and al. have published a paper where it is described that 40% of car accidents and 30% of work accidents are sleep related, for domestic accidents the main risk factor is having a sleep disorder. Sleepiness is related to accidents but again, there is a lack of studies with covering the relation with noise.

Discussion

Knowledge exists for sleep reduction and risk of accidents but insomnia does not always lead to sleep reduction. Attention has to be paid to speculation around this theme, only clear and solid results should be used.

The results of occupational noise can probably be used, Prof Lercher has carried a meta-analysis for the ICBEN congress in 1998 and has found some data for noise at workplace and accidents.

An accidents risk assessment can also probably be made using insomnia as a model. When insomnia is caused by noise what is the risk of accidents? Can an extrapolation be made from the existing literature that noise at night causes x work accidents, y traffic accidents, z domestic accidents?

Dr Staffan Hygge - Night noise and cognitive impairment

Prof Hygge has performed a review on the effects of night noise exposure on cognitive performance. Searching PsychINFO for (noise AND night AND (memory OR learning OR attention)) resulted in nine hits, all related to sleep or irregular sleep patterns or night shift work. In contrast to this lack of reported research, searching Google with the same keywords and

operators yields 2.7 million hits. Subtracting all texts with a reference to sleep left a remainder of 1.8 million hits. This discrepancy in numbers between texts reporting research and other texts containing the same keywords may reflect a situation of high interest but little of hard facts.

There are links between night time exposure and reduced sleep quality and between reduced sleep quality and cognitive performance; however the links between night noise exposure and cognitive performance are missing. This is the ones we will have to concentrate on.

Several difficulties were encountered relating to:
- day and night time exposure
- children and adults differences
- restoration
- communication and noise

During the night we are not “learning” but there are important functions occurring in the brain like memory storing. It can also be the case that noise doesn’t have an impact on cognitive system, but it only lowers motivation and ambition that possibly lead to problems in school.

Discussion

Can the processing memory mechanism be described? And what part is affected by night time noise? It is known that Cortisol plays a crucial role for memory process, for instance if there is an increase of cortisol there is a lack of next day memory but we don’t know if the memory is affected through other mechanism. It is also known that it is not cortisol that affects memory, a disruption of its normal secretion rhythm shows a disturbed sleep. Cortisol is a stress hormone, people highly stressed during the day need a better sleep, and everything is linked.

Knowledge on noise and hormones is rather limited! Specialized literature exists for the “normality” and the role of the hormones on a good health. The guidelines should consider having a chapter on this and infer on the possible effects of noise. Like in other health determinants the group should use the precautionary principle and be based on the existing knowledge.

Professor Hartmut Ising - Animal Studies

Noise has often been used as a stressor in animal studies. Most of the modern animal studies testing the pharmacological effects of drugs are carried out with and without various stressors. The animal model for auditory effects in humans has been established in great detail, a quantitative transference of results from animals to humans is possible. However, inner ear damage generally occurs at much higher noise levels than the environmental levels usually met in daily life.

Extremely intensive unpredictable noise near the inner ear pain threshold triggers, in awake mammals, a defeat reaction with increases of ACTH/cortisol while the catecholamines adrenaline and noradrenaline remain normal or are slightly decreased.

Beside cardiovascular stress effects, chronically increased cortisol may induce neuronal degeneration and thus accelerate the ageing also of the brain not only in rats but in humans as well.

The importance of calcium magnesium shifts was confirmed by post mortem studies of hearts from victims of ischaemic heart diseases (IHD). The tissue samples were taken from areas of the myocardium not affected by the infarction and the results were stable after controlling for several confounders.

Long-term experiments with magnesium deficient and noise-stressed rats showed that connective tissue and calcium in the myocardium increased with age while Mg decreased. Hence, stress caused by noise or cold is enhanced by sub-optimal Mg intake and accelerates the ageing of the heart and decreases the life span.

Rhesus monkeys exposed to traffic noise for 10 h/day during 9 months (Lmax=97 dB, Leq=85 dB) developed significant blood pressure increases persisting 3 weeks after termination of exposure. A replication of this experiment with a different species of primates failed to show an increase of blood pressure.

Some important factors influencing health effects of chronic noise exposure:

- Unpredictability of noise events
- Genetic factors (inbreeding)
- Combination with other stressors

Animal studies shown that chronic noise-induced stress accelerates the ageing of the myocardium and increases the risk of myocardial infarction. The involved pathomechanisms include: increases of catecholamines and/or cortisol and an interaction between endocrine reactions and intracellular Ca/Mg shifts accelerating the ageing of heart and circulation.

Discussion

Magnesium deficiency is an effect of noise exposure but there are contradictory results. A person needs time of no-stress for reabsorb magnesium. Cortisol is a better indicator than noadrenaline. This patterns need to be well described.

Professor Oliviero Bruni - How to take into account the specific needs of children. Children's sleep

In the last years, a growing body of literature demonstrated the strict relationships between sleep disturbances and daytime dysfunctions. Results from recent quantitative research on noise as an environmental stressor consistently demonstrate that children are more vulnerable than adults to the adverse effects of noise exposure especially when considering the effects on cognitive performance, motivation and annoyance.

Data shows that children with sleep-related obstructive breathing disorders (SROBD) are less reflective, more impulsive and show poorer sustained and selective attention, memory impaired and showed reduced academic performance and learning. Early treatment showed that attention deficits in OSAS (Obstructive Sleep Apnea Syndrom) children are reversible. Actigraphic study in school children showed that lower sleep efficiency and longer sleep latency were associated with a higher percentage of incorrect responses in working memory tasks. Almost thirteen percent of children that have frequent and loud snoring during early childhood report low academic (versus five percent with high academic performance). Healthy children with fragmented sleep showed lower performance on neurobehavioral functioning (NBF) and had higher rates of behaviour problems.

Children and adolescents with disturbed sleep report more depression, anxiety, irritability, fearfulness, anger, emotional instability, inattention and conduct problems, and drug/alcohol use in adolescence. At 3, 4 years of age persistence or recurrence of infant sleep problems is common and associated with higher aggressive behaviour and somatic problems and maternal depression scores.

A study exists for the increased risk of accidents. In Italy for 292 injured children who presented at the Children's Emergency Center of Udine, there was a direct association between injury risk and sleeping less than 10 hours. Among 3 to 5 years old boys there was reported an 86% increase risk of injury.

Inadequate sleep duration and lack of daytime naps (in school) are transient exposures that may increase the risk of injury among children.

Although we should take into account that it is at present impossible to indicate whether children are possibly more sensitive than adults to other direct biological effects of night-time noise, we can hypothesize the effects of night-time noise during sleep in children based on inferences of adult studies.

It is known that adults' physiological responses appear at an Leq in the bedroom of approximately 40 dB(A) and behavioural awakening occurs when the bedroom *Lmax* exceeds 55 dB(A). Since child's autonomous nervous system is more readily activated by noise (acoustic stimuli of much lesser intensity 10 to 15 dB) when sleeping than an adult's, it can be assumed that children are more sensitive also to detrimental effects of noise exposure.

In the following table the effects of sleep disruption specific to children, based on literature evidence are described.

Effects of sleep disruption in children induced by noise

Short term	Behavioral	Daytime fatigue; decreased performance and concentration, memory difficulties; difficult behavior; increased motility
	Medical	increased heart rate; use of sleeping pills and sedatives
	Mortality	Increased risk (Sudden Infant Death syndrome) ?
Long term	Behavioral	Difficulty in modulating impulses and emotions; poor performance at school, fatigue, memory difficulties, concentration problems; impaired well-being and motivation: increased risk of accidents; increased motility
	Psychiatric	Depression, anxiety conditions; aggressive and delinquent behaviour; attention-deficit/hyperactivity disorder; alcohol, smoking, caffeine and other substance abuse (?)
	Medical	Increases in sleep disorders (parasomnia); changes in blood pressure; changes in carbohydrate metabolism; changes in immune system (?);use of sleeping pills and sedatives
	Mortality	Increased risk (Sudden Infant Death syndrome) (?)

During what can be considered a "normal" sleep in average a child should not have more than 7 arousals per hour.

In order to quantify or to specify limits for sleep disturbances induced by noise in children the following table can be established:

	Preschool	Sleeping in School
Night noise limit	*Leq* <30 dB(A)	*Leq* <30 dB(A)
Arousal n°/h	< 7	< 9
Cyclic AP rate	< 30%	< 35%
N° awakenings	> 2 lasting > 15’	> 2 lasting > 15’

Children should be considered as a risk group, specially the ones that have a low birth weight, are small for gestational age babies, have had preterm birth, are already having learning disorders behavioural disturbances (i.e. hyperactivity) and already have sleep disorders.

Discussion

This presentation raised a large discussion around the proposed guideline values. There was a problem with the metric used (SEL and Leq) that is already corrected in this report. The proposal of the CAP rate and number of arousals was questioned but Professor Bruni shown the results of the studies where he has based his results.

Professor Bruni was asked on the number of arousals and Cyclic Alternating Pattern (CAP) and its relationships with noise. How can this be measured?
During NREM, sleep maintains an oscillating pattern that reflects different levels of arousal that has been coded as CAP. CAP is a periodic EEG activity of NREM sleep characterized by repeated spontaneous sequences of transient events (phase A) which clearly breaks away from the background rhythm of the ongoing sleep stage, with an abrupt frequency/amplitude variation, recurring at intervals up to 1 min long. The return to background activity identifies the interval that separates the repetitive elements (phase B).
Simplifying the CAT rate can be an indicator of sleep quality. The potential indicators of sleep disruption in children are mainly represented by arousals and cyclic alternating pattern (CAP).

Literature shows that reactions smaller than a sleep stage change correlate better to the noise intensity than awakening reactions. Arousal analysis, CAP analysis and identification of new parameters of sleep fragmentation (sleep pressure score) in children can give new insights on the relationships between sleep disruption and cognitive consequences.

Lnight can not be the only descriptor of a good sleep, the number of arousals, the CAP rates and awakenings will also have to be defined. The guidelines will have to consider descriptors of good sleep.

The remaining question that was rose is how to calculate these parameters, what should be the relevant metric to put into legislation!

Dr Mueller – Wenk - Determining the severity of noise-related human health effects within an established health metric system

Amongst noise-related health effects, small impairments affecting large fractions of a population may be more important than a small number of noise-related severe effects, even deaths. To obtain a comprehensive picture of noise-related health damages, it is advisable to link dose-effect-relations to the severity weights of the respective health impairments.

The DALY concept, as widely used by WHO, is an advantageous health metric system for assessing noise-related impairments. Until now, health endpoints of available dose-effect relations are not enough precise for an attribution of severity by medical experts.

Noise-related sleep disturbance is clearly a disability (and not merely an annoyance), and its disability weight was rated by medical experts roughly equal to 'chronic Hepatitis B infection without active viral replication'. A second study with Obstructive Sleep Apnoea Syndrome (OSAS) confirmed that this rating is conservative. Sleep disturbance is the dominant component of the road-noise-related part of the Swiss burden of disease.

In order to strengthen the scientific support for more effective noise abatement, Dr Wenk recommends the group to concentrate on the following activities:
- make a provisional ranking of the importance of the health effects to be considered in the perspective of a person's burden of disease.
- agree on properly defining the health endpoints of dose-effect-relations, so that it is possible to attribute a severity weight to these health endpoints, preferentially within the rules of the DALY system;
- agree on the question of positioning disabilities as hypertension, increased blood lipids, interference with speech communication, annoyance.
- decide on the most important and necessary dose-effect relations, and proceeds to a collection of available preliminary data for these.
- agreeing on a list of missing disability weights covering the noise-related health effects.

Discussion

The group agreed with using the DALY methodology because it is a common international health metrics and will allow a common language for the major health determinants. In addition it is of interest for the decision makers when having to make decisions regarding different pollutants.

Dr Martin Van den Berg – deriving guideline values for night noise

Dr Van den Berg has based his analysis on three documents, “Evaluation and use of epidemiological evidence risk assessment (WHO publication)”, the “Air quality guidelines for Europe (WHO, 2nd edition)” and the recent report from the Health council of the Netherlands “Influence of Night time noise on sleep and health”.

A general approach for deriving guidelines consists in undertaking several steps:
1. Assessment of the impact on the population;
2. Evaluating the impact;
3. Assessing options to avoid or reduce impact considered undesirable;
4. Cost-benefit analysis of the options or of mix of options;

5. Assessment of the preferred option;
6. Implementation.

As steps on Health Impact assessment (following the WHO document evaluation and use of epidemiological evident), the data has to be collected and validated, the metrics of exposure have to be specified and the health outcomes defined. Then the methods for estimating the exposure response relationships and the baseline frequency of health outcomes specified for assessing the attributable cases to noise.

The criteria for evaluating studies could be by separating the biological and health effects, then evaluating the significance of health effect, establishing the exposure-effect process based on the strength of the evidence for each relationship .

The health council of Netherlands reports that noise affects the quality of sleep, general well – being, social contacts and concentration and medical conditions reducing consequently life expectancy.

