

Exposure and Effect Indicators of Environmental Noise

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1. Summary

Annoyance and sleep disturbance are generally accepted effects of environmental noise. Traffic noise is the most important source of noise induced annoyance. Long-term derogation of the recovery function of sleep by traffic noise has the potential to increase the sensitivity to daytime noise annoyance as well as the risk of cardiovascular and several other diseases. Noise induced arousal and secretion of cortisol especially in the first half of the night seem to be more sensitive indicators for noise induced detriment of sleep than remembered awakening reactions.

The evidence of cardiovascular risk due to traffic noise is assessed as “limited or sufficient”. In most studies the daytime traffic noise level was related to the increase of cardiovascular risk. Nevertheless there is evidence that traffic noise exposure at night plays a more important role in the pathogenesis of noise stress induced increase of the cardiovascular risk than traffic noise exposure during the day. This was confirmed by the Spandau Health Survey, where a clear distinction was made between day time and night time traffic noise exposure. Among other diseases also chronic bronchitis and bronchial asthma were found to be related to traffic noise. These findings are in agreement with the results of study in children under combined exposure to traffic related noise and air pollution. The relative risks of chronic bronchitis, asthma and neurodermitis of the exposed children cannot be explained by air pollution alone. Therefore long-term traffic noise induced derogation of sleep must have had an adjuvant effect in the pathogenesis of these diseases.

Finally noise induced hearing impairment due to military low altitude flight noise, impulsive noise from toy pistols etc., and long-term overexposure by loud music are summarised. Acoustic limiting values are proposed to avoid inner ear damage. To avoid traffic noise related health defects maximal indoor levels should be $L_{\max} < 40$ dB(A) or in the case of predominant low frequency noise $L_{\max} < 60$ dB(C).

2. Introduction

A short definition of noise is unwanted sound. Accordingly, unwanted or hazardous sound effects are called noise effects. In the following we will consider different effects of environmental noise.

The Federal Immission Protection Act (BImSchG, 1990) specifies adverse environmental noise effects on the general public or in specific neighbourhoods as

- hazards, such as health risks,
- substantial losses, e.g. in property values,
- substantial disturbances.

In principle, any perceptible sound may be experienced as disturbing and rated as causing annoyance (Interdisziplinärer Arbeitskreis 1990). Substantial disturbance, however, is normally caused by louder noise and often assessed as “highly annoying”.

The issue of personal losses in terms of material assets will not be considered in this paper.

The best known noise induced health hazard is the inner ear damage. Since this paper is confined to environmental noise effects, effects of occupational noise will be omitted. The evidence of auditory damage due to military low altitude flight (MLAF) noise will be addressed briefly as well as leisure noise induced inner ear damage..

Acute noise events which do not cause permanent health impairments are considered as non-substantial. However, effects of long-term noise exposure, which do not habituate but increase the long-term risk of physical damage, are assessed as health hazards. Examples for this are long-term sleep disturbances and neuro-endocrine reactions especially during sleep. The latter may lead to increases of the cardiovascular risk and – according to a recent study – to health risks due to neuro-immune detriments.

In the case of road traffic noise it is difficult to distinguish between the effects of noise and air pollution. Therefore we will mention recent studies on the combined effect of noise and air pollution on allergy related diseases in children.

3. Bibliographic review of noise effects

3.1 Annoyance (Guski, 2001)

Annoyance is regarded by experts from 7 nations as the most important adverse effect of environmental noise (Guski et al. 1999). Lindvall and Radford (1973) defined annoyance as “a feeling of displeasure associated with any agent or condition known or believed by an individual or a group to be adversely affecting them”. According to Guski et al (1999), however, disturbances of intended activities (i.e. communication or recreation) which have become conscious and a feeling of powerless discomfort are of similar importance for annoyance.

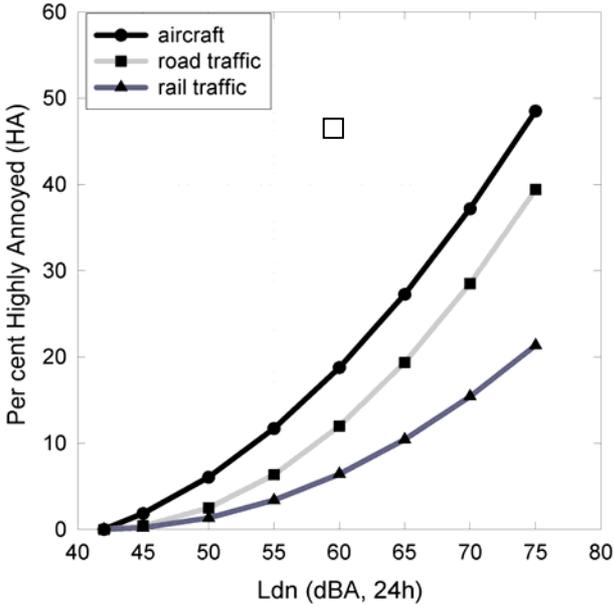


Fig. 1 Dose-response curves according to Miedema & Vos (1998). □: Annoyance due to military low altitude flight noise (Ising et al. 1990).

Schultz (1978) suggested to consider only the percentage of “highly annoyed” person in the population. In a 5-point scale, for example, this would be all persons who stated stages "4" or "5". The percentage of these "highly annoyed persons" (HA%) is then plotted against the noise level and, using root mean square regression, curves are obtained such as those published by Miedema & Vos (1998) in their synopsis of traffic noise annoyance (Fig. 1). From these curves it is obvious that aircraft noise is more annoying than road traffic noise and that rail noise has the lowest effect concerning annoyance.

The result of a representative survey in Germany (Ortscheid and Wende, 2002) are given in Tab 1 for the five predominant noise sources. In this survey a 5-point response scale was used starting with “not at all disturbed and annoyed” up to “extremely disturbed and annoyed”. It is obvious, that traffic noise is the predominant source of annoyance, leading to a total of 29% highly annoyed persons. For this reason and since governments are responsible for it we will concentrate in the following on traffic noise.

The individual annoyance can be predicted to maximal 33% by acoustic parameters (i.e. noise level, frequency and time dependence etc.). Factors which moderate the effect of noise without influencing the noise load are called moderators. During the night, late evening and at the weekend noise is more annoying because quietness is expected. Maschke et al. (1998) argue on the basis of circadian biological rhythms, that humans have to be protected against noise especially during the night and the transient periods between day and night.

Tab. 1 Percentages of highly annoyed persons by different noise sources in the Federal Republic of Germany (Ortscheid and Wende, 2002).

Source	Percentage of “highly annoyed”
Road traffic	17%
Aircraft	7%
Neighbours	6%
Rail	5%
Industry	4%

The individual sensitivity to noise is the most important personal moderator for annoyance reactions due to traffic noise (McKennell, 1963; Finke et al, 1980).

According to Guski (1998) factors with social character are important because these factors apply for whole groups of the population and can be used to reduce noise annoyance:

1.) *General appraisal of a noise source*

Most of the exposed people take into account non acoustic features of noise sources. For instance in Europe rail road is taken to be a social means of transportation connected with lesser danger for health than cars and aeroplanes. The non-acoustic assessment of different noise sources was in several cases more important for the prediction of annoyance than the mean noise level (Finke et al. 1980).

2.) *Confidence in persons responsible for noise and noise abatement*

The general noise annoyance by military flight noise was shown to depend strongly upon the confidence of the exposed people into the persons responsible for noise reduction to the absolute necessary minimum (Borsky, 1961). Similar effects were observed for civil flight noise (Leonard and Borsky, 1973; Tractor Inc. 1970).

3.) *History of noise exposure*

In the neighbourhood of airports in Germany the annoyance increased in the course of several years although the energy equivalent noise level was slightly reduced. The exposed

population counted obviously the increased number of overflights more than the reduction of the overflight noise level.

Finally, an important change of the highly annoyed percentage of the population due to flight noise in Germany will be described and discussed. Table 2 summarised the results of eight representative surveys between 1984 and 1994.

During this time no substantial change of civil flight noise occurred. The protest against military low altitude flight (MLAF) noise intensified till Sept. 1990, when the minimal altitude for flights was raised to 300 m in all areas. Before this time the minimal flight altitude was generally 150 m and 75 m in special areas. - Between 1985 and 1990 a major study on health effects of MLAF noise in Germany-(West) was carried out (Ising et al.1991).