Sleep quality	Reduced perceived sleep quality	Sufficient evidence
	Difficulty getting to sleep, difficulty staying asleep	Sufficient evidence
	Sleep fragmentation. Reduced sleep time	Sufficient evidence
	Increased average motility when sleeping	Sufficient evidence
Well being	Sleep disturbance	Sufficient evidence
	Health problems	Sufficient evidence
	Use of sleeping drugs and sedatives	Sufficient evidence
	Increased daytime irritability	Limited evidence, plausible
	Impaired social contacts	Limited evidence, plausible
	Impaired cognitive performance	Limited evidence, plausible
Medical	Insomnia	Sufficient evidence
	Hypertension	Limited, indirect evidence, plausible
	Depression (in women)	Limited, indirect evidence, plausible
Premature mortality	Cardiovascular disease	Limited, indirect evidence, plausible
	Occupational accidents	Limited, indirect evidence, plausible

Effects on health and well being of prolonged exposure to noise during the sleep period (the health council of the Netherlands, 2004)

Deriving guidelines (taking the example of the air quality) is always a complex process. There is always lacks on knowledge and it is very difficult to have all the evidence with full confidence. Quoting the air quality guidelines: “Ideally, guideline values should represent concentrations (…) that would not pose any hazard to human population” - “Although a certain risk can be tolerated” - “The use of a standard approach is impossible”.

There are different approaches for establishing guidelines for night time noise, a first possibility is to write a document with the state of the art without providing guideline values, another possibility into provide DALY-weighting for all end points and the last avenue could be to give the same protection as other existing and solid guideline values (e.g air quality – Lnight of 40 dB same protection as NO2= 40 ug/m3 (WHO air quality guidelines)).

For controlling noise during the night a restriction of night time activities can be envisaged, or the use of only quiet equipment during night time. Another type of measures is the banning of noisy

activities from sensitive areas (e.g. residential areas) or the other was around taking sensitive activities from noisy places or insulating buildings. All these measures are very expensive and not easily put in practise.

It could be concluded that firstly all the information and evidence have to be collected, that, based on solid facts the group has to decide if and what guideline values to use. The evidence should be systematically assessed for validity and strength.

Discussion

Guidelines values will be established, if the group produce only recommendations nothing will change on the noise field. The guidelines should have a protective role and will be based on the best available evidence. Risk assessment methods and DALY approach could be used when there are minor gaps in the knowledge.

Mr Colin Grimwood - Neighbourhood Noise - guidelines

Mr Grimwood's presentation concentrated on how to consider neighbourhood noise in the night noise guidelines. Should guideline values be defined or just recommendations and good practice advice be provided?

Deriving guideline values for neighbourhood noise is a very difficult, not to say impossible, task. First a clear definition for neighbourhood noise has to be agreed upon. The existing definitions are:

- Transportation noise - noise from the normal operation of a mechanised mode of transport, primarily road vehicles, rail vehicles and aircraft.
- Neighbour Noise - noise from people, pets and the use of domestic appliances in and around residential premises.
- Neighbourhood Noise - all other noise sources produced in the neighbourhood!

Mr Grimwood had not undertaken a literature review but he can confidently state that there are less studies on night-time neighbourhood noise than transport noise, and only those made in last few years are likely to be suitable for intercomparisons.

The second major question raised was whether a night-time neighbourhood noise guideline would be an appropriate approach for all neighbourhood noise sources. How would a guideline value be enforced? Additionally some countries (e.g. Switzerland) have different institutions dealing with the different noise sources. Environmental, occupational and neighbourhood noise are under the responsibility of different governmental agencies and levels (neighbourhood noise is usually dealt at local level).

The approach for regulating neighbourhood noise has to be a holistic approach. Firstly a noise impact assessment has to be made, considering the absolute level of noise and any possible noise changes. In addition the non acoustic factors need to be taken into account (time-of-day, season of year, type of area, attitudes to source, neuroticism, negative affectivity, individual sensitivity to related pollutants, expectation, history of noise exposure, employment or other connection with the noise source, education, home ownership, type of dwelling, length of residence, etc). The context is very important and factors like behaviour and other social factors need to be considered as well as other factors such as acoustical insulation.

The way different countries deal with neighbourhood noise is also different. For example, in England the generalised findings on dose/response, sleep disturbance, effects of change, role of non acoustic factors applicable to the different types of neighbourhood noise are not used in primary legislation for any neighbourhood noise source. Instead the law allows environmental health practitioners and the courts to take such factors into account when assessing the likelihood of noise nuisance. Nevertheless, there are a few examples of guideline values in use in England for night time neighbourhood noise (mixed sources, outdoor concerts, industrial noise, night time neighbourhood noise).

An issue that needs to be explored through further research is if there are unique factors (acoustic and non-acoustic) that are applicable to the different categories of neighbourhood noise.

Examples of existing guideline values for some sources were presented for England.

Industrial Noise

- Assessing likelihood of complaints from industrial noise
- Uses a 'relative guideline'
- Noise is measured or calculated (LAeq, T), corrections applied (impulsiveness, tonality, irregularity, time of day etc.) and compared with background noise level (LA90,T)
- Applies to day and night (but BS4142 method further weights night noise by use of shorter reference T period)
- Note 'night' is decided locally but 'is intended to cover the times when the general adult population are preparing for sleep or are actually sleeping'.

Noise Act 1996 "Neighbourhood Noise"

- Specific for night (defined as 2300 - 0700)
- Uses a 'relative guideline'
- Where the underlying level of noise does not exceed 25dB, the permitted level shall be 35dB
- Where the underlying level of noise exceeds 25dB, the permitted level shall be 10dB in excess of the underlying level
- noise source measured as LAeq, 5min
- underlying level - level not exceeded for 0.6s in 5mins (e.g. LA99.8, 5min or LA99, 1min)

One very interesting fact is, based on two national noise surveys undertaken in 1990 and 2000 and involving over 1,000 measurements each, that environmental noise during the night is increasing whilst noise during the daytime is very slightly decreasing. If this trend continues then it would result in the same levels for night and day (currently there is a marked decrease at 00:00 but the noise just goes up again around 04:00 until reaching a peak at 08:00).

Discussion

The noise of neighbours is increasing, it is one main cause of complaints and dispute among communities, establishing guidelines will not provide a solution to all problems. There are a lot of non acoustical factors; the situation has to be seen case by case. Listing the noisy activities that should be banned at night (vacuum cleaning, having the television or the stereo at a high sound,…) and giving good practice recommendations is one possible approach.

Conclusions

1. In many countries there are indications that noise during the night period is increasing practically to the same level as during day (examples of Austria and England). The night period when the noise levels are low is becoming very short (3 to 4 hours).

2. When there are significant risks of damage to the public health, WHO should be prepared to provide guidance in order to allow authorities to take action to diminish those risks, even when the scientific knowledge is not conclusive.

3. The existing WHO guidelines are always based in experts best judgements. The consensus and advice of this group is one of the crucial elements of the final document.

4. Annoyance should be seen as a short term response to noise exposure, long term annoyance leads to ill health (higher susceptibility to disease).

5. Some short-term health effects when occurring on a long period of time may become chronic. In other words, there is no habituation, even after a long exposure time.

6. It seems plausible that night time noise has an effect on mental health. The links need to be further evaluated.

7. Noise sensitivity can be related to anxiety; anxious persons are more sensitive and have less capacity to adapt to a situation. A person already suffering from mental health problems (as anxiety, depression or paranoia) will be more vulnerable to noise than a healthy one.

8. Night time intermittent noise has worse health effects on sleep than continuous noise covering the same energy over the exposure period.

9. Guidelines should be recommended for hospitals, particularly at intensive care units.

10. Fatigue, drowsiness and excessive daytime sleepiness need to be more studied. As well the way and are associated with accidents (identification of a causal link).

11. More studies looking into associations, and if possible links, between children's accidents, aggressiveness, and hostility and noise should be carried.

12. WHO should organize a new meeting of experts in chronic insomnia covering in detail its causes and health effects.

13. Cortisol excretion during the night and its effects should be better evaluated. A review covering the knowledge and the existing evidence of cortisol excretion should be included on the guidelines.

14. The internationally agreed WHO metric DALY, should be used to assess the severity of health impact and to support guidelines each time possible.

15. when studies concentrating on the night period are missing, extrapolations from exposure during daytime can be made.

16. In case of gaps in knowledge guideline values can be established in order to give the same protection as existing guideline values in other environmental fields (water, air,…). The evidence should be systematically assessed for validity and strength.

17. Population more at risk to night noise are:
    Children:
        Low birthweight
        Small for gestational age babies
        Preterm birth
        Dyslexia and hyperactivity

    Adults:
        sensitive subjects (anxious and with neurotic tendencies);
        individuals with pre-conditioned lighter sleep (women during pregnancy and perimenopausal period, elderly people and shift workers);
        patients in intensive care units;
        residents and disabled persons in nursing homes.

Main gaps in Knowledge at this stage

1. The role of stress hormones on the autonomic and cognitive functions need to be better evaluated, especially excretion of Cortisol during the night.

2. Causes and health effects of insomnia should be evaluated and described in detail.

Guideline development – a possible way forward

The guideline development will be fundamented on establishing values that prevent the occurrence of harmful health effects. At this stage the plausible health effects, either direct or indirect, of exposure to noise during the night can be listed. For some effects solid evidence exists and noise thresholds values can be defined, for others a protective threshold can be established. For the effects for which solid evidence is missing an approximate approach based on day time findings, supported by animal studies, and based on the precautionary principle, if needed, will be used.

For children the guidelines values will the ones ensuring a healthy sleep. For the time being (based on Professor Bruni's paper) this can be the lower level of noise for which studies demonstrate a clear and significant disturbance of the sleep of the children. A more in depth discussion needs to be carried, especially on the health impacts on children of a disturbed sleep on a long term basis.

For adults the effects to be considered are sleep disturbance and its associated effects, cardiovascular effects, the possible mental health effects, especially among the population already suffering from a poor mental health.

The metric to be adopted should consider the average background noise level that does not provoke adverse effects and will have to allow evaluating the number of on arousals and awakenings.

Follow up work

In the plenary session on the last day of the meeting, the group established and agreed to have the third meeting on the 26th, 27th of April 2005 in Portugal. The table of contents (annex 1) and the authors of each chapter have been updated. The partners will review and finalize their papers according to the comments made during the present meeting and the final version will be discussed on the next meeting.

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Professor Beaumont; Dr Kephalopoulos; Professor Lercher; Professor Levy; Dr Licitra

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Dr. Leja Dolenc Groselj

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Dr Maschke

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Dr Miedema, Prof Muzet

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Professor Nevismalova

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Annex 5: Report on the third meeting on night noise guidelines

Report on the third meeting on night noise guidelines

Lisbon Portugal, 26th - 28th April 2005

European Centre for Environment and Health
(Bonn Office)
World Health Organization Regional Office for Europe

ABSTRACT

On the 26th, 27th and 28thof April 2005 the Night Noise Guidelines project's third meeting was held in Lisbon. This meeting reviewed the final version of the background papers and established the future work.

Keywords
NIGHT NOISE
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Background

The WHO European Centre for Environment and Health - Bonn Office and 17 partners started in June 2004 the implementation of a project, co-financed by the European Commission aiming at deriving night time noise guidelines (NNGL). For more information on the project please consult http://www.euro.who.int/Noise/activities/20040721_1.

WHO convened the first project meeting in Bonn on the 7th and 8th of June 2004. The project partners, experts and national government officers met to define the work plan and discuss the organizational issues. They also allocated responsibilities, looked at the timetable, team coordination, the logistical aspects and finance.

On the 6th and 7th of December the project's second meeting was held in Geneva with the support of the Swiss Agency for the Environment, Forests and Landscape (SAEFL). Technical discussions have taken place on central issues like exposure assessment, metrics, health effects, guideline set-up. The partners have presented the first drafts of their papers for the different themes and detailed discussion took place for each one of them.

On the 26th, 27th and 28th of April 2005 the **project's third meeting** was held in Lisbon in the premises of the Portuguese General Directorate of Health (DGS). This meeting reviewed and discussed the final version of the background papers and the way forward.

In addition a discussion took place during this meeting on the "Aircraft noise and health" document that WHO is producing. The group of experts were consulted on the document' scientific background, if it does a correct interpretation of the science, on the rationale leading to the recommendations and on the clarity of presentation for non-scientific audience. The minutes of this discussion are on annex 1.

Summary of the meeting

Dr Francisco Jorge, Deputy Director General of DGS opened the meeting. He welcomed the participants to the Directorate and Lisbon and made votes for a successful meeting.

Mr Xavier Bonnefoy made the meeting introduction by summarizing the project development and presenting what is planned for the future. The project is planned to end in June 2006. At this stage the exposure and metrics are the weak parts, group one has send a paper but it is rather incomplete.

Presentations

Mr Van den Berg – methodological aspects

Methodology aspects for deriving the guideline values was the topic of this presentation. Mr Van den Berg updated and completed the presentation he had already made to the group during the second meeting (in Geneva).

The following procedure can be envisaged to derive the guideline values:
1. Collection of relevant literature

2. Evaluation of data in terms of strength of evidence
3. Evaluation of data in terms of biological effects, health & wellbeing
4. Ranking of guideline values

This procedure is already used in other guideline documents, but for noise the steps have to be more explicitly formalised because sound is an essential environmental aspect, which makes the use of a no-effect level (NOEL) not useful. The use of No Observed Adverse Effect Level (NOAEL) would be more appropriate.

A case study of the night regulations in the Amsterdam airport was presented. In 1994 a legal requirement of LAeq, 7 hours, inside of 26 dB(A) (approximately Lnight 49 dB(A)) was established. In addition, in 1995 the airport development authorities had to create the necessary conditions for not having more than 39 000 people with their sleep disturbed. For achieving these two goals night flights restrictions were put into place. Afterwards a study on the health effects of sleep disturbance was carried out. This study shown that there were 130 000 people having their sleep seriously disturbed by noise, therefore several measures were studied to reduce this number: a night quota, a quieter fleet, quiet approach procedures, land use planning, insulation and public awareness campaigns. After applying these measures the number of people with sleep disturbance was reduced by 18% and the airport turnover reduced by 1%.

In this case study, the threshold values were considered very important for assessing the effects and defining the areas of interest, and the dose-effect relationships important for estimating the impact. The DALY methodology was also considered very useful for proving the case.

Discussion

A wide discussion on general points to be considered in the guidelines final document was carried out after this presentation.

Regarding the metrics, at least for aircraft, the group stressed that a guideline value expressed in LAmax will have to be complementary to the Lnight! Even a very low number of flights during the night can disturb the sleep of a large number of people. The number of events is of extreme importance. This is important especially on what regards awakenings. For road and trains, because they are a more continuous source, a threshold in Lnight can be applied.

The guidelines document should also have an educative role – what are the effects, how can they be estimated, what can the decision makers do – these would be very valuable elements. Even if the group should concentrate on the ideal situation in terms of noise levels, simply ignoring the political scene will not have a positive result, only providing a number is politically naïve and may have as a consequence that the guidelines will never be put into place. A consequently and responsible analysis of the feasibility would avoid extreme criticism and a rejection from member states. The DALY approach will be important for this specific point; the politicians will be able to make comparisons between pollutants and will be able to decide on which measures to implement first.

Even with no consensus on how to deal with the point mentioned above the group agreed that, whenever possible, the necessary measures to implement the suggested guideline values should be described.

Professor Peter Lercher - Noise management philosophies

The Alpine valley case study was presented in detail. The measures were performed approximately 1000 meters from the highway, 800 meters from the main road and 400 meters from the train track. Measures were made every six to eight minutes. Different indicators were used and the results differ considerably.

Professor Lercher proposed a meeting in September with the experts from group 1 for finalising the chapter on metrics and exposure. This meeting will discuss how to consider quiet areas, how to measure the different sources and combined sources. At this stage it is predicted that Lnight will be the metric to use but LAmax and the number of events will have also to be considered. The 10 dB of penalty will be also discussed on this meeting.

Discussion

The main questions around this theme are:

- should the guidelines have source specific indicators?
- should only the acoustical side be considered or also the time of the night (with short LAeq and night distribution)?

New data from trains have shown differences with data from motorways – one train event does not have the same distribution as a truck passing by for example.
No question on the fact that the indicators should be health oriented. LAmax and number of events should, when possible, be described, although for road traffic noise it is almost impossible to count the number of events.

A consultation of the decision makers took place in Canada, they said that they want few metrics and easy to communicate – Lnight and Laeq. The night events were also considered important to consider, particularly in the shoulder hours.

Seeing the difficulty to reach an agreement a small meeting should be organized to discuss and agree on this guidelines chapter. This meeting will discuss which extra descriptors will be used with Lnight and how to deal with the different sources.

Dr. Stylianos Kephalopoulos – uncertainty

Dr Kephalopoulos made a brief presentation. He mentioned the importance of doing a risk assessment for noise, based on existing data. The uncertainty should be dealt at the exposure side, the health effects should be based in solid evidence not having uncertainty.

Uncertainty exists mainly on estimating the number of people reporting annoyance and sleep disturbance based on surveys and/or outdoor measures. The guidelines values are important mainly indoor, therefore the aspects of insulation should be considered on the document.

Whenever possible, the policy requirements should be addressed and recommendations made for developing future studies. WHO should recommend that they separate sources and assess the exposure-response relationships. Dr Kephalopoulos suggested that for each kind of health effect one metric should be established.

The guidelines should advice on noise health impact assessment suggesting a harmonisation of studies – how to consider the sources, multi-sources, specific groups of population, etc.

Something about the economic aspects of noise impact and reduction should also be included, if possible with a forthcoming view.

The participants suggested that these points are discussed in detail in the meeting of group 1.

Dr Gaetano Licitra – exposure data

Dr Licitra suggested that the group, on one hand uses the open doors of the END directive for proposing a metric that will complete the existing ones, but on the other hand, seen that the first maps (20% of the population) will be ready soon and, to use this data. The future maps will have a lot of information that can be used to identify the problems and support MS to identify the priorities in terms of noise pollution and apply the guidelines.

Discussion

Although the END directive is a crucial tool, and will support the guidelines document the group will concentrate on what is pertinent in terms of public health and find the best metrics to assess exposure.

Dr Golenj – normal sleep, insomnia

The aim of Dr Golenj's contribution was to clarify what could be considered as a normal sleep and what is sleep disturbance caused by night-time noise exposure.

Disturbed sleep is an important non-auditory effect of noise, defined as "one of those effects on health and well-being which are caused by exposure to noise, with the exclusion of effects on the hearing organ and the effects which are due to the masking of auditory information" (Stansfeld and Matheson, 2003). Noise is therefore an important environmental factor, which has a huge influence on physical, mental and social well being.

The main effect of noise on sleep is the sudden awakening; the repetition of sudden awakenings throughout the night may lead to chronic sleep loss with its usual consequences such as chronic fatigue, sleepiness during daytime, and a global poor life quality.

The definition of environmental sleep disorder and insomnia were provided. The symptoms of insomnia are similar to the ones experienced when having sleep disturbed by noise. The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) establishes the following diagnostic criteria for Primary Insomnia: a) the predominant complaint is difficulty initiating or maintaining sleep, or nonrestorative sleep, for at least 1 month, b) the sleep disturbance (or associated daytime fatigue) causes clinically significant distress or impairment in social, occupational, or other important areas of functioning, c) the sleep disturbance does not occur exclusively during the course of Narcolepsy, Breathing-Related Sleep Disorder, Circadian Rhythm Sleep Disorder, or a Parasomnia, d) the disturbance does not occur exclusively during the course of another mental disorder (e.g., Major Depressive Disorder, Generalized Anxiety Disorder, a Delirium) and e) the disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

Excessive daytime sleepiness is a consequence of disturbed night sleep and can be objectively assessed providing an objective quantification of "sleepiness". There are significant effects of noise-induced sleep disturbance. A suitable noise descriptor should be used, having maximum

noise levels and a maximum number of noise events. Changes of sleep quality and quantity appear at levels above 35 dBA.

The American Sleep Disorders Association (ASDA) devised a scoring system, taking sequences of 3-15 seconds into account for transient arousals which are not transferred to macroscopic behavioural awakening. An arousal index providing the arousal density (events/hour of sleep) was taken as a measure of the degree of severeness. 10-20 arousals/hour are considered as normal in the healthy adults. However, the use of EEG arousals with the ADSA definition provides no sufficient explanation of daytime sleepiness (Pitson and Stradling 1998, Ayas et al 2001) unless they are accompanied by vegetative arousals.

Regarding noise, different vigilance level assessment in various functional systems are important. Koella (1988) proposed investigations of vegetative, motoric, sensoric functions independently of each other. One of the possible factors indicating disturbed sleep is a vegetative arousal index. A vegetative arousal index of more than 30/h is certainly considered as serious, more than 20/h as intermediate and more than 10 as light forms of sleep disorder.

Discussion

Arousals are normal during a healthy sleep. 10 to 20 arousals per hour are considered normal in an adult, simply the analysis of arousals is not sufficient. Arousals are activated by the central nervous system and are very important for sleep processing; the problem happens when they are provoked by external factors and abnormally increased. Conscious awakenings are completely different. Per night one or two occur, that can be followed by 10 to 20 minutes going back to sleep.

There is also a noteworthy age effect related to this “normality”. The best way forward could be to talk about sleep quality and subjective sleep quality. Knowledge of the sleep duration is also important.

There is still a problem among different sleep laboratories because the standards are different. Nowadays technology is easier transportable, therefore the results between field and laboratory studies are merging.

Professor Alain Muzet – Short-term effects of transportation noise on sleep with specific attention to mechanisms and possible health impact

The following presentation by Professor Alain Muzet is based on a common (draft) paper by Dr Henk Miedema (who had to excuse for not being able to attend the meeting) and Professor Alain Muzet. He made a brief overview of the characteristics of normal sleep and of the mechanisms through which noise affects sleep. Afterwards the findings for EEG response, cardiovascular response, body movement, and conscious awakening were presented.

Noise may cause the following short-term effects on the sleep structure: delay of sleep onset, awakenings, sleep stage changes, electroencephalographic arousals, global modifications in temporal organisation of sleep stages, total time spent in different sleep stages, and premature final awakening. In addition to these effects, other short-term effects caused by noise are modifications in the autonomic functions (heart rate, blood pressure, vasoconstriction and respiratory rate) and body movements (manifestations of noise-induced arousal during sleep).

Limited arousal reactions without further consequences for sleep can be observed at low noise intensity. Indications of limited arousal are changes in the EEG pattern for a few seconds (disappearance of slow waves or sleep spindles, occurrence of alpha and/or fast EEG waves) together with autonomic signs of activation (increased heart rate for a few seconds, peripheral vasoconstriction). At a higher level, the arousal reaction may be accompanied by sleep stage changes most often associated with EEG and EMG artefacts due to body movements of the sleeper. High arousal can cause a sudden transition from sleep to wake.

The relations between motility and conscious awakening imply that motility is sensitive to noise and has a relatively low threshold, while conscious awakening (the strongest instantaneous interference of noise with sleep) has the highest threshold of the instantaneous effects considered.

In one of the most sophisticated field studies (Passchier-Vermeer et al (2002)), increased probability of instantaneous motility was found for events with a maximum sound level *LAmax* ≥ 32 dB(A), while in a meta-analysis conscious awakening was found for events with *LAmax* ≥ 42 dB(A) (Passchier-Vermeer, 2003). Above their threshold, these effects were found to increase monotonously as a function of the maximum sound level during a noise event (aircraft noise). It is important to note that in another recent sophisticated field study (Basner et al., 2004), the threshold found for EEG awakening was *LAmax* = 35 dB(A), i.e., only a little higher than the 32 dB(A) found for noise-induced awakenings. This strengthens the evidence that noise starts to induce arousals at *LAmax* values in the range 30 – 35 (A).

When deriving a health based limit the dose-dependent effects of a single noise event needs to be considered. With respect to the dose-dependent effects of a single event, adverse effects can be distinguished from effects that by themselves need not be adverse but can contribute to an adverse state. According to this, it was proposed to classify conscious awakenings by noise as an adverse effect during sleep. Conscious awakenings have been estimated to occur at a baseline rate of 1.8 awakening per night, so that an additional noise induced awakening means on the average a substantial increment of conscious awakening. Since in general falling asleep after conscious awakening takes some time, and this latency is longer after noise induced conscious awakening that will often also induce an emotional reaction (anger, fear), it will also reduce the time asleep and may affect mood and functioning next day. *LAmax* = 42 dB(A) was proposed as the currently best estimate of the threshold for conscious awakening by transportation noise. This would mean that the No Observed Adverse Effect Level (*NOAELAmax*) for transportation noise events is at most 42 dB(A).

The most sensitive instantaneous effect that has been studied extensively in field studies is motility. A single interval with (onset of) noise induced motility by itself cannot be considered to be adverse. However, noise induced motility is a sign of arousal, and frequent micro-arousal and accompanying sleep fragmentation can affect mood and functioning next day and lead to a lower rating of the sleep quality. Therefore, motility is relevant for adverse health effects, but more than a few intervals with noise induced motility are needed for inducing such effects. Although additional, more sophisticated analyses could be performed to refine this estimate. *LAmax* = 32 dB(A) was proposed as the currently best estimate of the threshold for motility induced by transportation noise. It is important to note that the above given *NOEL LAmax* ≤ 32 dB(A) and *NOAEL LAmax* ≤ 42 dB(A) are indoor levels, in the sleeping room.

The following groups may be hypothesized to be more vulnerable to noise during sleep: old people, ill people, people with chronic insomnia, shift workers and people resting during daytime, people with tendency to depression, light sleepers, pregnant women, people with high anxiety and high stress levels. Furthermore, children need special attention because of their relatively

high exposure during sleep, and because they are in a crucial phase for neurocognitive development during which having an undisturbed sleep may be particularly important.

Discussion
The German sleep association claims that even when a person does not recall being awake there are possible adverse consequences. Conscious awakenings are the extreme event, for example sleep apnoea patients do not complain about their sleep and their sleep is not efficient. The noise level necessary for provoking an awakening also depends on the sleep stage a person is in.

What we are missing as well is the hormones secretion during sleep. There is still a big gap on knowledge on this matter – although some evidence exists for road traffic.

Lnight of 42 dB(A) and *LAmax* ≤ 32 dB(A) indoor are the NOAEL (for "normal and healthy" sleepers. Can also a number of events be associated with these figures?