Comparing annoyance in areas with frequent and extremely loud military overflights with civil flight noise, revealed that civil flight noise causes the same percentage of highly annoyed persons as MLAF noise with 17 dB lower energy equivalent sound level (see Fig. 1). Interviews with exposed people revealed that the sudden and extremely intensive noise of fast and low direct overflights were esteemed as unbearable since they caused shock reactions and inner ear pain in adults and children and in a number of cases convulsions followed by long and intensive crying in babies. After shifting the minimal altitude for all flights to 300 m in Sept. 1990 none of the above described symptoms was reported and the sensitised annoyance reactions of the population began to normalise.

Tab. 2 Population percentage of “highly annoyed” by flight noise between 1984 and 1994

Year	Percentage of “highly annoyed” persons
1984	11%
1986	17%
1987	14%
1989	20%
1991	17%
1992	17%
1993	14%
1994	10%

Statements on annoyance can only reflect the conscious part of human experience. We may be able to gather information on the quality of sleep and annoyance experienced during the night by those afflicted, but the validity of such information is somewhat doubtful since these data is at present easier to correlate with daytime noise levels than with those at night (cf. Scharnberg et al. 1982). However, long-term derogation of the recovery function of sleep by traffic noise has the potential to increase the sensitivity to daytime noise and the reported annoyance

3.2 Sleep disturbance

The most obvious effect of noise exposure during the night is the disturbance of sleep with possible detriment of efficiency during the day and even long-term health impairment.

Intermittent noise causes a fragmented sleep progress (e.g. aircraft flight noise, road traffic with separated vehicles) or, in cases of quasi-continuous noise (e.g. highway traffic noise), as shallow sleep. Both kinds of noise have the overall result of shortening the times of deep sleep (phases 3 and 4) and the REM phases. The activation instigated by noise may lead to the

process of awakening. Effect thresholds for immediate reactions can be found in the following table. The information content of noise as well as the sound level is important for the person asleep. The alarm function of the sense of hearing may cause awakening even when the noise is very soft if the noise contains information important for the sleeping person. Remarkably high noise levels of 90 dB(A) and more may be slept through, especially by children.

Arousals – short-term noise induced activation while asleep

Noise effects can cause functional changes. This process is commonly referred to as activation. Besides the concept of activation, the expression "arousal" has established itself. The branch of medicine pertaining to sleep designates an arousal as a narrowly confined chronological change in condition, raising the organism from a lower level of excitation to a higher one. Under physiological conditions, arousals represent protective reflexes, wherein distinctions have to be made between vegetative, motorial and EEG arousals. Motorial arousals occur by changing position, coughing, muscular spasm, and are usually accompanied by EEG or vegetative arousals.

EEG arousals are connected with reduced theta and delta wave stages as well as an induction of alpha and beta wave stages.

Various studies (e.g. surveyed by Maschke in 1997) have shown that (traffic) noise can induce vegetative, motorial and EEG arousal. The data collected for EEG arousals are comparatively large. However, the majority of these studies were conducted before 1980 and are mainly concerned with the awakening reaction (awake phase) instigated by noise, in terms of the definition by Rechtschaffen & Kales which embodies an arousal period of at least 30 seconds. The American Sleep Disorder Association (ASDA) requires an arousal episode of 3 – 30 seconds. Other working groups also include micro-arousal (1–3 seconds) in the definition of an EEG arousal. The consequence is that results obtained from medical sleep research can only be compared with difficulty to the results of noise effects research.

EEG arousal always represent an interruption of the progression of sleep. Should this interruption last for a period of approx. 1 – 4 minutes (Griefahn et al. 1976) the EEG arousal passes into conscious awakening. The time depends on the sleep phase preceding arousal (Hecht 1992). Conscious awakening can be viewed as a cognitive arousal in the sense of intruding thoughts. It may be combined with a delayed re-onset of sleep and in this form represents a serious disturbance of the progression of sleep.

Health significance of arousals

Arousals serve the maintenance of homeostasis as an integrated whole. Their assignment is to prevent life-threatening influences or events by the activation of compensation mechanisms. Frequent occurrence of arousals lead to a deformation of circadian rhythms. Deformation is revealed by a fragmented progression of sleep. Sympathetic tonus is increased as a result of the fragmentation of sleep during the night or of the multiple arousal occurrence. This leads to impaired sleep quality and to decreased performance capacity, drowsiness and tiredness during the day.

Viewed collectively, long-term disturbance of circadian rhythms has a deteriorating effect on clinical symptoms [Hanly et al. 1989, Zuberi-Khokhar 1996, Biberdorf et al. 1993, Bonnett and Arand 1995, Mercia and Gaillard 1991]. A disturbance is also indicated by multiple awakening reactions.

For the above reasons, phases awake induced by noise have to be assessed as abnormal and in the long-term as a health risk. On the other hand, serious disturbance of the progress of physiological functions already becomes apparent below the awakening threshold.

Tab. 3 Thresholds for immediate reactions to traffic noise (Source Maschke et al. 1997)

Parameter	Continuous noises	Intermittent noises
overall duration of sleep	shortened from $L_{Aeq} = 45$ dB(A)	shortened at $L_{Amax} = 45$ dB(A) (50 episodes)
latent sleep phases	sleep onset latency prolonged from $L_{Aeq} = 45$ dB(A), deep sleep latency prolonged from $L_{Aeq} = 36$ dB(A), tendency to prolongation of dreaming sleep latency	no data on sleep onset latency, deep sleep latency at $L_{Amax} = 45$ dB(A) (50 episodes) prolonged, tendency to shortening of dreaming sleep latency
arousal reactions and change of sleep phase		induced from $L_{Amax} = 45$ dB(A)
awakening reactions	increased above $L_{Aeq} = 60$ dB(A)	induced from $L_{Amax} = 45$ dB(A)
duration of phases awake	prolonged above $L_{Aeq} = 66$ dB(A)	prolonged from $L_{Amax} = 65$ dB(A) (15 episodes)
duration of light sleep	prolonged above $L_{Aeq} = 66$ dB(A)	prolonged at $L_{Amax} = 75$ dB(A) (16 episodes)
duration of deep sleep	shortened from $L_{Aeq} = 36$ dB(A)	shortened at $L_{Amax} = 45$ dB(A) (50 episodes)
duration of REM-Schlaf	shortened above $L_{Aeq} = 36$ dB(A)	shortened at $L_{Amax} = 55$ dB(A) (50 episodes)
irregularity of pulse		frequency may be increased if $L_{Amax} > 50$ dB(A)
heart rate		increased from modulation depth of 7 dB(A)
body movements	more frequent above $L_{Aeq} = 35$ dB(A)	more frequent induced at $L_{Amax} = 45$ dB(A)
subjective quality of sleep and mood in the morning	poorer from $L_{Aeq} = 36$ dB(A)	already poorer by about 25% at $L_{Amax} = 50$ dB(A) (64 episodes)
memory of awakening		increased from $L_{Amax} = 55$ dB(A), increases further with the value of L_{Amax} and number of episodes
performance	poorer above $L_{Aeq} = 45$ dB(A)	poorer at $L_{Amax} = 45$ dB(A) (16 episodes)

Induced: reaction in a timeframe after the noise episode (the timeframe varies between 30 and 90 seconds in the individual investigations)

In earlier noise effect research the awakening reaction was taken to be the only important health related reaction. Findings derived from arousal and stress hormone research make possible a new access to the noise induced nightly health risk.

Noise exposure during sleep which causes frequent arousal leads to decreased performance capacity, drowsiness and tiredness during the day. Long-term disturbances of the described circadian rhythms have a deteriorating effect on health, even when noise induced awakenings are avoided.

Additionally, the deep sleep phases in the first part of the night are normally associated with a minimum of cortisol and a maximum of growth hormone concentrations. These circadian rhythms of sleep and neuroendocrine regulation are necessary for the physical as well as for the psychic recovery of the sleeper. Endocrine effects of noise will be discussed next.

3.4 Neuro-endocrine dysregulation

Sudden, unexpected noise events set off a series of stress reactions with an initial startle reflex, followed by an orienting reaction directed towards locating and validating the sensory stimulus. This reaction habituates relatively fast. The sequence of the stress reactions under concern is genetically programmed and provide biologically vital protection strategies without adverse effects on health as long as they occur seldom.

Henry and Stephens (1977) developed a psycho-physiological stress model – then further developed by Henry (1992)- from which they derived two different physiological reaction types with regard to the mental capacity of coping with stress situations. If a stress situation is controlled with success, the primary stress hormone to be released is norepinephrine (noradrenaline). If, however, constant efforts are needed to gain or keep control, the predominant stress hormone is adrenaline. A loss of control may lead to a defeat reaction which is characterized by an increase in ACTH and cortisol concentrations (Figure2).