Dr Sona Nevismalova - Long term noise-induced health risk mediated particularly by sleep disturbances

The aim of this presentation was to describe the possible long-term consequences of sleep disturbances evoked by noise as the main environmental stressor. Insufficient nocturnal sleep and sleep restriction give rise to behavioural and cognitive impairment, even neurological and other medical problems, and reflect negatively on the affected subjects´ quality of life.

Dr Nevismalova updated her paper according with the second meeting and has proposed some noise levels for avoid sleep disturbance by noise.

Critical health effects can appear when maximum average nocturnal energy level inside bedrooms exceeds Leq 30 dB(A) and outside bedrooms Leq 45 dB(A) and in the presence of a limited number of events per hour with an energy exceeding the baseline level of LA max. 45 dB(A) level (inside bedrooms) and/or 60 dB(A) outside bedrooms. The peak level of 60 dB(A) inside bedrooms and/or 75 dB(A) outside bedrooms should never be exceeded in any case.

Apart from those for the healthy population, norms should be devised and strictly adhered to in hospitals, particularly at intensive care units.

Professor Stephen Stansfeld, Dr Michal Skalski – night noise and mental health

Professor Stansfeld completed and updated the background paper himself and Dr Skalski produced for the second meeting. Noise exposure at night may be more disturbing than daytime noise because it interferes with rest and sleep at a time when people want to relax. It seems plausible that night time noise might have a particular effect on mental health. However, there is little direct research on night time noise and mental health and it is first necessary to consider the evidence for environmental noise and mental health in general.

At the present time, exposure-effect associations have not been established between parameters of sleep disturbance (number of behavioural awakenings, body movements or EEG awakenings) and the onset of depressive and anxiety disorders although there is some evidence that insomnia is a risk factor for developing depression (Riemann et al, 2001; Roberts et al,

2002). A number of longitudinal prospective studies in different age groups have found associations between self reports of insomnia and the subsequent onset of psychiatric disorder, in particular major depression.

The evidence is not strong for the association between noise exposure and mental ill-health. The existing evidence suggests that noise exposure may be responsible for psychological symptoms above 70 dBA Leq. Almost all studies have only examined the effects of day time noise on mental health, but it is possible that night time noise, during sleep time, may have effects on mental health at lower levels than day time noise.

The most powerful evidence of noise on mental health comes from studies of military aircraft noise. There is also some evidence that intense road traffic noise may lead to psychological symptoms. There is no evidence of any effects of railway noise on mental health.

Discussion

A person's house is a place where to feel secure from outside aggressors. Noise has an intrusive character and might provoke anxiety. Being able to control it is of crucial importance in terms of mental health effects. The level of anxiety that it can provoke depends on the individual coping with the noise.

There is a genuine lack of evidence. Insomnia predicts all kind of health effects therefore it is very difficult to use it as a proxy. There is not enough knowledge of what happens before (depression or insomnia).

Dr Wolfgang Babisch - Health effects related to stress mechanisms - Cardiovascular effects

Dr Babisch, likewise other partners, also updated his background paper for this meeting. He presented the comprehensive meta-analysis we carried out and added guideline values for the CVD effects.

Sufficient evidence exists for an association between community noise and ischaemic heart diseases; limited/sufficient evidence exists for an association between community noise and hypertension; Most information comes from road traffic noise studies but there is normally little information regarding night noise, in particular. But night time values can be extrapolated from day time results.

Below 60 dB(A) for Lday there is no noticeable increase in MI risk to be detected. Therefore for the time-being, Lday = 60 dB(A) can be set as the NOAEL ("no observed adverse effect level") for road traffic noise and myocardial infarction (Babisch, 2002). For noise levels greater than 60 dB(A), the MI risk increases continuously, and is greater than 1.2 for noise levels of 70 dB(A).

Discussion

Normally CVD effects manifest after 10 years living in a noisy area.

There was a question whether the results from daytime could be translated to night time. If a person reacts the same way while sleeping, and the answer was yes. In the Spandau study the results are not so different.

However, sleeping a person reacts to lower levels, therefore the 10 dB from the Lnight was established to consider it, although the sleep labs consider 10 to 15 dB difference. There is also literature showing that even if the noise was 15 dB lower the person reacted 2 times more! When a person is asleep he/she don't cope with the noise.

The aspect of measuring at the façade and having the bedrooms in the back of the dwelling was raised. If we are analysing day time effects the fact that people may sleep in the back has to be considered. Dr Babisch clarified that these studies have a conservative approach considering only the lower exposure side of the dwellings with control groups that don't have noise, during the day and during the night, so what can happen is a underestimation and not the other way around.

Significant results for hypertension and military aircraft were found in Japanese and German (Ising) studies, but, for example, on the German study there were no night flights. Maybe it would be possible to find if night flights can be extrapolated from the Okinawa study. This would be important to establish a number of events.

Professor Staffan Hygge - Night noise and cognitive impairment

The relationships between night time levels and cognitive performance mediated by reduced sleep quality were analysed. Professor Hygge concentrated on night time levels, children and adults differences, and on the role of restoration. The results of the Munich study and RANCH (by Professor Stansfeld) were presented.

On the Munich study there were errors in reading that were significantly reduced after the airport closure. On the other hand the errors increased in the school close to the new airport. But there does not seem to be any effect of night time noise exposure on children's self reported sleep quality, in spite of a substantial average shift in combined day-night noise level in the exposed group at the old and new airports.

Unfortunately there is no information on night noise levels. It is not known exactly how much aircraft night noise there was at the two airports, only that the airport operated further into the night. Considering the sleeping hours for children aged 9-12 years, there should have been some early night noise level difference between groups. However this did not show up as loss of sleep quality, neither for the children, nor for their parents.

The RANCH study focused on noise at schools during the day and relationships were found for reading, episodic memory and increased noise.

In the United Kingdom, 842 children were surveyed from 23:00 to 7:00 with a noise level of 53 dB(A) in day, 42.9 dB(A) night. As results night time aircraft exposure showed no additional impact on reading and recognition memory beyond the effects of daytime noise exposure.

It has to be strongly acknowledged, though, that neither the Munich nor the RANCH studies were designed to assess the impact of night noise on cognitive functions. There is only a survey question on the self assessed sleep quality, and it is known that this is not the best way to assess sleep quality.

Discussion

On both studies presented we don't know how the children slept, therefore is it difficult to derive any conclusions. The results are not enough to make conclusions neither it has or it does not have an effect.

It is also a known fact that people living close to an airport quite often misevaluated their sleep quality.

Children do not wake up as easily as adults, they seem to have a protective "system" that protects their sleep, but there is a strong reactivity to noise and there are effects occurring at the autonomic functions.

Dr Snenaza Yovanovic – Children accidents

From the existing sleep literature it can be concluded that children with disturbed sleep present cognitive dysfunction and behavioural disturbances, abnormal growth hormone release, increase of diastolic blood pressure, increased risk of accidents and use of sleeping pills.

According to a study (Menchini et al. 1985) childhood accidents occur more frequently during the spring and the summer. Falls, cuts and laceration, unintentional ingestion, pedestrian injuries, burns and scalds, choking episodes and animal related injuries are the most common types of injuries and also the most likely linked to sleep disturbance in young children. Results suggest that children with more frequent injuries had significantly more sleep problems. And that there is an increased prevalence of sleep disturbances in preschoolers with increased injury rates.

Sleep disorders are often more mentioned by children than by their parents, likewise sleep disturbances by light and noise!

Results of a study on sleep disturbance and injury risk in young children show that inadequate sleep duration and lack of daytime naps are transient exposures that may increase the risk of injury among children. Among children (boys particular) from 3 to 5 years of age, sleeping less than 10 hours a day was associated with an 86% increase in injury risk.

Daytime sleepiness in children is often manifested by externalizing behaviours noted by parents or teachers such as increased activity levels, aggression, impulsivity, as well as by poor concentration, instantiation and moodiness (Fallone et al., 2002).

Duration of sleep required to minimize injury risk in childhood is not simply quantified, because normative values for sleep duration vary across cultures. In a study with 40 000 children (11 to 16 year old) from 11 countries, significant differences of average sleeping times between countries were found. Israeli children are the ones that sleep less time and Swiss sleep the longest (Tynjala et al. 1993). As an example, American infants in the 1990s slept 13 hours per day at age of 3 months, and Dutch babies slept 15 hours per day.

Analysing attendance to school, data shows that accidents take place at school (25.6%) and at home (22.0%), and there is a statistically highly significant greater total accident rate in boys than in girls. The most frequent injuries happening at school are fractures and dislocation of joints, being the head injuries more common among school injuries compared with spare-time injuries. Most injuries occurred when children are in sport areas and 25% of all injuries were caused by other pupils by intentional violence.

Regarding sleep disturbance and accidents in adults data shows that 15 - 45% of all patients suffering from sleep apnea, 12 – 30% of all patients suffering from narkolepsie and 2 – 8% of all patients suffering from insomnia have at least one accident (in a life time) related to sleepiness (statistics from the Stanford Sleep Disorders Clinic).

A very interesting analysis was carried by Leger et al showing the costs of road, work, home and public accidents and the estimation of this costs attributed to sleepiness – normally in all cases the attributed cost is very high (e.g motor vehicle accidents total costs are 70.2 MRD$ being the costs attributed to sleepiness 41,5% of this amount).

Discussion

The fact that people are sleepy because they decide not to sleep was raised. Can a differentiation be made between the ones that don't want to sleep and the ones that are prevented from sleeping? This is very difficult to differentiate, of course, but we can have an attributable fraction of people being disturbed by noise and then extrapolate on the percentage of accidents being caused by it.

Dr Christian Maschke - Effects of noise on the immune system

The presentation of Dr Maschke concentrated on hormone secretion and its health signification. It is not a simple topic and the mechanisms have to be explained in detail.

In the last 20 years psycho-neuro-immunology has developed into an independent discipline. Psycho-neuro-immunology deals with the interactions between the nervous, hormone, and immune systems. Stress, behavior, condition and sleep are the focus of research. Psycho-neuro-immunology has expanded the knowledge of the hormonal system considerably. Until now, noise effect research has not kept pace with this development.

The hormonal system is active in every single cell of a human being. The hormonal system contains the endocrine part that consists in secretion of hormones from special glands, each with a special function. The endocrine part includes the hypophysis as the control gland of the endocrine system, controlled by the hypothalamus by means of inhibiting or stimulating hormones and the thyroid, adrenal cortex, pancreas, parathyroid, gonads, epiphysis (pineal body) and the thymus, which also maintains the connection to the immune system.

The adrenal medulla also represents an endocrine gland. However, it is controlled by the vegetative nervous system.

The paracrine part of the hormone system consists of special cells which are distributed throughout the entire body. The hormones which are produced in these cells are described as tissue hormones. While the endocrine system is secreting the hormones into the blood, the paracrine hormones are passed on through inter-tissues and brought to the final destination.

Nerve cells are also able to produce hormones (neurohormones and neurocrine part of the hormone system). Neurohormones can be secreted into the blood or taken to the synapse directly.

The immune system is narrowly connected with the hormonal system. Certain hormones stimulate the receptors of the immune system and trigger immunologic effects.

Already from this short description it is obvious that the neuro-endocrine activity is much more complicated than was described by Hans Selye [1936]. Therefore, an isolated view of the so-called stress hormones (catecholamine, cortisol) does not do always justice to neuroendocrine activity.

To conclude, even if information is available on cortisol secretion when of a noisy event, it is not an easy task to assess its health significance. The most advanced studies on this field have never considered noise. Extrapolating a NOEL could be done, but its health significance is not possible to quantify at this stage. The hormones are not health significant alone.

Discussion

Even with limited knowledge on noise and hormones can some of the existing knowledge of hormone secretion be included on the guidelines? It is a difficult question, a normal level of cortisol, per se, does not mean anything, an abnormal cortisol level means that there is a deregulation but the literature does not say anything about its health meaning. There are only two studies on noise and cortisol, we need mores studies! Results from other existing studies are contradictory.

If included on the guidelines this topic needs to be more detailed. The group proposed WHO to organize a small meeting for this topic exclusively. This meeting should deal with the available knowledge, existing evidence, identify gaps in knowledge and if possible and have a pedagogic character for noise experts and as reference for future studies.

Professor Oliviero Bruni - How to take into account the specific needs of children. Children's sleep

Professor Bruni updated the paper he had prepared for the second meeting according with the meeting discussion. More precisely he had been requested to give the assumed criteria and explain in detail the table he had proposed on the Geneva meeting.

For children, the guidelines values should be those ensuring a healthy sleep. For the time being, this can be the lowest level of noise for which studies demonstrate a clear and significant disturbance of sleep in children.

The night period, when the noise levels suppose to be low, is becoming very short (3 to 4 hours). Recently, a traffic analysis in most Italian cities showed that noise limits are exceeded almost every night and, in percentage, more during night-time than during daytime.

The CAP rate (mostly A1 index) shows a strict relationship with Growth Hormone (GH) secretion and with growth velocity. The effects of noise could act disrupting Slow Wave Sleep microstructure and therefore GH secretion.

The Slow Wave Sleep (SWS) and REM sleep play an important functions in children's sleep. Having an undisturbed sleep during these stages is related to normal growth and learning. If the noise occurs during the 1° cycle of SWS when growth hormone is secreted, it could have more detrimental effects than during other phases and if it happened in adults.