In waking persons under noise exposure also three different types of psycho-physiological stress responses can be observed (Ising et al. 2001):

- (1) Exposure to familiar noise such as occupational noise, but with levels above 90 dB(A), predominantly induces a release of norepinephrine from synapses of the sympathetic nervous system, partly entering the blood circulation.
- (2) Exposures to unfamiliar noise, and more so if signaling a threat or danger, the primary response is an orienting reaction. If a noise is subjectively associated with fear or anxiety, sympathetic activation evokes a reaction pattern which is directed towards elimination of the source of threat, i.e. the fight-flight reaction. The primary hormone in this case is adrenaline, released from the adrenal medulla. There are indications of an increased adrenaline effect of continued neural alpha-stimulation for hours after plasma concentration has subsided to normal (Majewski et al. 1981, Blankenstijn et al. 1988).
- (3) Exposures to extremely high-intensity noise, especially to unexpected noise with fast level rises and maximal level above 120 dB(A), are usually followed by a defeat reaction as a result of subjective lack of control of such stress situations. The primary hormone in this case is cortisol, released from the adrenal cortex.

These alternatives of noise induced stress reactions were derived from the results, which are listed in Table 4. In the majority of the studies, noise exposures were experimentally modified under real life conditions, and respective acute stress hormone reactions assessed.

(1) In brewery workers exposed to noise of different intensities under plain work conditions (Ising et al. 1980b), an increase in norepinephrine excretion was assessed after experimentally increasing the noise exposure levels by not using ear protection. Comparing norepinephrine excretion values in three groups of long-term, differently noise exposed persons (Ising et Braun 2000) showed persistent norepinephrine increases in the case of work noise exposures above 95 dB(A) lasting for years.

(2) Exposures to unfamiliar noise as occur at motorcycle races (Ising et al. 1980a) with mean

levels $L_m = 85$ dB(A) and maximum levels $L_{max} = 100$ dB(A), as well as exposure to low-frequency white noise (Ising 1983a, Ising et al. 1982) at $L_m = 50-60$ dB(A), induced significant increases in serum adrenaline.

(3) Exposures to experimental MLAF noise of $L_{max} = 125$ dB(A) resulted in significant increases in cortisol serum concentrations compared to noise exposures with levels of 20 dB less (Ising et al. 1990). Similar results were observed in animal experiments (Ising et al. 1991). Following a single simulated MLAF noise event at a maximum level of 105 dB(A) and a duration of 3 seconds, ACTH concentration was significantly increased, and in 48% of the probands the values amounted to two times the standard concentration (Marth et al. 1988).

Activities like concentrated work or verbal communication disturbed by noise of relatively low sound levels may still lead to stress reactions. Sophisticated manual tasks, carried out under play-back exposure to road traffic noise of 75 dB(A), induced significant increases in norepinephrine (Ising et al. 1982). During verbal communications disturbed by road traffic noise, a mean level of 60 dB(A) was sufficient for effecting a significant increase in norepinephrine (Ising et al. 1983b, Ising et Braun 2000). During sleep, acute and chronic stress hormone increases are already detected at considerably lower noise levels (Table 5).

Neuro-endocrine effects during sleep

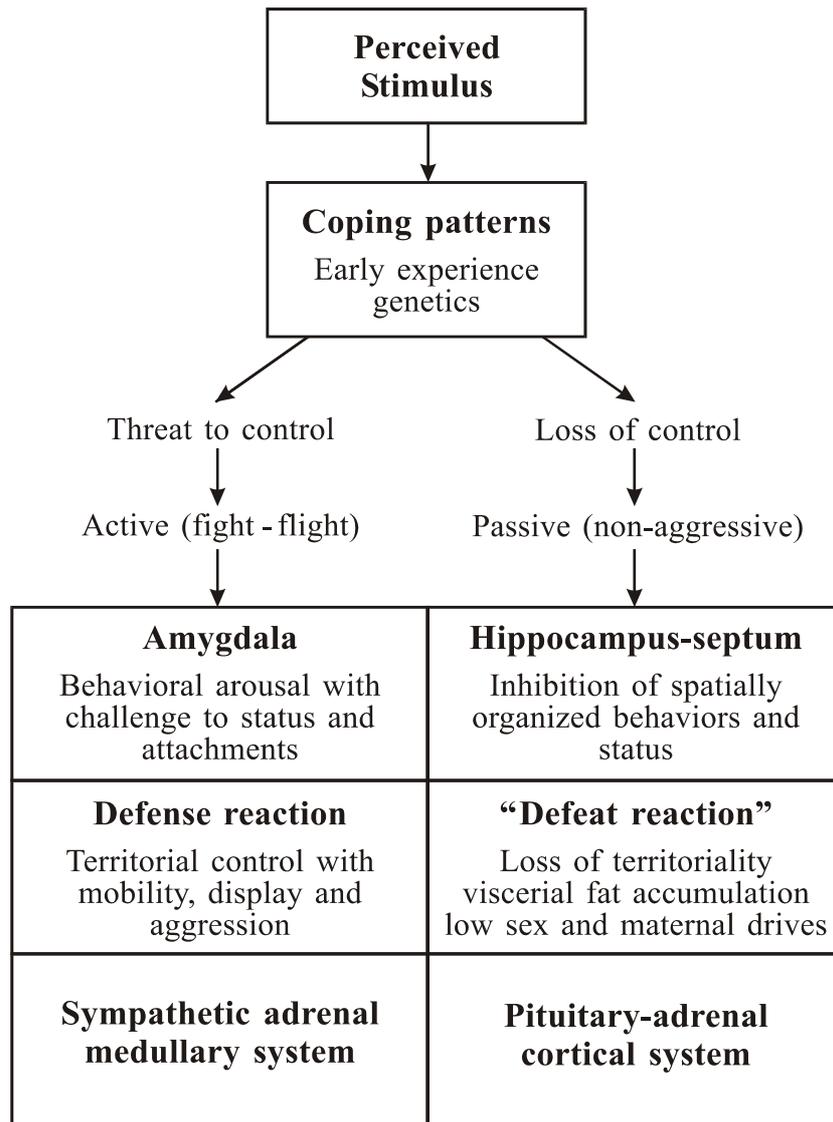
The following is focussed on why the sleeping human organism responds to low noise levels with considerable cortisol increases. Our ear is on the alert day and night serving as our most important sensory warning system. This is a reason why environmental sounds are perceived and processed whether a person is awake or asleep. In sound processing, specific subcortical areas of the CNS play an essential part in increases of cortisol concentration during sleep at low sound levels. In explanation of these processes Spreng (2001) gives the following details:

“With regard to sensory activation processes of the CNS one has to differentiate between two systems which are primarily controlled by subcortical centres, but differ in their major functions. These are the ascending reticular activating system (ARAS) and the vegetative nervous system (VNS).

The ascending reticular activating system (ARAS) activates the cerebral cortex, thereby regulating among others the central rhythm of the sleep-phases and in addition controlling arousal. The leading element in this system is the subcortical formatio reticularis, a network of neurons reaching from the brainstem to the midbrain, containing neurons essential for circulatory regulation. The reticular formation is instigated by sensory stimuli on the one hand, and by the limbic system or “emotional brain” on the other. Therefore, internal as well as external stimuli can influence the waking and the sleeping organism.

The lateral core region of the amygdaloid body constitutes a regulatory component of the auditory system. Numerous findings of recent research suggest that the amygdala is a cerebral structure which plays an important role in emotional learning processes especially with regard to coping with anxiety. This central anxiety-related system is of a remarkable plasticity, or learning capacity in judging adverse noise events as negative noise stimuli.

The amygdaloid body closes the causal chain between over-excitations of the central auditory system with ensuing over-activations of the hypothalamic-pituitary-adrenal axis and the effects of extrahypothalamic hormone excretion. This might explain why quite often noise stimuli below the critical noise limit values of auditory damage, i.e. noise-induced hearing loss, and those below waking level may both have adverse effects on health" (Spreng, 2001).



Control	Striving	Loss of control
Noradrenaline ↑	Adrenaline ↑	ACTH ↑
Corticosterone ↔	Prolactin ↑	Corticosterone ↑
Gonadotrophins ↑	β-Endorphins ↑	Catecholamines ↔
Testosterone ↑	Renin ↑	Gonadotrophins ↓
Oxytocin ↑	Fatty acids ↑	Testosterone ↓
	Glycogenolysis ↑	Pepsin ↑

Figure 2. Psycho-physiological stress model according to Henry [1992].