Although it should be taken into account that it is at present impossible to indicate whether children are possibly more sensitive than adults to night-time noise, it can be deduced that the effects of night-time noise during sleep in adults also apply to children. For the effects for which

solid evidence is missing we should use an approximate approach based on daytime findings and based on the precautionary principle.

The metric to be adopted should consider the average background noise level that does not provoke any effect and should allow the evaluation of the number of arousals and awakenings. It is known that adults physiological responses appear at an LAeq in the bedroom of approximately 40 dB(A) and behavioural awakening occurs when the bedroom Lmax exceeds 55 dB(A). Since the children autonomous nervous system is more activated by noise (acoustic stimuli of much lesser intensity 10 to 15 dB) when sleeping, it can be assumed that children are more sensitive also to the detrimental effects of noise exposure.

All children (0-12) should be considered at risk, but especial attention should be paid to some specific groups such as low birth weight, small for gestational age babies, preterm infants, learning disorders, behavioural disturbances (i.e. hyperactivity) and sleep disordered children.

Professor Hartmut Ising - Animal Studies

The results of animal experiments, experiments on humans, and epidemiological studies show consistently that chronic exposure to environmental noise increases the risk of hypertension.

According to a model, established by Professor Ising and Dr Kruppa, arousals precede endocrine reactions and appear within 1 second after a noise stimulus, hormones like catecholamines take several minutes, and cortisol about 10 minutes to be increased. This leads to the conclusion that noise exposure when it does not evoke arousals in sleeping persons will not induce adverse health effects.

On a study with 68 children the excretion of cortisol was measured in the first and in the second half of the night during a period of 5 years (by their physicians). Night time exposure to road traffic noise was correlated to an increased cortisol excretion in the first half of the night and this increase was significantly correlated to an increased frequency of physician contacts (reporting bronchitis).

Animal studies have shown that noise increases the Calcium and Magnesium accumulated on the cells, provoking a premature aging of the heart. Ageing is one of the biggest risk factors for heart attack and this effect of noise should not be underestimated.

Long term exposure of children to the combination of traffic noise and air pollution aggravates respiratory diseases more than exposure to air pollution alone.

Discussion
The type of animals used should be described on the final document, because some types of mice don't absorb magnesium.
How can we translate these results to humans? The experiments described are with chronic noise exposure, which almost never happens in humans. This has to be accurately acknowledged.

Mr Colin Grimwood - Neighbourhood Noise - guidelines

Mr Grimwood presentation consisted of a series of very pertinent questions about the future guidelines document.

The first question was about the nature of the noise. Are the guidelines being defined by noise type 'continuous noise', and 'intermittent noise events' (like the existing WHO Guidelines) or by noise source (specific?/general?)? Transport noise , neighbour noise - noise produced by a person's neighbours (and their pets), neighbourhood noise - all noise produced in the neighbourhood, but not noise from neighbours or noise from transportation (i.e. includes entertainment, sport and leisure, industry, commercial premises, construction and demolition, roadworks, farming, agriculture ……), or even by effect(s) of noise? (sleep effects the only critical health effects at night?), or applied to specific environments? (adults'/ children' / pre-school bedrooms? Recommending use of time limits - times when neighbourhood noise can and can't be made (or heard) e.g. no lawnmowers before 9am or after 9 pm, no fireworks after midnight)?:

Establishing a threshold level for neighbourhood noise is an almost impossible task, should inaudibility be used as the no effect level for night time? Inaudibility is currently used as a guideline for night time entertainment noise in Scotland. At present the WHO 30 dBA guideline value is often cited in legal proceedings as a justification to continue making night time neighbourhood noise. Neighbourhood noise complaints are common at noise levels of 30 dBA.

The other questions addressed were (and answer):

1. Are existing laboratory and field (transport noise) research findings on dose/response, noise events and motility, habituation, sleep effects, effects of change in noise level, role of acoustic and non acoustic factors applicable to all the different types and sources of neighbourhood noise? Not sure

2. Are there unique factors (acoustic and non-acoustic) that are applicable to the different types of neighbourhood noise? Yes!

3. Do the following acoustic factors mean that it is not appropriate to derive guideline values for neighbourhood noise?
   - Sound pressure level
   - Loudness
   - Audibility
   - Spectral characteristics, low frequency content
   - Tonality
   - Impulsiveness
   - Irregularity
   - Intermittency
   - Unpredictability
   - Duration
   - Emergence over background

4. And these also relevant non acoustic factors?
   - Time-of-day, season of year
   - Type of area
   - Attitudes to source
   - Information content
   - Neuroticism
   - Negative affectivity
   - Individual sensitivity to related pollutants
   - Expectation

- History of noise exposure
- Employment or other connection with the noise source
- Education
- Home ownership, type of dwelling, length of residence ….

5. Does the mentioned above facts mean that the existing body of noise research is not sufficient to allow specific authoritative guideline values to be derived for individual neighbourhood noise sources? Yes!

Do these factors mean that it is not appropriate to derive guideline values for neighbourhood noise? Not sure!

Or

The use of a Guideline Value may be appropriate but only in circumstances where the acoustic and non acoustic features of the neighbourhood noise are not important?

Or

That the general research findings on sleep effects and noise are considered relevant to neighbourhood noise irrespective of the acoustic and non acoustic factors?

Or

That the general research findings on sleep effects and noise are considered relevant where the type of neighbourhood noise has similar features to transport noise (i.e. continuous or consists of a number of intermittent but similar events)?

6. What should WHO NNG say about neighbourhood noise?

- That the no effect level for many types of neighbourhood noise may be inaudibility?

- That the no adverse effect level for some types of neighbourhood noise may be the same as that derived from transport noise studies particularly where the neighbourhood noise is continuous or consists of a number of intermittent but similar events?

- That the no adverse effect level for many types of neighbourhood noise is probably lower than that derived from transport noise studies because of the special features of this type of noise?

This very provocative presentation caused a lively discussion; most of them were also addressed at the plenary section and originated conclusions.

Discussion

Inaudibility can not be the guideline value, this would cause very strong reactions and it will be a value impossible to achieve. If WHO proposes this value it can originate adverse effects if taken out of the context and put in risk the credibility of the group.

Neighbour and neighbourhood noise will be a separate chapter of the guidelines, they are clearly different from transport sources. This chapter can also have a pedagogic character to providence guidance for future studies, studies are needed on this field.

Dr Mueller – Wenk - Determining the severity of noise-related human health effects within an established health metric system

Dr Mueller Wenk, similar to other partners, updated his paper according to discussions of the second meeting.

Dr Mueller Wenk considered sleep disturbance caused by a long term exposure to nocturnal road noise as a disease itself. He based this statement on the judgements of patients having Obstructive Sleep Apnea Syndrome as well as with noise-related sleep disturbance.

Under the conditions of a highly developed and densely populated country like Switzerland, the night time noise due to road traffic causes approximately 50000 cases of sleep disturbance per million of persons. With a disability weight of 0.055, this results in a Burden of Disease that is at least as important as the Burden of Disease due to premature deaths caused by road accidents.

Even if calculations are made on the basis of recent studies that are considered to underestimate the road noise exposure of the population (ARE 2004), as well as the night-time noise sensitivity of the population (DLR study Samel 2004), it can be shown that the corresponding 'low' value of the Burden of Disease due to road-noise-related sleep disturbance is of a magnitude that justifies and requires public action.

Discussion

Is the method used for Switzerland also valid for the other countries? WHO has a parallel working group for estimating the Burden of disease of noise and the night time noise results will be included on the guidelines.

Conclusions

1. The guidelines will be established for, mainly, sleep protection (objective and subjective sleep quality), only a small part of the document will be dedicated to annoyance for awakened people during the night;

2. The possible measures that Member States should undertake to implement the guideline values should be described when possible;

3. The final document should include success stories and noise reduction case studies;

4. The message has to be clear to be understandable by the majority of people;

5. Times of night; number of events; and duration of the events should be considered, but the metrics should be kept simple. Lnight and LAmax per source will be used;

6. A small meeting could be organized on exposure and metrics (probably in September in Vienna);

7. Sleep is measured objectively and subjectively - The final document will describe sleep quality and insomnia;

8. There is unanimous consensus that a disturbed sleep has serious health effects – solid evidence exists in sleep medicine, the insomnia model will be used as a proxy and its causes and effects described on the final document;

9. The analysis of the evidence suggest that L_{night} outdoor>42 dB(A) induces sleep disturbances;

10. The NOAEL (non observed adverse effect level) (during sleep) is L_{night} 42 dB(A) outdoors;

11. Positive attitude towards noise can minimize its effects;

12. Standards should be devised and strictly adhered to in hospitals, particularly at intensive care units;

13. The autonomic nervous system is more easily activated in children than in adults; there seams to exist a “protective” mechanism that keeps them sleeping – sleeping is crucial;

14. Studies carried out in the United States will be included – Dr Michaud will provide the pertinent ones;

15. Noise does not seem to have a direct influence on mental health, maybe sleep mediated – but there are not enough studies – the evidence simply does not exist!

16. For military aircraft dose response relationships between aircraft noise and ‘mental instability’ and ‘depressiveness’, were found but only for high levels >70 Ldn;

17. Perceived “controllability” of noise (possibility of switch it off) is of crucial importance;

18. The NOAEL for Myocardial Infarction is L_{day} = 60-65 dB outdoors and L_{night} outdoors = 50 – 55 dB for road traffic;

19. It was not shown that the recorded 24 h noise levels in the Munich study impaired self reported sleep quality, neither for the children, nor for their parents but the study was not developed to analyse sleep disturbance;

20. Chronic exposure to environmental noise increases the risk of hypertension;

21. Animal studies show that excessive exposure to noise causes aging of the heart – the main risk for myocardial infarction;

22. The guidelines will be, for transportation noise defined by source; In addition continuous noise’, and ‘intermittent noise events’ will be described;

23. The “deriving guidelines” chapter on the final document will summarize the background papers trying to have an easily understandable structure (per source and per effect);

24. A first draft of the chapter “deriving guidelines” should be produced by Mr Van den Berg, proposing an answer to the questions around it and the expert group will then comment and improve it!

25. A chapter should be devoted to (neighbour) neighbourhood noise – its contents will be “different” from the other sources - No specific guidelines values will be developed for neighbourhood (and neighbour) noise, only when possible they will be derived from transportation noise studies – with special restrictions;

26. A chapter should be developed on the burden of disease of night noise supported by the work WHO is developing in parallel;

27. Each time possible the health effects of noise exposure to levels above proposed guidelines should be described and quantified;

28. A smaller meeting on hormone secretion and their significance could be organized;

29. developing a table like the following was considered useful, if feasible:

Effect	**Lnight outdoor**		**Number of events with duration**	**Lamax outdoor Or difference between background level**	**Duration of exposure**
Sleep disturbance Children	Road	42 dB(A)	Arousals /CAP rate – which level?		
	Rail				
	Air				
	Combined				
Sleep disturbance Adults	Road				
	Rail				
	Air				
	Combined				
Cardiovascular diseases - Ischemic heart disease - hypertension	Road	50 dB			10 years
	Rail				
	Air				
	Combined				
Other					

Follow up - Timetable

According to the contract with the European Commission, the project should end in June 2006. For be able to finalize within the delay two smaller meetings could be organized in

- Sept 2005 – exposure, metrics – in Vienna
- October 2005 – health significance of hormone secretion

The partners should finalize their papers as soon as possible (or sent their paper to secretariat if not yet presented). The final document will summarize them according to the table of contents presented in annex 2 (still not agreed upon).

A professional senior editor will be hired to compile and summarize the background documents. He/she will produce a first draft that will be revised by this group. After corrections, an external group of experts will also be requested for review the document and the results will be discussed on a final meeting.

In May 2006 the project final meeting with the participation of WHO / EC Member States will be organized in Bonn. This is envisaged to be a technical and political meeting, 1 day for exclusively technical aspects and one or two days for the political part.

Annex 1 – Proposed table of contents of the final document

Night noise Guidelines – protecting sleep!
Executive summary

CHAPTER I - INTRODUCTION: METHODS AND CRITERIA

1. Preface

2. Strength of evidence

3. Considerations with regard to night-time noise indicators
3.1 Length of night – shoulder hours
3.2 Event or long-term descriptor
3.3 Number of events
3.4 Inside / outside differences

4. Exposure in the population
4.1. Noise sources
4.1. Noise levels
4.2 Surveys

CHAPTER II - SLEEP AND HEALTH

1. Sleep, normal sleep, sleep disturbance, characteristics mechanisms, the insomnia model
2. Long term noise-induced health risk mediated particularly by sleep disturbances
3. Health effects of disturbed sleep in children
4. Accidents related to sleep quality
5. Animal studies
6. Burden of Disease
7. Conclusions

CHAPTER III – STRESS MODEL

CHAPTER IV - EFFECTS OF NIGHT TIME NOISE ON SLEEP

1 Short term effects of transportation noise on sleep with specific attention to mechanisms and possible health impact
1.1. Dose-effect relations
1.2. Chronic effects

CHAPTER V. EFFECTS OF NIGHT TIME NOISE ON HEALTH

1. Self-reported (chronic) sleep disturbances
2. Medicine use
3. Cardio vascular effects
4. Effects on performance
5. Effects on psychic disorders

CHAPTER VI. GUIDELINES AND RECOMMENDATIONS

1. NO(A)ELs
2. Dealing with situations exceeding the NOAEL's
3. Neighbourhood noise and noise from neighbours
4. Protection measures & control
5. Recommendations
6. References

Annexes

Annex 2 - List of participants

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Annex 6: Meeting report on the fourth meeting on night noise guidelines

Report on the fourth meeting on night noise guidelines

Den Hague, 16th September 2005

European Centre for Environment and Health
(Bonn Office)
World Health Organization Regional Office for Europe

Participants:
Professor Jacques Beaumont
Mr Martin van den Berg
Mr Xavier Bonnefoy
Mr. Dick Botteldooren
Dr. Danny Houthuijs
Professor Peter Lercher
Mr. Henk Miedema
Ms Célia Rodrigues

Background

The meeting was coordinated by Martin van den Berg and Peter Lercher to discuss the acoustic aspects of the night noise guidelines document. Discussions took place on the relevance and practicability of different indicators.

Introduction

Professor Lercher introduced the group with some thoughts about the tasks that should be considered by the group:

- Ensure that the application of the guidelines will be easy reproducible and affordable

- The use of an integrated indicator for the setting of limits has advantages and disadvantages (see Muzet 2002). One major merit of using an integrated indicator is the ability to have a global noise description of an area that can be easily compared and areas suffering most easily identified. On the other hand, in impact assessments studies (SEA, EHIA), guidance is needed to assess the various options. In this case, the use of one indicator (Leq or Lden) is not enough to provide sufficient guidance. For example, when dealing with airports, it does not help much to take only sanctions against loud aircrafts, the number of aircraft movements is also important. Here, in addition, a single noise event indicator is required which can easily be monitored (Muzet 2002).

- The advantages or disadvantages of various indicators for different purposes should be described, providing a framework.

Mr Bonnefoy started the session by reminding some of the unanswered questions in the NNGL project:

- Is there one or few indicators to assess the various impacts on health & well-being?

- One of the goals of the NNGL project is also to make a proposal on the validity of the night penalty factor to be allocated to night time noise in the calculation of Lden. A report describing the expert consensus on possible ways to amend the penalty added to night noise levels in the calculations of Lden should be provided. The group has, therefore, to pay attention to this issue. Is there enough evidence to revisit the 10dB penalty?

The data used for establishing this penalty was based on annoyance surveys. The group agreed to say that there are no new data available to re-review the evidence to this date. It is well known, however, that the 10dB were a compromise. The true night-time sensitivity from this earlier database (Jansen and others?) was around 15 dB. No studies on cardio vascular were considered, mainly because it is difficult to separate the day and night exposure.
The final report will refer to this problem and propose continuing to use a 10 dB penalty until further evidence is available.

- Are there sufficient data to prove whether the cardio vascular effects are related to night time?

This question comes on the follow up question of the 10 dB penalty. If the penalty of Lnight is increased the night time guideline would be more protective. However the current evidence on cardio vascular effects does not allow assessing which period of exposure (day or night) is more important as the studies carried out until now do not differentiate the exposure period. The evidence is insufficient (Babisch) to maintain the status quo in terms of recommendation.

Mr Bonnefoy also reminded the group that sleep during shoulder hours should be protected. A way of assessing the number of events that are above the average background noise level should be established, in order to protect the sleep during these critical periods of time.

Professor Jacques Beaumont's presentation

Professor Beaumont has prepared for the "Forum Acusticum 2005" in Budapest a paper called "Event descriptors for qualifying the urban sound environment" describing the main existing indicators and making a proposal for an indicator to be used in urban situations exposed to traffic noise in counting emergences with respect to fluctuant background noise.

He presented this paper and made a proposal for two indicators to be used for railway noise and aircraft noise.

Professor Beaumont proposed to use "event" indexes instead of energy indexes. This type of index, contrary to energy indexes that average sound level over often long periods, characterise sound events localized in time and space. The energy developed is no longer the main criterion. He suggested the use of:

for train: TEL (Transit Exposure level - integration of level & number)
for aircraft: Lenp (weighting of noise characteristics)

these indicators take into account at the same time the energy of an event and the durations of its occurrence. They describe better situations with fluctuating traffic and allow evaluating peak effects and the magnitude and frequency of variation of the noise level over the studied period.

As metric to be used in the guidelines Professor Beaumont proposed a combined use of these two indexes and Lmax > 70 dBA and number events exceeding it >5/8 calculated in the shoulder hours and outdoors.

The concept of "emergence" is already used in French guidelines. It represents the difference in dB between the background level and the energy of the event.

Discussion

Mr Bonnefoy reminded the group that "sleep pathologists" and their findings on the adverse effects of arousals would lead to consider that: 10dB above average value (emergence) gives arousal (which is a common rule of thumb -also used in the assessment of neighbourhood annoyance by industrial noise).
Therefore the number of events with such an emergence should be integrated in a useful manner.

Professor Lercher: A framework should be provided that gives guidance on how to protect best the night against *different* sources and *source combinations* in *different* situations (different background noise levels, different propagation. The group felt that we should have a step wise approach first with simple sources, and then with combined sources, if at all possible.

Mr Botteldooren: threshold thinking is a one common approach: 1 event of 65 or 50 events of 40 Lmax produce the same Lnight. However, these two situations with different background levels are very different.

Dr Houthuijs: A 75dB event, causes an arousal and possibly an awakening. What is not enough considered is that such a high noise event will affect more people in a larger area, because more people can hear it. The number of people exposed should be integrated as well in the guidelines.

Mr Van den Berg quoted the Basner table (DLR-study) about the relation between number of events and effect, trading number of events against level, and the importance of time window of exposure. He further elaborated his points with a written contribution after the meeting in October 2005 (see Annex 1 below). Studies of awakenings have shown that it is rather simple to determine the likelihood of an arousal by a single event (see e.g. Fidell study or Passchier-Vermeer study). What is more difficult is to determine the effect of multiple events – what is the chance of re-awakening after an awakening has occurred.

Mr Van den Berg mentioned the actigraphic mobility method, the long-term exposure versus instantaneous. It could help answering some questions. The Passchier-Vermeer study has shown that this measure is useful. Although, certainly, a drawback is that cardiovascular arousal is not covered and therefore an underestimation of important health effects may occur.

Mr Bonnefoy reminded the sleep pathologist's concern with arousals. Arousals seem to be the common denominator: an excessive number of non-physiological arousals should be avoided to prevent cardiovascular/hormonal effects.

Mr Bonnefoy then draw a table on the blackboard which he wanted to have filled in by the acousticians. The aim of this table was to test, whether different background levels have different effects - given different N of events - on a proposed continuous guideline level.

Background noise		**No**		**30 dB**		**35 dB**	
Number of aircraft events	Lamax (dB)	LAeq 1 h	LAeq 8 h	LAeq 1 hour (dB)	LAeq 8 hours (dB)	LAeq 1 hour (dB)	LAeq 8 hours (dB)
1	**60**	**37**	**28**	**38**	**30**	**39**	**29**
	70	**45**	**36**	**45**	**37**	**45**	**39**
	80	**53**	**44**	**53**	**44**	**53**	**45**
2	**60**	**40**	**31**	**40**	**34**	**40**	**33**
	70	**42**	**39**	**42**	**40**	**42**	**40**
	80	**48**	**47**	**48**	**47**	**48**	**47**
	85	**60**	**51**	**60**	**51**	**60**	**51**
5	**60**	**44**	**35**	**44**	**36**	**44**	**36**
	70	**52**	**43**	**52**	**43**	**52**	**43**
	80	**60**	**51**	**60**	**51**	**60**	**51**
50	**60**	**54**	**45**	**54**	**45**	**54**	**45**
	70	**62**	**53**	**62**	**53**	**62**	**53**
	80	**70**	**61**	**70**	**61**	**70**	**61**

The following efforts did not reveal the wished results. While in general it seams easy to fill in this table with “abstract” values, however, it seems not to be easy to have “typical” over flights or pass by’s available for calculation. The EC position paper on indicators made some calculations with number of events and “background” levels and Lnight performed well.

Mr Dick Botteldooren’s presentation

Dick Botteldooren presented a series of slides about concepts and data which might be useful for the NNGL.

He presented data about the noise distribution during the night and during the “shoulder hours” from Belgium (Colin Grimwood has shown similar data with similar conclusions from the UK). After an approximate decrease between midnight and 3:00 hrs of 5 dB, a linear increase of about 10 dB happens until 6:00 hrs. This follows population activity pattern.
Among the parameters to be considered, the nightly sound pattern should also be considered: E.g. as duration of quiet periods or the duration of uninterrupted, undisturbed periods.

He also mentioned the potential (mis)use of parameters (to exchange level for number of events). Consequently the question comes up: Can we have a single indicator for all purposes?

By using the FICAN 1997 curve, that provide the probability of awakening, he demonstrated this curve may not apply when multiple events occur as it assumes that events are independent one from another.

Mr Botteldooren compared the different effects using the two strategies - when you use the various indicators mentioned by Professor Beaumont and in the table prepared by Mr Van den Berg - limiting the number of events (corresponding to a 10 dB reduction) versus a continuous noise limit.

He then presented a possible general extension of the NNI (noise number index) by replacing number of events by the duration of time over a given threshold: TNI70.

He presented noise data from a survey along the Brenner highway. By testing various noise sequences he demonstrated substantial differences between NNI, Lnight and TNI70. Although these data are not likely to represent situations found elsewhere it showed that Lnight is not able to distinguish sufficiently existing differences in potential disturbance.

It was again mentioned by several participants that the overwhelming majority of health effects papers are based on Leq and that only recently the Lnight was used.

Discussion

According to WHO, the chosen indicator should describe the average Lnight, the Lmax, the number of events, the number of events exceeding a certain threshold relative to the background noise level to capture the emergences. This is quite an impossible challenge.

Some participants consider that most of these indicators are well known but have not shown to have big advantages over the classic integrated ones (Leq).

In particular Henk Miedema warned that any consideration of background noise may carry the risk for arguments to increase the background noise in order to avoid health effects. It is also possible that rural areas may also be more sensitive to the effects because of the so-called “recruitment” phenomenon.

Professor Beaumont's proposed to take the background level at 40dB and use emergences by 10, 12, 15 dB as a basic framework.

Dr Henk Miedema asked for making a clear distinction between a legal framework and a scientific framework.

Professor Lercher's suggested that a series of indicators could be proposed, examples of their potential use shown in an appendix. This would open the opportunity for further testing in both research and practice and therefore serve both purposes. Then the group would go for a single indicator.

Conclusions

There are primary differences in approaches and concepts on using the Laeq indicators. In France the background level is measured and in the Netherlands it is calculated. Hence the Lnight in The Netherlands already takes into consideration all the events because it is calculated in function of average number of vehicles. This makes the concept of emergence hard to use with the Dutch legislation for example.

If the concept of emergence and number of events is to be used in the guidelines, a recommendation to measure LAeq and not calculate it will have to be proposed (similar to the existing WHO guidelines).

There are well established dose-effect relationships between a single event (SEL) and arousals and SEL and awakenings – what needs to be further investigated is the effect of multiple events – this could perhaps be achieved by analysing existing data differently or to consider the cardio vascular effects as the outcome.

Annex 1: Relation between number of events and night time LAeq for some effects (discussed by Martin van den Berg, October 2005)

1. Although it is not often recognized, the equivalent A-weighted sound level (LAeq) "adds" up number of events over the period the LAeq refers to. Implicit in the LAeq formula is that events (number N) are not linearly added but logarithmically, with 10*log (N). This implicit assumption has been verified to be correct for annoyance[i] and for self reported sleep quality[ii]. Relations with other effects like CVD and performance have been found to be statistically significant using LAeq as the metric. The question is if this 10log(N) relation also holds for other effects.

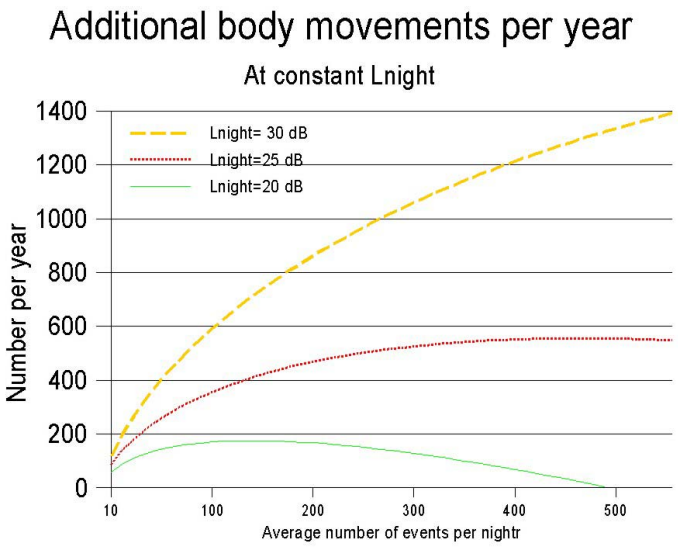
2. There are few studies looking into this problem, and the outcomes usually are not very convincing. A problem is often that for one source high correlations are found between LAeq, N and single event levels, so analysis on beforehand is difficult.
   One possible approach is to use the relations between single events and effects to model the total effect. The trap here is that in order to derive the single event relations it must be assumed that the reactions are statistically independent. This almost certainly not the case, and one only can hope that the interaction doesn't disturb too much the outcome.