“Defense reaction is activated when organism is challenged but remains in control. With loss of control there is activation of the hypothalamo-pituitary-adrenal axis, and the gonadotropic species preservative system shuts down. Visceral fat accumulates with a Cushingoid distribution, and there is a shift from active defense to a passive non-aggressive coping style.” (Henry 1992).

During undisturbed sleep the cortisol concentration declines to a minimum, then rises to maximum values at around wake-up time in the morning. The cortisol nadir coincides with the onset of slow-wave sleep phases.

Tab. 4 Investigations of the Institute for Water, Soil and Air Hygiene for testing stress hormone increases. + significant increase, ø not tested.

Reference	type (duration) of noise	acute/ chronic noise	Leq (L _{max})	per- sons	measure- ments	adren- aline	norad- renaline	free corti- sol
			[dB(A)]	n	n			
Ising et al. (1990)	MLAF (5s)	acute	(105/125)	12	120	=	=	+
Ising et al. (1980a)	motor cycle races (8h)	acute	85 (100)	57	114	+	=	ø
Ising (1983a), Ising et al. (1982)	low frequency sound (8h)	acute	50-60	18	54	+	=	ø
Ising et al. (1980b)	work noise (years/8h)	acute & chronic	71-102	47	77	=	+	ø
Ising et al. (1982)	road traffic (8h)	acute	75 (90)	18	54	=	+	ø
Ising et al. (1983b)	road traffic (8h)	acute	60 (75)	43	82	=	+	ø
*) outdoor noise level								

During the first half of the night, slow-wave sleep phases are associated with a minimum cortisol release. This circadian neuro-endocrinological regulation pattern is characteristic of undisturbed sleep (Born & Fehm, 2000). If this specific pattern loses balance through nocturnal noise disturbances, there will be a decline in the recreative function of sleep which is only achieved at minimum cortisol levels.

When assessing cortisol excretion, a method ought to be used which guarantees the specific assessment of free cortisol which constitutes the biologically effective component of total cortisol.

Epidemiological studies

Table 5 gives a survey of recent epidemiological research on noise-induced stress hormone increases. Following exposures to MLAF noise at maximum outdoor levels up to 125 dB(A) lasting for years, the test persons showed significantly increased 24-hour excretions of cortisol and metanephrine (a catecholamine metabolite) compared to persons exposed to modest or no MLAF noise (Schulte et al. 1993).

In a cross-sectional study (Evans et al. 1995) significant increases in adrenaline and norepinephrine excretion were found in a highly exposed group of children.

Additionally, in a prospective intervention study with children (Evans et al. 1998), significant increases in adrenaline and norepinephrine excretion were found after a new airport had been opened. Total cortisol only increased tendentially. Free cortisol was not assessed.

In the studies by Kastka et al. (1998) and by Stansfield et al. (2001) both the exposure and the method of stress hormone assessment differed significantly from the other studies. There was flight noise exposure only during the day time and urine samples were collected during the day (Kastka et al.1998) or saliva samples in the morning (Stansfield et al. 2001). However, according to the noise stress model (Ising et al. 1990), cortisol increases can be expected in awake persons only, when the maximal sound level is near the threshold of pain. In sleeping persons cortisol secretions occur, due to the signal detection capacity of the amygdala [Spreng, 2000 ; 2001], at much lower levels. Therefore, the negative results of these two studies can be neglected. From the remaining four studies only one (Haines et al. 2001) revealed no association of flight noise exposure and stress hormone increases.

The four road traffic noise studies with stress hormone assessment from night time urine samples showed significant relationships between long-term noise exposure and stress hormone increases. One study with saliva cortisol as endpoint showed a tendency of association. Of special interest is the result of the study, where cortisol was measured in a morning blood sample (Babisch & Ising, 1986).

Tab. 5 Epidemiological studies on the association between traffic noise and stress hormones

A Flight Noise

Schulte 1993	< 30/ > 60 day (Lmax ≤ 125*)	60 adults	∅ (+)**	∅ (+)**	+	Urine: 24h CS
Evans 1995	59/68 day + night	135 children	+	+	∅ ***	Urine: night CS
Evans 1998	53/62 day + night	217 children	+	+	∅ ***	Urine: night CO +I
Kastka 1998	None/(Lmax: 64 –69) day	112 adults	∅	∅	=	Urine : day CS
Stansfield 2001	< 57 / >66 day	238 children	∅	∅	=	Saliva: morning CS
Heines 2001	<57/ >63 day + night	204 children	=	=	=	Urine: night CS

B Road Traffic Noise

First Author/ Year	Leq dB(A) (Lmax) time	Persons/ Measurements	Adrena- line	Noradrna- lin	Cortisol free	Sample/ Study Type
Babisch 1986	< 56/ >60 day	2415 males	∅	∅	-	Blood: morning CS
Babisch 1996	45-75 night	195 females	=	+	∅	Urine: night CS
Braun 1999	< 45/ >52 night	44 adults (102)	=	+	+	Urine: night CS
Poll 2001	50/65 day	28 adults	∅	∅	(+)	Saliva: day course CS
Evans 2001	< 50/ > 60 day + night	115 children	=	=	+	Urine: night CS
Ising 2002	< 50 / >60 night (30-54/ 55-78 dB (C) indoor)	56 children	=	=	+	Urine: first half of night CS

*: Military low altitude flights; **: Metanephrine (a metabolite of catecholamines) increased; ***: no free but total cortisol measured. CS: cross-sectional; CO: cohort; I: intervention.

In the group exposed to $L_{eq} > 60$ dB(A) at day time and >50 dB(A) during the night free cortisol in a morning blood sample was significantly decreased. This result corresponds to a tendency of cortisol decrease in urine collected from 1 hour after midnight till morning (Ising, H. & Ising, M. 2002). In this study increases of cortisol were observed only in the first half of the night, when - due to the circadian rhythm - cortisol has its minimum. The quotient of the cortisol excretion in the first half of the night divided by that in the second half proved to be the most suitable parameter for detection of noise induced stress reactions. To avoid cortisol dysregulation, maximal indoor levels should be $L_{max} < 40$ dB(A) or in the case of predominant low frequency noise $L_{max} < 60$ dB(C). Long-term distortion of the normal cortisol rhythm leads to chronic deterioration of the recovery function of sleep (Born & Fehm, 2000).

In combination with other stressors this may lead to long-term stress reaction with chronic increases of cortisol even above the normal range. An example for this seems to be the field experiment of Harder et al. (1999), who determined free cortisol excretion after three test nights without noise and 37 nights with simulated flight noise by means of loudspeakers in the test persons' bedrooms. Only for the second and third night with flight noise exposure, an acute increase of mean group values in cortisol excretion was found. The mean cortisol values then returned to standard, except for a 7-day fluctuation overlapping the circadian cortisol rhythm. The key results were increases in cortisol excretions which significantly exceeded standard values during the final two weeks of nocturnal flight noise exposure (Maschke et al. 2002). This study shows that long-term nocturnal noise exposures may in vulnerable individuals lead to persistent cortisol values which even exceed the normal range.

To sum up the results: From the presented research it can be concluded that long-term environmental noise exposure lasting for years, may in a number of exposed people lead to chronic dysregulations in their endocrine system.

3.5 Cardiovascular risk (Babisch, 2001)

Cardiovascular disorders have been in the focus of studies on extra-aural noise effects, on the one hand from the results of laboratory experimental research on nonspecific stress reactions to noise, and on the other hand from the considerable significance of these disorders in public health legislation (Doll, 1992, Statistisches Bundesamt, 1998, WHO Regional Office Europe, 1999). Fig. 3 reflects the reaction scheme on which epidemiological questions and test hypotheses in noise research is based (Babisch, 2000a, Babisch et al., 2001). Noise directly or indirectly activates the sympathetic and endocrine systems including cortical and sub-cortical brain structures. Dysregulations in metabolic equilibrium cause chronic changes in values of biological risk factors, which increase the risk of cardiovascular diseases (Interdisziplinärer Arbeitskreis für Lärmwirkungsfragen beim Umweltbundesamt, 1990). In the causal chain one has to differentiate between stress indicators (for example stress hormones), risk factors (hypertension, blood lipids), and manifest diseases (like myocardial infarction). Their clinical relevance increases in the order mentioned above:

- *Stress indicators* by themselves are not of direct clinical relevance, but serve in assessing effect mechanisms. As short-term reacting parameters they appear first in the reaction-effect chain.
- *Risk factors* are considered as directly relevant to health. Because they act in most cases as continuous variables, even minor non-pathological changes may be included in the evaluation of effect-associated correlations. Nonetheless, for quantitative risk

estimation, data from external sources have to be taken into account ("What effect does an x-percent increase in cholesterol have on the risk of myocardial infarction?").

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

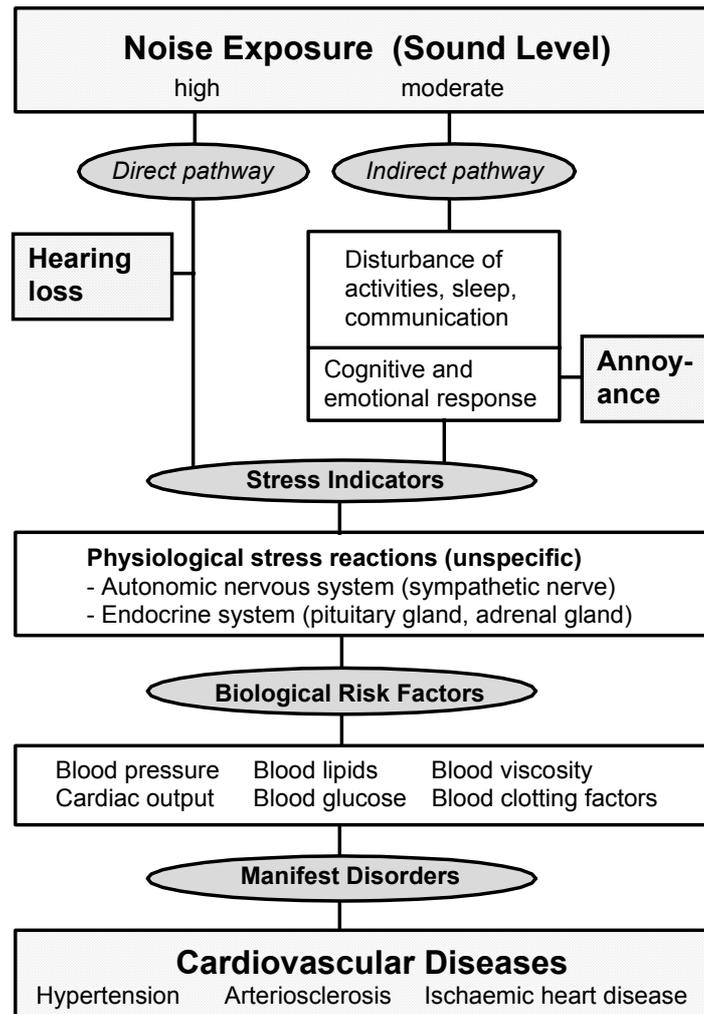


Fig. 3 Epidemiological view of noise effects model

Risk and risk assessment

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

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

From the evaluated results, a risk assessment is carried out ("risk characterization") (Patton, 1993). Examination of the scientific evidence of available data requires a critical discussion of

the validity of the studies (influence of chance and systematic errors (bias)), their transparency and the complete literature used (Neus and Boikat, 2000, WHO Regional Office Europe, 2000). A quantitative effect estimator has to be calculated (e.g. regression coefficient, relative risks) which then – with reference to the population ("attributable proportion") - serves as key information for any follow-up "risk management" (Jasanoff, 1993, Walter, 1998).

In public discussions of environment and health hazards the application of the statistical term "risk" often proves to be problematic. In colloquial language it is understood as a synonym for "danger", thus making competent reasoning rather difficult (Fülgraff, 1992). While the term "danger" is used in defining a qualitative relationship between exposure factor and health, the term "risk" is used in the same relationship for a quantitative assessment (Zeger, 1991).

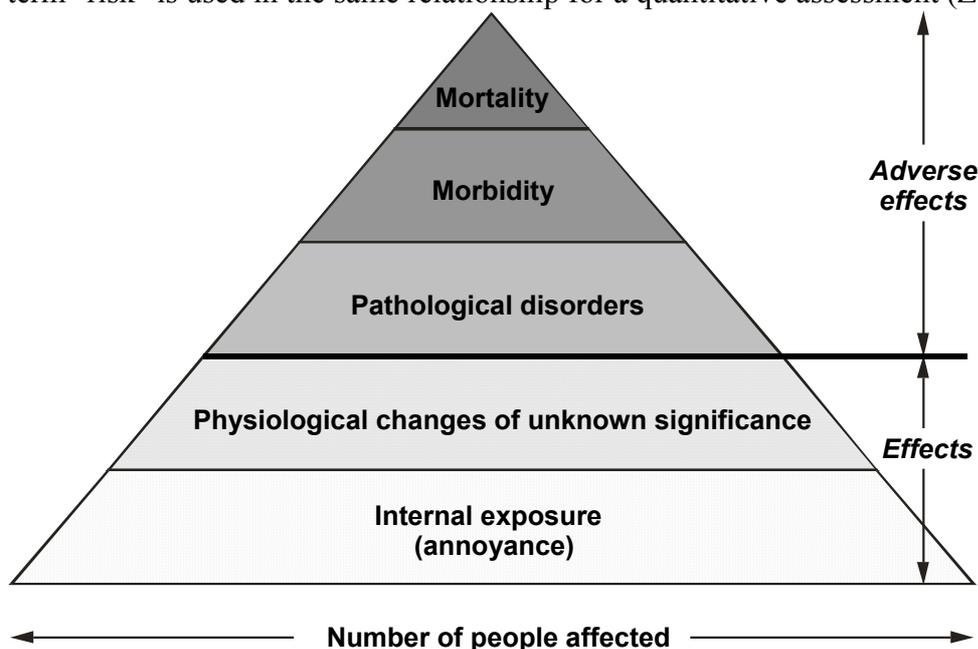


Fig. 4 Grading of noise effects on health

For risk assessments of environmental factors and the setting of environmental standards, the severity and prevalence of health outcomes play an essential part. This is outlined in Figure 4 and was taken from the "Handbuch der Umweltmedizin" (Wichmann and Ihme, 1999), and adapted for the issue of noise effects (Babisch, 2002). Since the diagram was originally designed for chemical exposures, the lowest effect grade ("internal exposure") was substituted by "annoyance". Unlike chemical noxae, noise as such is not immediately measurable in an organism, only its effects are measurable. This is one reason why toxicological test methods can only partially be applied to noises. The WHO defines adverse health effects as follows (WHO, 1994):

"Change in morphology, physiology, growth, development of lifespan of an organism, which results in impairment of the functional capacity to compensate for additional stress, or increase in susceptibility to the harmful effect of other environmental influences."

Traffic noise studies

In comparison to other environmental disciplines, there are relatively few epidemiological noise studies from the sphere of the environment. Misclassification of exposure, no or incomplete control of confounding factors and lack of dose-response relationships are

problems in some of the traffic noise studies (Babisch, 2000b). This does not necessarily mean of course that provisional conclusions cannot be drawn from the available data (Rose, 1992, Scheuplein, 1993). Fairly consistent results of investigations can be recognized, particularly where ischaemic heart disease is involved as the final health outcome, independently of considerations of significance. The findings have been summarized in graphs and tables as shown in Fig. 5 (Babisch, 2000a). The entries are relative risks with 95% confidence intervals. Relative risks were calculated as risk ratios or odds ratios. The dark-shaded beams in the diagram refer to studies where the noise exposure was determined objectively (noise levels), the light-shaded beams where it was determined subjectively (annoyance). Road traffic and aircraft noise studies are viewed here together. No corresponding results are available for rail traffic studies.