3. In this way 2 reasonably reliable dose-effect relations have been derived: for motility and for conscious awakenings from aircraft noise[iii]. Both give the probability of an effect in relation to the SEL or LAmax for a single event above threshold.

4. It has been demonstrated that the combination of an Lnight with a linear or quadratic relationship between a single event and the probability of an effect leads to a maximum in the number of effects at a given LAeq at a single event level (SEL or Lmax) which is just over the threshold (see Annex).

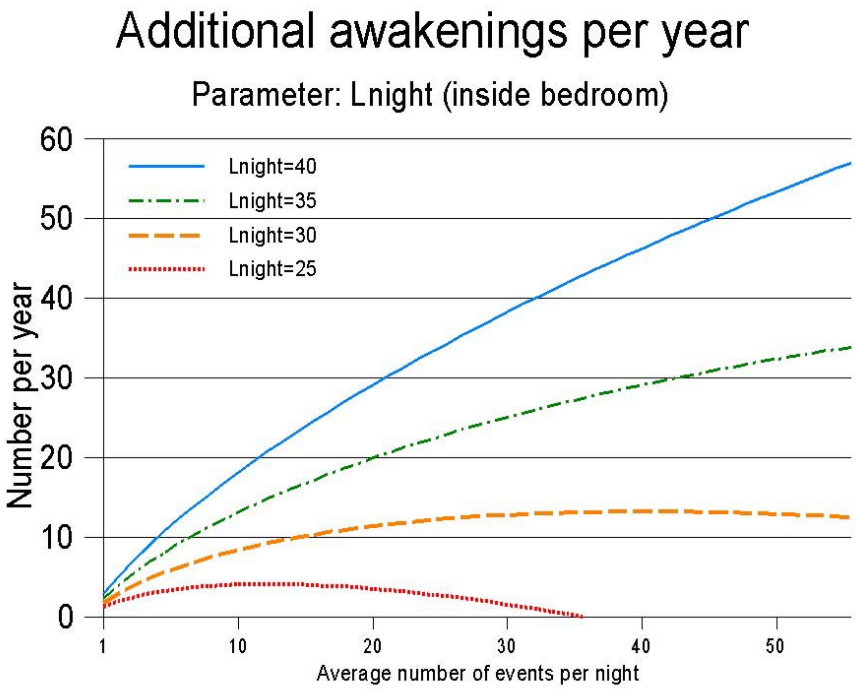
5. The same maximum effect is of course also present when looking at the number of events N, and this can easily be simulated. In figures 1 and 2 this is shown for a realistic combination of inside LAeq values and number of events.

In fig 1 this is done for body movements. The maximum of 500 events per night is chosen because that means an average of 1 per minute; a situation which can be found near main roads. At higher intensities the events start to merge. As expected the maximum in the total effect occurs also with respect to the number of events, and is the maximum dependent on the LAeq level (of course, because at the same SEL).



Figuur 1. Noise induced body movements at constant Lnight inside the bedroom

In order to keep Lnight constant while increasing number of events, the level per event decreases. At Lnight = 20 and 25 a clear maximum can be seen. With this choice of parameters awakenings almost donot occur.



Figuur 2. Noise induced awakenings at constant Lnigt inside the bedroom

This choice of parameters is more representative for airports and railway lines. At Lnight=20 no awakenings occur, and at Lnight=25 a definite maximum occurs. At higher Lnight level this maximum occurs after the event-horizon. As can be seen from figure 1,

no maximum in body movement occurs for these situations. The figures are not combined to avoid confusion.

6. If we concentrate on numbers which are now common for airports and railway lines (up to 50 events per night), close to the airport (high Lnight levels) the effects increase with the number of events. This might mean that when a number noise planes or trains are replaced by more, but quieter planes, the total number of effects in the population increases. However, further away (and that implies a larger part of the population), the inverse occurs.

7. This is a nice exercise, but the question is if this is a realistic image of what occurs in reality. There is a lot of uncertainty if these individual relations may be used to derive population means, and if the underlying assumptions (independence) hold.

8. Assuming this to be realistic, it is difficult to derive additional indicators. For awakenings the recommendation could be to eliminate events below the threshold (SEL=53), but for motility the threshold is SEL=38, which could be produced by aircraft at cruising height.

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i Miedema, Vos, Community reaction to aircraft noise: Time-of-day penalty and tradeoff between levels of overflights, JASA, 107, 2000
ii Miedema, De rol van aantallen gebeurtenissen bij zelfgerapporteerde slaapverstoring, TNO-report 31-12-2004, ref 04-6B-086-64023
iii EU-Commission: Position Paper on Lnight, 2004,
http://europa.eu.int/comm/environment/noise/home.htm

APPENDIX I: relations between L_{night} and instantaneous effects

Statement 1

Let f be a function of *SEL* that gives the expected number of instantaneous effects caused by a single event. With a given L_{night} and a given number of events *N*, the expected number of times that an effect occurs in the night, *n*, is maximal if all events have equal *SEL*, provided that f◦10lg is increasing but negatively accelerated.

Statement 2

If

$$n_{max} = 10^{(Lnight-SEL+70.2)/10} \cdot f(SEL),$$

has a maximum over *sel* and f is the quadratic function $f(SEL) = a\ SEL^2 + b\ SEL + c$, then the maximum occurs irrespective of L_{night} at

$$SEL_0 = 4.34 - A \pm [(A - 4.34)^2 - (c/a) + 8.68A]^{½},$$

where A = b/(2a). (Only with + at the place of ± the value will come in the realistic range of SEL)

Statement 3a

If the shape of the time pattern of the sound level has a block form, then $SEL = L_{Amax} + 10lgT$, where L_{Amax} is the maximum sound level (integrated over 1-s) and *T* is the duration of the noise event in s.

Statement 3b

If the sound level increases with rate *a* (in dB(A)/s) and after time point *t* = 0 decreases with rate –*a*, then $SEL \approx L_{Amax} - 10lga + 9.4$.

Annex 7: Report on the final meeting for consensus building on night noise guidelines

EUROPE

Report on the final consensus building meeting on night noise guidelines

Bonn, 14th December 2006

European Centre for Environment and Health
(Bonn Office)
World Health Organization Regional Office for Europe

Background

The final conference was the conclusion of the ”Night Noise Guidelines (NNGL)” project co-financed by the European Commission and other partners. The aims of this project are to provide expertise and scientific advice to the Commission and its Member States to support the development of future legislation in the area of night noise exposure control and surveillance. The project has followed the WHO methodology for the development of guidelines.

This meeting intended to be a consensus building meeting where the partners, additional experts, national government officers and stakeholders reach agreement on the headlines of the Guideline documents. The discussions focussed on:

1. Methodology to be followed to reach guideline values;
2. The list of health effects and various end points of night time noise exposure;
3. Validity of dose-effect relations and thresholds
4. Weight factors to determine burden of disease
5. Guidelines and recommendations

Summary of the meeting

The meeting was attended by the invited experts and stakeholders representating industry, nongovernmental organizations and policy makers including European Environment Agency and DG Environment (see the list of participants below). As the main document was very comprehensive addressing extensive evidence review from which guidelines were derived, it was not expected to build a complete consensus on all aspects of the document. However, on behalf of transparency and the openness of peer-review process, all participants were requsted to review and comment on the drafted guidelines document thoroughly before the meeting. The participants were presented the summary of the contents of the final draft document at the meeting, and provided final comments. For the most important chapter on the guideline values (Chapter V), all participants read and revised the text line by line collectively, reaching the final consensus. The WHO temporary advisor and secretariat prepared the final version of “Night Noise Guidelines for Europe” as the technical report of the NNGL project.

Summary of major comments on the draft NNGL document

This is a summary of major comments on the final draft, passing by suggestions for obvious improvement of language and order, additional data etc.

General:

- If self-reported sleep disturbance is considered more of a sleep-quality issue than health& well-being, it should be placed in chapter III instead of in Chapter IV

- Delete all sections with “negative”results (II.4, IV.6, IV.7, IV.8) and add short statements to that effect in the introduction.
- More information on sleeping times and shoulder hours, also in chapter V.
- More information of what END really may contribute.
- Copy parts of the Community guidelines 2000 in this report:
    - Section 5, “Noise Management”, first paragraphs with bullet points a-c, is a vital component of any discussion on Community Noise and should, in our view, feature very early on in the draft – these are crucial statements
    - Section 5.7.6 “Cost benefit analysis”, Section 5.7.7 “Review of standard setting”, Section 5.7.8 “Enforcement of noise standards: Low-noise implementation plans”, and Section 5.8 “Conclusions on Noise Management”, pages 86-89 inclusive
    - Chapter 6, “Conclusions and Recommendations”, should be edited into the relevant Chapter in the draft under consideration, again for consistency and clarity
- discuss the final Report at the next suitable meeting of the steering group of the WHO Charter on Transport, Environment and Health working groups and/or the related high-level Ministerial meeting(s).

More specific suggestions to be discussed:

- Page 12, chapter I, 3.2, Event or long-term descriptor

Knowing the pros and cons of long-term descriptors (stable exposure curves, easily calculated <-> poorer relation with awakenings) and event descriptors (instable exposure curves, difficult to evaluate <-> better linked with awakenings) one could mention the compromise of an 1-hour Leq limit value that was successfully introduced in the Swiss regulation for air traffic noise. The discussion of what kind of descriptor should be chosen will be of importance in the practical implementation of the guidelines, say, fixing limit values for the night time

- Page 14, chapter I, 3.4.3, Inside to outside

The arithmetical calculation that lead to a value of 21dB is not really convincing as using this value would considerably underestimate the exposure of 75% of the population. The usually applied value for the inside-to-outside difference is 25dB for close windows, 15dB for tilted windows and 10dB for open windows .

I therefore suggest to mention an value for the inside-to-outside difference of 15dB, covering in this way at least 75% of the population. However, inside limit values should be used as indicator as they correspond best with the noise exposure.

Moreover, chapter 3.4.3 and 3.5 could be merged, as both deal with the same topic.

- Page 21 , chapter II, 1.1 Normal sleep (objective measurements), sentence between table II.1.1 and table II.1.2

...while the number of EEG awakenings including cortical arousal averages 10-12 per night....

The DLR-Study found an average of 21 arousals. I suggest to check the value.

- Page 81, chapter IV, 7.1 Cognition and slow-wave sleep

Referenced literature (Benedict et al., 2004) is missed in chapter V 8. References.

As has been showed from the same group of researchers [Plihal, W., & Born, J. (1999). Effects of early and late nocturnal sleep on priming and spatial memory. Psychophysiology, 36(5), 571-582.], for procedural memory, REM-Sleep is (nearly) as much important as SWS sleep is for declarative memory. Although for general well-being SWS sleep might be a bit more important, one stands on shaky ground in my opinion when concluding that „e.g. aircraft noise before midnight, would be particularly damaging to memory and related cognitive functions“. Procedural memory is also important, e.g. when driving a car. Beside that, all event correlated studies show that noise events do in fact have a greater impact (increasing awakening probability) in the second half of the night than in the first.

- . 101: In the first sencence on chapter IV you say that evidence for the direct effects of night noise on health are given. In my understanding, 'direct' means 'not via degraded sleep'. If I am right, the word 'direct' should be cancelled, because section IV / 2 concerns sleep disturbance.
- p.101: in last paragraph, it would be desirable to stick to the terminology of p.22ff., e.g. 'noise-related Environmental Sleep Disorder' instead of 'environmental noise ..?... disturbance'
- p.102: You write "In the table all effects are summarised ....". Please clarify here if you mean 'effects from day or night noise' or 'effects from night time noise'
- I assume that table V.2.1 refers to day or night noise, whilst table V.2.2 refers exclusively to night-time noise.
- p.102: May I repeat that tables V.2.1 and V.2.2 should contain a reference per each of the rows, indicating the origin of the information.
- Page 102, chapter V, 2, table V.2.1

I suggest to give all indicators for inside as these values correspond best with the level at the ear of the sleeper and thus with the health effects. The calculation from inside to outside should be done according to the building structure and is beyond the scoop of WHO

- The item “zoning” in Ch.V, page 105, should be deleted because it contains incorrect statements about the Quotum Count system.
- p. 106: I would not call the situation above 55 dB 'unacceptable', because this will almost automatically lead politicians to the conclusion that Lnight below 55 dB is 'acceptable'. Instead of qualifying the situation above 55 dB, I would simply describe what happens, as it is done for the lower noise levels.
- In addition, I would include in section 6 the Burden of Disease aspect, for instance as follows: "Health protection from noise should be seen from the Burden-of-Disease aspect: Because Lnight along roads is widely higher than 45 dB but lower than 60 dB, the noise-related Burden of Disease is typically dominated by a large number of inhabitants suffering from sleep disturbance due to road traffic. National health policy should adequately take account of this high Burden of Disease, in comparison to current non-noise Burdens of Disease.'

Conclusions and recommendations

At the final conference in Bonn, Germany, on 14 December 2006, representatives from the working group and stakeholders from industry, government and nongovernmental organizations reviewed the contents of the draft document chapter by chapter, discussed several fundamental issues and reached general agreement on the guideline values and related texts to be presented as conclusions of the final document of the WHO Guidelines for Exposure to Noise at Night as the table below.

Table 1. Effects of noise on health	
$L_{night,outside}$ **up to 30 dB**	Although individual sensitivities and circumstances differ, it appears that up to this level no substantial biological effects are observed.

$L_{night,outside}$ of **30 to 40 dB**	A number of effects are observed to increase: body movements, awakening, self-reported sleep disturbance, arousals. With the intensity of the effect depending on the nature of the source and on the number of events, even in the worst cases the effects seem modest. It cannot be ruled out that vulnerable groups (for example children, the chronically ill and the elderly) are affected to some degree.
$L_{night,outside}$ of **40 to 55 dB**	There is a sharp increase in adverse health effects, and many of the exposed population are now affected and have to adapt their lives to cope with the noise. Vulnerable groups are now severely affected.
$L_{night,outside}$ of **above 55 dB**	The situation is considered increasingly dangerous for public health. Adverse health effects occur frequently, a high percentage of the population is highly annoyed and there is some limited evidence that the cardiovascular system is coming under stress.

After the meeting, WHO Secretariat added the following interpration of the guideline values to assist policy-makers to inptrete the threshold values agreed at the meeting. Considering the uncertainties of exposure-response relations, the need to protect the most vulnerable population, and the impacts on the public health, three stepwise guideline values for night noise levels were proposed: the ultimate target value (NNGL), interim target 1 (IT-1) and interim target 1 (IT-2) for flexible adoption and implementation by the European Union Member States as below.

Table 2. WHO night noise guidelines	
Interim target I (IT-I)	$L_{night,outside}$ = 55 dB
Interim target II (IT-II)	$L_{night,outside}$ = 40 dB
Night noise guideline	$L_{night,outside}$ = 30 dB

In order to prevent any potential adverse health effects in the population due to night-time noise, it is recommended that the population should not be exposed to noise levels greater than Lnight,outside 30 dB during the night when most people are in bed. This is the ultimate target for countries having a high priority in protecting public health from night-time noise.
The interim targets (IT-I and IT-II) are proposed for countries where the above guideline level, Lnight,outside 30 dB, cannot be achieved in a short period. Such countries should aim at reducing the number of the population exposed to levels over IT-I and IT-II as effectively as possible, depending on policy prioritization in the country. It is highly recommended to carry out risk assessment and management activities targeting the exposed population, and aiming at reducing night-time noise from the source to the level below IT-I and IT-II.
Countries can choose to achieve the second interim target (IT-II) after achieving IT-I. However, IT-II can be used to evaluate new projects (highways, railways, airports or new residential areas) even before the achievement of IT-I. IT-II can be used for risk assessment and management of the whole population.
In the long run the night-time noise guideline would be achieved by control measures on the sources along with other approaches.
Especially in the range Lnight,outside from 30 to 55 dB, a closer look may be needed into the precise impact as this may depend much on the exact circumstances. Above 55 dB the

cardiovascular effects become the dominant effect, which is thought to be less dependent on the nature of the noise.
As of 30 March, the executive summary of the final document is accessible on the WHO website at http://www.euro.who.int/noise.

Appendix 1. Minutes of the Final Meeting, Bonn, 14 Dec. 2006

Present:
Dr. Hans Bügli (Agency for the Environment, Forests and Landscape, Switserland),
Prof. Dr. Rüdi Müller-Wenk(Universität St.Gallen),
Anna Bäckman(European Environment Agency),
Martin van den Berg (WHO-temporary advisor),
David Delcampe (European Commission, DG-ENV).
Dr.Leja Dolenc (Institute of public health, Slovenia),
Jeff Gazzard (Greenskies Alliance, UK),
Danny Houthuijs (National Institute for Health and Environment, Netherlands),
Prof. Staffan Hygge(Laboratory of Applied Psychology, Centre for Built Environment ,Sweden),
Stylianos Kephalopoulos (Joint Research Centre, Italy),
Dr Rohko Kim (WHO),
Dr. Christian Maschke(Forschungs- und Beratungsbüro Maschke, BRD),
Dr. Henk Miedema (TNO, Nehterlands),
Dr. Matthias Mather (Deutsche Bahn, BRD),
Prof. Dr. Sonja Nevismalova(Department of Neurology, Czech Republic) ,
Nina Renshaw (Transport & Environment, Brussels),
David Tompkins (European Express Association),

Minutes: Janneke Tanja.

Dr Kim welcomes the participants to the meeting. The aim of this meeting is to discuss the policy-side of the document. More detailed comments can be sent to Martin van den Berg separately.

Over the course of the day four people have presented different aspects of the subject. The sheets of these presentations are available, the minutes will only reflect some general points and the discussion that followed the presentation.

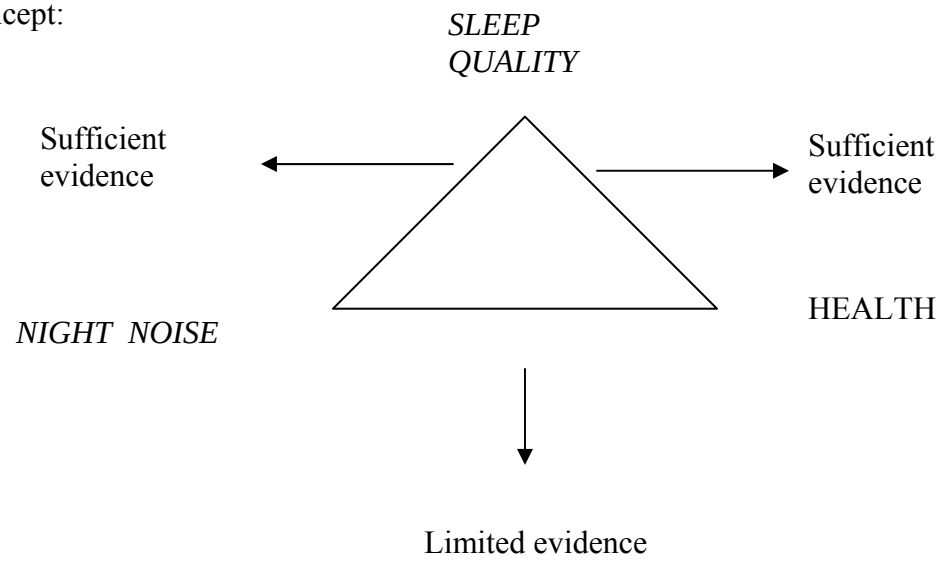
GENERAL OUTLINE PROCESS AND AIMS – M. vd Berg

The NNGL as it stands today is the result of a process that started with a EU grant to the WHO in 2003. During the years that followed may experts from different fields have been involved in it. The final document is related to

- WHO community guidelines on noise (2000)
- Directive on environmental noise (EU)
- Position paper on Lnight (EU)

Right now, the Lnight limits in Europe differ widely, ranging from 45 to 62 dB. The NGGL does not set a (new) limit, but it provides governments with the necessary information to set a proper limit for their country.

Basic concept:



The link between night time noise and health is still difficult to prove. But the fact that there are proven links between night time noise and sleep quality and between sleep quality and health strongly suggests that there is such a link and it should be taken into account.

The report shows the strength of evidence. Sufficient evidence indicates a proven causal connection and / or a plausible (biological) pathway. With limited evidence, a relation is plausible and either:
- observed, but coincidence, bias and distortion cannot be ruled out
- no observed relationship, but indirect evidence is of good quality

Indicators that were used are SEL, Lmax, and Laeq, depending on the purpose. There is at the moment no real alternative for Lnight as a long time indicator.

Discussion

There is some debate on the question whether or not you can use an average value here, when there is a difference in disturbance between people who sleep with open windows and those that don't. Also, can you use calculated values, or have exact measurements.
What you look for in a paper like this is a basic average value that can be used to say something on long-term exposure in general, and as a 'default value' when nothing else is available.

HEALTH AND SLEEP - L. Dolenc

Sleep disturbance can lead to a number of health effects like insomnia, cardiovascular diseases, obesitas and accidents and has an effect on the way a person functions during the day. Sleep patterns are age related, and change over time. An elderly person sleeps less, awakes more often during the night, and also the phases of the sleep change. If a person has insomnia, he will also awake more often during the night. Research using EEG shows that there are more moments of awakening during the night than people report themselves.

Discussion

Is it possible to use the relationship between sleep and heath to prove a relationship between noise and health? What percentage of insomnia-cases is caused by noise for example?
It's difficult to say, because noise is not taken into account in this study. People who can't sleep because of noise usually don't go to a sleep-specialist. They already know why they can't sleep. There is no evidence-based study on the link between noise and sleep-deprivation. That means that if we want to draw conclusions from this research, we need to know if the same effects are found with people who are sleep disturbed because of noise.

NOISE AND HEALTH – D. Houthuijs

Mr Houthuijs gives a short presentation on the reliability and strength of the evidence available for the relationship between nighttime noise and health. Self-reported sleep disturbance for example seems to be subjective, but there is actually a lot of evidence from other sources that support the findings.

Discussion

There is a clear difference in the way epidemiologists and medicals scientists look at evidence, and it surfaces in the discussions after this presentation. Where the medical scientists derive their evidence from looking at the individual, epidemiologists look for the patterns in the population as a whole. This leads to a discussion about what is proven and what not.

New research show a stronger link between hypertension and noise than could be proven from the available evidence. But since these studies are not published yet, they cannot be taken into account here.

There is discussion on the definition of insomnia; are we talking about primary insomnia here, or should it be environmental insomnia? Leja advises not to get too deep into that discussion, since it is an extremely complex issue. Conclusion was that there is indeed evidence for a link between (primary) insomnia and noise.

NOISE AND SLEEP – H. Miedema

Noise is something you can't turn of. Even in your sleep your body will register it and give a response. That response varies from an increased heart rate up to actually waking up. Most effects have been observed in a laboratory-situation, but there is some fieldwork available. The threshold on which the effect become apparent is lower than was expected: between Lmax 32 – 37.

Discussion

There is a relationship between the different observed effects. It seems the whole system sort of wakes up when it's noisy. Question is if these effects are strong enough to cause disorders, but there are plausible biological mechanisms to suggest they are.

Levels were measured indoors, and the threshold is defined as the point were the curve approaches 0%. It still is a number for the average sleeper, so there is some concern that the health effects for disturbed sleepers might be less visible this way.

GUIDELINES AND RECOMMENDATIONS – M. vd Berg

Discussion

The discussion focuses around the main table on page 102 and the text of the section ""Recommendations for health protection" (pg 106). There are some issues raised that will lead to changes in the table. The basis of this guideline however is formed by the text, the tables should reflect and strengthen that. So there is a limit as to what can be changed in the table.

An important part of the discussion focuses on how to deal with the difference in available evidence. For instance, can you give a NO (A)EL for an effect that has only limited evidence, and how does that compare to a NO (A)EL for an effect with sufficient evidence? The result o the discussion is that Martin will split the table in two (a table for the effects with sufficient evidence and a table with limited evidence), to avoid misunderstanding.

It is not always clear what research some classifications are based on. And be careful to draw conclusions for one source base on the figures from another source. You can make some educated guesses, but make sure that that is specified in the text.

Since they would help to understand the material that is presented, some basic information on and reference to DALYs will be made, with a reference to another WHO-paper that explains things more clearly. The column with disability weights will be skipped from the tables.

Agreed on the following qualifications:
Increase average motility – sufficient
Insomnia – limited, a discussion of the methodology will be added
Hypertension, Myocardial infarctions – both points have been extensively discussed. The problem is that we know there is more and stronger evidence on the way. But right now, it just isn't available jet. That means we can only qualify the evidence for both of them as 'limited', although it would be wise to add a threshold-value as well. At the end a level of 50 dB for both was agreed.

As we gain more knowledge, it becomes clear that effects occur at far lower levels than we thought. This should be reflected in the text. It might call for more or earlier measures, and that of course will lead to higher cost; that can be a problem. With the methods described in Chapter I an analysis of the situation may show if cost and benefits are well balanced.

Avoid terms like ‘unacceptable levels’. The reality is that we sometimes do accept those levels in certain situations. But besides that, the tone of this paper needs to be neutral. WHO states the scientifically proven health effects. Accent should therefore be on public health, not on costs or measures. It is a basis others will use to make choices. The text of paragraph V.6 (page 106) was carefully life-edited until the complete satisfaction of all present.

Closing remarks

Kim: Compliments the members of the project group and the original project leader, Mr Bonnefoy. Contributions by all were highly valued by WHO.
Delcampe: States that this work is important to the EU-Commission. WHO is always taken into account. Not automatically copied, but they have to look at it. Lot of legislation will come into review in coming years; this is an important piece.
Tompkins: Operators also needs accurate, unbiased information to base decisions on; congratulations to the project group.
Gazzard: There are also WHO meeting at transport and health; recommends to take information there. Get these guidelines out into the world so they will be used.
Renshaw: NGO’s will make sure it will be heard.
Maschke: still miss some words on max. noise levels.
Vd Berg: there actually is a lot on that, large part of table is in Lmax.
Miedema: Difficult to say something on single events. If you do, make it on number of events
Vd Berg: that looks easier than it is... You’ll end up with a complicated discussion.

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