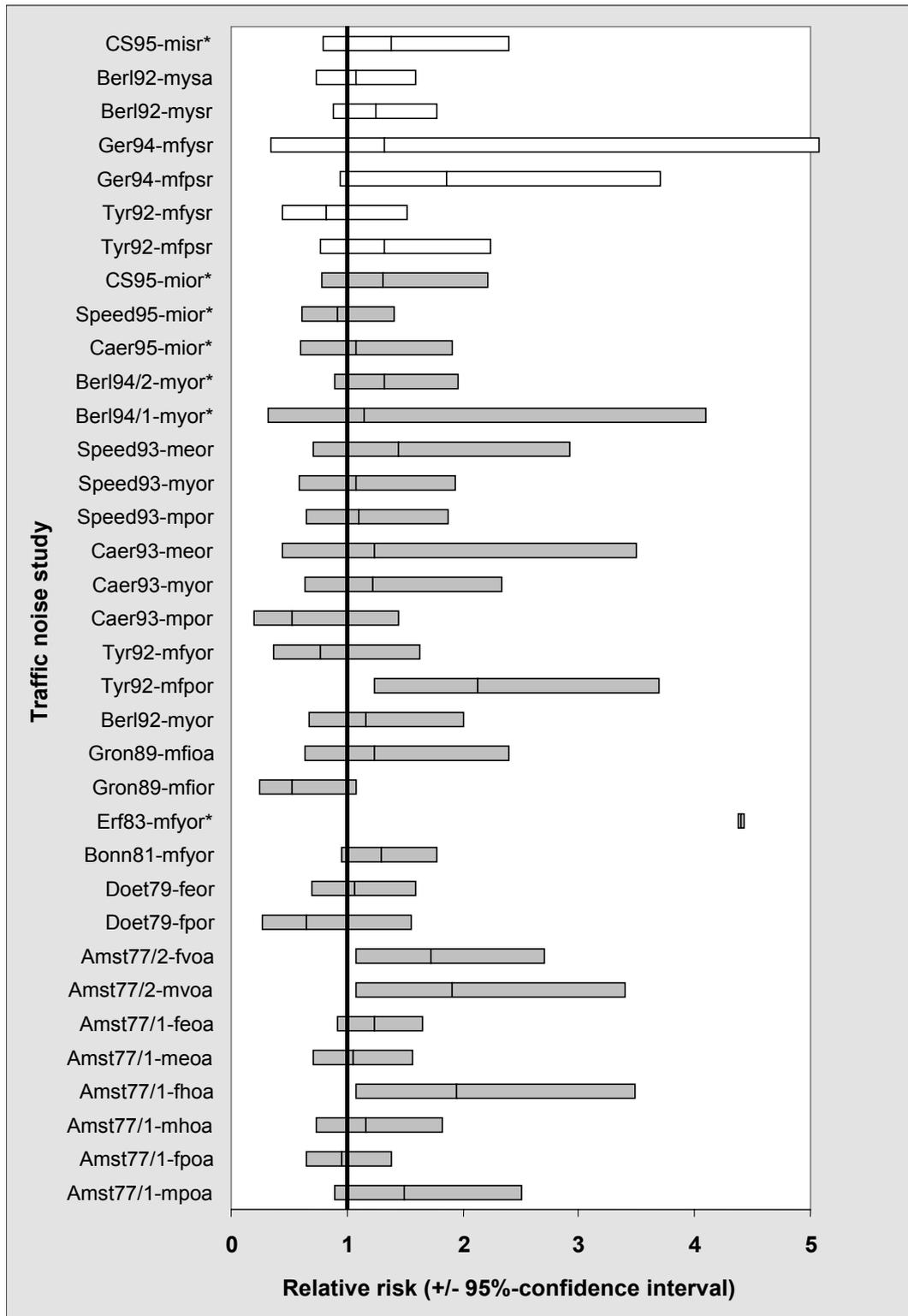
(Note: If different subgroups of the population were taken into account (males/females) or different final health outcomes observed, specific studies would appear several times in the illustration. When a series of studies from a particular area under investigation were published in the same year, it is indicated by a serial number behind the year. For example, Amst77/1-mpoa means Amsterdam, 1977, Study 1, males, angina pectoris, objective exposure = sound level, aircraft noise).

Most of the investigations are related to road traffic noise, only a few to aircraft noise. The few flight-noise studies offer no information on dose-response correlation. When comparing extreme groups in traffic noise exposure, a shift of the relative risk estimators to values above 1 can be mainly discerned in strongly exposed groups of people (range approx. 1.1 to 1.5). Using careful and critical evaluation of the findings, the results were so interpreted that the relative risk of ischaemic heart disease to persons living in areas subject to high traffic noise exposure may be slightly higher (Babisch, 2000a). Our own work, enabling assessment of the effects in terms of graded exposure categories (5 dB(A) classes) indicate an averaged daytime level of 65–70 dB(A) as a possible threshold for noise effects where traffic noise affects health (Babisch and Ising, 1992).

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

With regard to aircraft noise and the night-flight problems in the vicinity of busy airports, at present no other alternative exists than the approximate transfer of the quantitative risk observations derived from road traffic noise studies to aircraft noise. However, since aircraft noise acts on all sides of a building, i.e. different to road traffic noise, the suspicion exists that the effects induced by aircraft noise could be greater than those induced by road traffic. This may be due to of the lack of evasive possibilities within the home, and the greater annoyance reactions to aircraft noise, which are usually expressed in social surveys (Ortscheid and Wende, 2002). Such effects have yet to be examined in future studies.

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



Captions: sex: f female, m male; noise measurement: o objective (sound level), dark-shaded beam; s subjective (annoyance), light-shaded beam; type of noise: a aircraft noise; r road traffic noise; ischemic heart disease: e ECG-ischemic symbols, h heart complaints, i ischemic heart disease, p Angina pectoris, v cardiovascular complaints in general, y heart attack; type of study: prevalence studies; * = cohort or case-control studies

Fig. 5 Results of epidemiological studies on the association between traffic noise and ischaemic heart disease

Evidence of the findings

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

- *Sufficient evidence* is given if a positive relationship is observed between exposure to the agent and the health outcome (cancer), in studies in which chance, bias and confounding can be ruled out with reasonable confidence.
- *Limited evidence* is given if a positive association is observed between exposure to the agent and the health outcome (cancer), for which a causal interpretation is considered by a Working Group (experts) to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence.
- *Inadequate evidence* is given if the available studies are insufficient in quality, consistency or statistical power to permit a conclusion regarding the presence or absence of a causal association.

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

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

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

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

- *“Inconclusive” evidence for a causal link between noise exposure and hypertension.*
- *“Sufficient” evidence for a causal association between noise exposure and ischaemic heart disease.*

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

- *“Limited” evidence for the relationship between noise (including occupational noise) and biochemical effects.*
- *“Sufficient” evidence for an association between ambient noise and hypertension.*
- *“Sufficient” evidence for an association between ambient noise and ischaemic heart disease (observation threshold: Leq,6-22 h: 70 dB(A)).*

Babisch, 2000 (Babisch, 2000b):

- *“No” scientific evidence for association between transportation noise and mean blood pressure readings (exception: in children consistently higher readings were found in the exposed groups).*

- “Little” evidence regarding the association between transportation noise and hypertension.
- “Some” evidence regarding the association between transportation noise and ischaemic heart disease. The latter was viewed as being “sufficient” for action.

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

- “Limited” evidence regarding the association between traffic noise and ischaemic heart disease.

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

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

3.6 Traffic noise related results from the Spandau Health Survey (Maschke et al. 2003)

Since 1982 a longitudinal survey has been carried out which is called the „Spandauer Gesundheits-Survey“ (Spandau Health Survey [SGS]). This survey is directed by the Robert Koch Institute in cooperation with the local health authorities of the borough office (“Bezirksamt”) of Berlin-Spandau. In this survey, the state of health of the participants is examined every two years, to derive general prevention strategies. In contrast to many other epidemiological studies, where the basis for the recruitment of subjects is an explicit sampling frame, is that the Spandauer cohort was made up of persons who heard of the study through appeals and were interested to take part in it. The 9th round of the SGS involved 2015 test subjects, 1714 took part in the study at least for the 5th time.

All participants received a medical evaluation of their “health check” and were requested to see a physician for substantiating diagnoses..

In case of risk factors (overweight, high blood pressure, dysfunction of the lipometabolism etc.) the test subjects were specifically informed about preventiv measures and courses for health improvement that were offered by the Bezirksamt (borough office).

In addition to the obligatory data collection in the 9th round of the SGS, the noise exposure of the participants was determined outside of their homes, and they were asked how much they were disturbed by traffic noise in their homes. The present study is one of a few epidemiological studies worldwide, which, independent of noise exposure during the day, also considers the noise exposure during the night as an independent risk factor for disease with regard to dose-effect relationships. The sound level caused by road traffic at their homes was taken from noise maps of the Berlin Senatsverwaltung für Stadtentwicklung (the “Berlin office for city development”) for the day and the night period. The database provided a map for each address, which made it possible to measure the location of the house (of the apartment) in relation to the street. This site information obtained from the noise map was completed or adjusted using questionnaire data from the subjects regarding the location of their living rooms and bedrooms. Based on these data, the study subjects were grouped into 5 dB(A)-categories of the average A-weighted continuous sound pressure level (sound “immission” level).

For the participants, who were exposed to aircraft noise in their residences, the flight noise exposure was also considered in the evaluation, on the basis of the aircraft noise zones of the Berlin Tegel Airport. The noise related analyses were carried out in the sub-sample that consisted of all subjects, who had filled in the noise questionnaire (N = 1718).

The main results of this study were associations between the night traffic noise exposure and disorders of the cardiovascular system (medical treatment for hypertension), the immune system (medical treatment for bronchial asthma) and the metabolism (medical treatment for

high levels of blood lipids). Day time noise exposure, however, was much less associated with the prevalence of medical treatment of the examined risk factors and diseases (exception: chronic bronchitis). Main results are shown in Tab. 6.

Tab. 6 Traffic noise related Health effects from the Spandau Health Survey

Health effects	Road traffic noise Day time	Road traffic noise Night time	Flight noise Predominantly day
Thyroid hormone	+ -	- -	+ *
Cholesterol	+ -	+ (*)	- -
Hypertension	+ -	++ *	+ -
Angina pectoris	+ -	++ -	- -
Myocardial infarction	+ -	+ -	- -
Migraine	- -	+ (*)	- -
Chronic Bronchitis	+ *	- -	- -
Asthma	+ -	++ *	- -
Allergy	+ -	- -	- -
Cancer	- -	++ -	- -
Psychic disorder	++ -	++ -	- -

(*): $p < 0.1$; *: $p < 0.05$, +: increasing dose effect relationship,

++: monotonously increasing dose effect relationship

This study has an explanatory character. Nevertheless it presents several important and new results:

- There are strong indications that night time traffic noise exposure is more dangerous to health than day time noise exposure.
- Subjective disturbance by noise showed a considerably lower association with the prevalence of medical treatments than the sound pressure level during the night at the subjects' homes.
- Findings that the prevalence of medical treatment for bronchial asthma, chronic bronchitis and allergy tended to increase with increasing noise exposure indicate to an interaction of noise induced endocrine reactions with the immune system. However, traffic related air pollution could have a strong confounding impact on these results.

3.7 Neuro-immune responses to traffic noise

Previous studies which have assessed concentrations of neurohormones such as interleukines (especially of IL-2) and of cellular immune resistance in connection with either recreative sleep or sleep deprivation (Born & Fehm, 2000) have given first indications of a supportive effect of a stress-free, recreative sleep on the immune memory. Correspondingly, the pathogenesis of allergic diseases can be stimulated by adjuvant effects caused by traffic related nocturnal noise (Ising et al. 2002; 2003) as well as by air pollutants such as NO₂ and exhaust particles from diesel engines. During sleep, noise signals which are known to be associated with danger, as for instance from lorry noise, have the potential to trigger stress reactions even if the noise level is low (Spreng, 2001). Associated increases of cortisol in the first half of the night seem to play an important role (Born et Fehm, 2000; Ising, H. et Ising, M. 2002).

Studies on children exposed to traffic-induced noise and air pollution

The hypothesis of an association between road traffic immissions and allergic diseases has been put to the test in a three-phase study on health effects on children.

(1) In a study on sleep disturbance in relation to stress hormone regulation during the night (Ising, H. et Ising, M. 2002), correlations were found by assessing children’s free cortisol excretions in their night urine collected at 1 a.m. and in the morning at wake-up time. The children lived either at a busy road with 24-hour lorry traffic, or in quiet areas, each half in number of the total group. During five consecutive nights the noise level was registered at the side of the road. In the children’s bedrooms, representative measurements of the short-term maximal sound level (L_{Amax} and L_{Cmax}) and of the frequency spectrum were taken.

Regarding the higher noise-exposed children, lorries with $L_{max} > 80$ dB(A) passed by their houses at two minutes’ intervals all night. The indoor levels were $L_{max} = 33-52$ dB(A) resp. 55-78dB(C) with frequency spectrum maxima below 100 Hz.

Results

- Stress hormone excretion was measured in their night and morning urine. The results gave strong indications of circadian rhythm dysfunction of nocturnal cortisol excretion in children who were living on streets with day and night lorry noise as compared with less exposed children.
- The endocrine dysfunction was correlated with substantial sleep disturbances and elevated prevalences of bronchial asthma and dermatological allergies.
- Children diagnosed with bronchial asthma or an allergy were found to have significantly higher excretion rates of cortisol metabolites in the first half of the night as compared with the second half (Ising, H. et Ising, M. 2002).

With specific reference to nocturnal lorry noise, indoor noise levels rated up to now as not interfering with a healthy sleep (Interdisziplinärer Arbeitskreis, 1982), may according to the presented results be considered as detrimental to health, if the C-weighted indoor level exceeds $L_{max} = 60$ dB.

(2) In a practice-based study using a blind interview design (Ising et al. 2002; 2003), the combined effects of chronic exposure to traffic-related air pollution and noise upon the risk of skin and respiratory diseases in children were investigated. The study sample comprised 401 children aged 5 to 12 years seeing their pediatrician in his clinic. The diagnoses for each child or the respective treatments in an observation period of one month were analysed together with the parents’ statements concerning the density of road traffic on their street, and several confounding factors. Multiple regression analyses resulted in significant increases of the relative risks of bronchial asthma, chronic bronchitis and neurodermitis in children living on heavy traffic streets (see Tab. 7). A comparison of the results with the literature on health effects caused by air pollution alone showed that traffic noise at night seems to have an additional, adjuvant effect on the pathogenesis of the quoted diseases.

Table 7: Odds Ratios (95% confidence intervals) for respiratory and dermatological diseases related to the traffic loads

Traffic load category:	low	moderate	high
Diseases and respective treatments			
Bronchitis or asthma diagnosed: n = 107	1 n = 213	4,36 (2,45 – 7,75) n = 114	7,65 (3,93 – 14,86) n = 60
Neurodermitis or allergies diagnose n = 124	1 n = 213	2,68 (1,60 – 4,50) n = 114	3,10 (1,68 – 5,71) n = 60

(3) In a current study traffic related air pollution and noise was measured outside the bedroom windows of ca. 10% of the patients. Based on these measurements immission charts were prepared in order to assess objective categorisation for air pollution and noise in the total group (n = 401). In addition, the contact rate of the patients with their physicians during the past 5 years for bronchitis and neurodermitis was determined. The final step will be an analysis of the correlation between the traffic related immissions and the annual contact rate with the physician for the quoted allergic diseases. The results are expected to be published in the second half of 2003.

3.8 Inner ear damage

Noise can cause inner ear damage either by acute overload or by chronic high exposure, which leads to metabolic exhaustion. Examples for the first mechanism is impulsive noise of toy pistols, fire crackers etc. and military low altitude flight (MLAF) noise. MLAF noise is a danger for the inner ear if L_{\max} exceeds 115 dB(A) and/or the level increase exceeds 60 dB/s. In Germany, the percentage of inner ear pain in 12-17 year olds caused by MLAF noise in area with 150 m and 75 m military flights were 0.7% and 1.1% respectively. Children with inner ear pain after MLAF noise had significantly elevated hearing thresholds as compared with controls (Ising et Rebentisch, 1993). The percentage of convulsions in babies after extreme MLAF noise exposure reported by parents from 75 m areas was found to be 0.17%. Six years later these children were found to have permanent hearing threshold shifts at high frequencies (Ising et al.1991). For other studies on aural effects of MLAF noise see (Ising et al.1998 and 1999; Joachims et al.1999;).

After shifting the minimal altitude for all flights to 300 m in Sept. 1990 none of the above described symptoms were reported and no area differences of hearing thresholds in children were observed (Ising et al. 1998).

Chronic exposure to mean levels per 24 h of $L_{eq} > 75$ dB(A) increases the risk of inner ear metabolic exhaustion and permanent hearing threshold shift. Leisure noise is a substantial danger for the inner ear especially of children, teenagers and young adults. Epidemiological studies of teenagers with no occupational noise exposure show an increasing number with a substantial and measurable irreversible inner ear damage. This is basically due to the wide spread exposure to noisy toys (cap pistols and squibs), fire crackers (Segal et al.2003) and exposure to electronically amplified music, e.g. from personal cassette players (PCP), at discos or concerts etc. (Ising et al 1997; for review see Maassen et al. 2001). Inner ear damage by impulsive noise is possible, when the peak level exceeds 140 dB and simultaneously the sound exposure level exceeds 125 dB(A). This limit is often exceeded by airbags (Hohmann, 1998).

Acoustic limiting values for prevention of inner ear damage

- MLAF noise:
 - $L_{\max} < 115$ dB(A) and the level increase is $dL / dt < 60$ dB/s
 - MLAF with minimal altitudes ≥ 300 m implies no hearing risk.
- Impulsive noise is a hearing risk if the peak level is
 - $L_{\text{peak}} > 140$ dB and the sound exposure level is
 - $SEL > 125$ dB(A)
- Loud music is a hearing risk if the exposure per week is
 - $L_{eq} > 85$ dB(A) for 40h or $L_{eq} > 95$ dB(A) for 4h

4. Overview of existing indicators

- The percentages of population annoyed by environmental noise during daytime is a general accepted indicator. As described under 3.1 the percentage of those highly disturbed in the population may be preferable for some reasons.

- During night time the percentage of population with sleep disturbance is the most suitable indicator, since self reported annoyance during sleep is either reflecting noise induced delay to fall asleep or awakening with noise induced difficulties to fall asleep again or – which is quite often reported – the people extrapolate their daytime annoyance further into night time. In the past noise induced sleep disturbances often have been defined as conscious awakening. Newer results of sleep research – as described under 3.2 lead to the conclusion, that frequent arousal and stress hormone secretion without conscious awakening seem to be detrimental to health if they appear over years.

Most of the existing noise regulation are based on these two indicators in order to limit the percentages of the population which are highly annoyed at daytime and/or have noise induced sleep disturbances at night.

5. Proposition of pertinent effect indicators and adequate exposure indicators

5.1 Noise effect indicator: Annoyance

- MLAF noise: km flight path with altitudes below 300 m per year and area (km²)
- Civil flight noise:
Percentage of the population in areas with $L_{den} = 45-49, 50-54, 55-59, \dots$ dB(A)
- Road traffic:
Percentage of the population with $L_{den} = 50-54, 55-59, 60-64 \dots$ dB(A),
- Rail noise:
km of rail in residential areas with $L_{den} = 55-59, 60-64, 65-69 \dots$ dB(A)

Application: MLAF – regional, the other three – international

5.2 Noise effect indicator: Sleep disturbance

Percentage of population with impaired recovery function of sleep due to traffic noise at night.

- MLAF noise:
km flight path with altitudes below 1000 m per year and area (km²)
- Civil flight noise:
Percentage of the population in areas with $L_{night} = 35-39, 40-44, 45-49, \dots$ dB(A),
- Road traffic noise:
Percentage of population with $L_{night} = 40-44, 45-49, 50-54 \dots$ dB(A),
- Rail noise:
Percentage of population with $L_{night} = 45-49, 50-54, 55-59 \dots$ dB(A).

Application; MLAF – regional, the other three – international

5.3 Noise effect indicator: Cardiovascular risk due to traffic noise

The most important condition to avoid an increased cardiovascular risk due to traffic noise is an undisturbed sleep.

Percentage of population with $L_{\text{night}} \geq 55$ dB(A), or

km of road in residential areas with more than z busses and /or lorries per night.

6. Conclusion

Annoyance and sleep disturbance in the sense of conscious awakening are generally accepted effects of environmental noise. According to newer literature, however, noise induced arousal and secretion of cortisol especially in the first half of the night seem to be more sensitive indicators for noise induced detriment of sleep than conscious awakening. Long-term derogation of the recovery function of sleep by traffic noise has the potential to increase the risk of cardiovascular and several other diseases. The evidence of cardiovascular risk due to traffic noise is assessed as "limited or sufficient". Children under combined exposure to traffic related noise and air pollution were found to have relative risks of chronic bronchitis, asthma and neurodermitis, which cannot be explained by air pollution alone. Representative studies are necessary to determine the magnitude of this health risk. Long-term traffic noise induced derogation of sleep seems to have an adjuvant effect in the pathogenesis of these diseases.

There is evidence that traffic noise exposure at night plays a more important role in the pathogenesis of diseases than traffic noise exposure during the day. Noise induced hearing impairment due to military low altitude flight noise, impulsive noise from toy pistols etc., and long-term overexposure by loud music are summarised and acoustic limiting values are proposed to avoid inner ear damage.

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Noise_E3	Population annoyance by traffic noise	DPSEEA
<i>Issue</i>	Noise	
<i>Definition of indicator</i>	Percentage of the population annoyed by traffic noise	
<i>Underlying definitions and concepts</i>	The indicator is based on the assumption that exposure to traffic noise (road, railway and air), induce annoyance in awake persons. Using dose effect relationship (Miedema & Vos, 1998) the population percentages of highly annoyed can be calculated from exposure data. Population: total population	
<i>Specification of data needed</i>	<i>Road traffic:</i> Percentages of the population exposed to $L_{den} = 50-54, 55-59, 60-64... \text{ dB(A)*}$ <i>Air traffic:</i> Percentages of the population in areas with $L_{den} = 45-49, 50-54, 55-59, ... \text{ dB(A)}$ <i>Railway traffic:</i> Percentage of the population exposed to $L_{den} = 55-59, 60-64, 65-69... \text{ dB(A)}$ Total population	
<i>Data sources, availability and quality</i>	National models of traffic noise exposure. Noise maps of cities. Maps of flight noise areas around airports. Available in Germany and other European countries Alternative: Representative annoyance surveys	
<i>Computation</i>	The indicator can be computed for each traffic source of noise as: $E (R_{level} * N_{level} / N_t)$ where N_{level} is the number of people exposed to a noise level category, R_{level} is the regression coefficient according to Fig. 1 and N_t is the total number of population.	
<i>Units of measurement</i>	Percentage	
<i>Scale of application</i>	National as well as local – residential settings	
<i>Interpretation</i>	The indicator provides a measure of the long-term substantial disturbances related to exposure to traffic noise.	
<i>Linkage with the other indicators</i>	Driving force: <i>Passenger and goods transport demand by mode of transport</i> Effect: <i>Population annoyance by traffic noise;</i> Action: <i>Application of regulations, restrictions and noise abatement measures</i>	
<i>Work needed</i>	Adaptation to highly annoyance only and to L_{den} from L_{dn} in Fig. 1	

*) An introduction of L_{de} is not necessary and not even correct since the day time annoyance is dependent on 24 h noise exposure. Therefore L_{den} is the basis for calculation of highly annoyed at day time.

Noise_E4 Sleep disturbance by night time environmental noise	DPSEEA
<i>Issue</i>	Noise
<i>Definition of indicator</i>	Percentage of the population with decreased quality of sleep
<i>Underlying definitions and concepts</i>	<p>The indicator is based on the assumption that exposure to night time noise by different sources, e.g. traffic (road, railway and air), industry, entertainment facilities, neighbours induces sleep disturbance. Underlying definitions are:</p> <p>Sleep disturbance: arousal reactions and change of sleep phases, duration of deep sleep and REM sleep, irregularity of heart rate, stress hormone dysregulation, body movements; alternative: subjective quality of sleep and mood in the morning .</p> <p>Population: total population surveyed</p>
<i>Specification of data needed</i>	<p><i>Road traffic:</i> Percentages of the population exposed to $L_{night} = 40-44, 45-49, 50-54... \text{ dB(A)}$</p> <p><i>Air traffic:</i> Percentages of the population in areas with $L_{night} = 35-39, 40-44, 45-49,... \text{ dB(A)}$</p> <p><i>Railway traffic:</i> Percentage of the population exposed to $L_{night} = 45-49, 50-54, 55-59... \text{ dB(A)}$</p> <p><i>Industry:</i> factories and manufacturers; building activities; load/ unload facilities</p> <p><i>Entertainment:</i> bars/ discos ; luna-parks etc ; noisy sports</p> <p><i>Neighbours</i></p> <p>Total population of the sample surveyed</p>
<i>Data sources, availability and quality</i>	<p>National models of traffic noise exposure. Noise maps of cities. Maps of flight noise areas around airports.</p> <p>Available in Germany and other European countries</p> <p>Alternative: Representative surveys of sleep disturbance</p>
<i>Computation</i>	<p>The indicator can be computed for each traffic source of noise as:</p> $E (R_{level} * N_{level} / N_t)$ <p>where N_{level} is the number of people exposed to a noise level category, R_{level} is the regression coefficient of dose-effect-relationship and N_t is the total number of population.</p> <p>Alternative: The indicator can be computed for each source of noise as:</p> $100 * (N_{sd} / N_t)$ <p>where N_{sd} is the number of sleep disturbed people and N_t is the total number of surveyed population</p>
<i>Units of measurement</i>	Percentage
<i>Scale of application</i>	National as well as local – residential settings
<i>Interpretation</i>	The indicator provides a measure of the long-term health effects related to exposure to different sources of environmental noise at night time
<i>Linkage with the other indicators</i>	<p>Main driving force: <i>Passenger and goods transport demand by mode of transport</i></p> <p>Effect: Noise-induced decrease of sleep quality.</p> <p><i>Population annoyance by traffic noise; and Cardiovascular risk due to traffic noise</i></p> <p>Action: <i>Application of regulations, restrictions and noise abatement measures</i></p>
<i>Work needed</i>	Dose-response-relationship of the above mentioned noise effects in sleeping persons to be assessed from existing literature