Health Effects caused by Noise: Evidence in the Literature from the Past 25 Years

H. Ising and B. Kruppa

Federal Environmental Agency (retired), Berlin

Traffic noise is the most important source of environmental annoyance. According to the Environmental Expert Council of Germany, severe annoyance persistent over prolonged periods of time is to be regarded as causing distress. Previously, extraaural noise effects were mostly assessed using a paradigm in which the sound level played the major role. On the basis of this paradigm the relatively low sound level of environmental noise was not considered to be a potential danger to health. In contrast to this numerous empirical results have shown long-term noise-induced health risks. Therefore a radical change of attitude - a change of paradigm - is necessary. For an immediate triggering of protective reactions (fight/flight or defeat reactions) the information conveyed by noise is very often more relevant than the sound level. It was shown recently that the first and fastest signal detection is mediated by a subcortical area - the amygdala. For this reason even during sleep the noise from aeroplanes or heavy goods vehicles may be categorised as danger signals and induce the release of stress hormones. In accordance with the noise stress hypothesis chronic stress hormone dysregulations as well as increases of established endogenous risk factors of ischaemic heart diseases have been observed under long-term environmental noise exposure. Therefore, an increased risk of myocardial infarction is to be expected. The results of individual studies on this subject in most cases do not reach statistical significance. However, according to the Environmental Expert Council, these studies show a consistent trend towards an increased cardiovascular risk if the daytime immission level exceeds 65 dB(A). Most of the previous studies on the extraaural effects of occupational noise have been invalidated by exposure misclassifications. In future studies on health effects of noise a correct exposure assessment is one of the most important preconditions.

Keywords: Noise, environmental, occupational, annoyance, stress hormones, cardiovascular risk.

Introduction

The Federal Immission Protection Act (Bundes-Immissionsschutzgesetz) 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.

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.

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

Most questionnaires used to evaluate the degree of individual noise disturbances, offer a scale of answers ranging from “not disturbed at all” to “very much disturbed”. To meet the criterion of substantial disturbance only such persons are included who classify themselves in the intermediate range or higher. Table 1 depicts mean noise levels L_{den} (reference time period: 24 h, with supplementary malusses of 5 dB and 10 dB added to evening and night time level respectively) at which 20% of the interviewed persons felt significantly or very much disturbed.

Table 1. Relationship between traffic noise levels and disturbance for different types of traffic noise

20% of interviewed persons: noise source:	(substantially) significantly disturbed at L_{den}	very much disturbed at L_{den}
aircraft flight noise	51 dB(A)	61 dB(A)
road traffic noise	57 dB(A)	65 dB(A)
rail noise	63 dB(A)	78 dB(A)

The data are taken from a recent meta-analysis (Miedema and Vos, 1998) of studies on the dose-response relationship between various types of traffic noise and their disturbing effects. It is clearly shown that at identical noise levels the disturbance by aircraft noise is greater than that by rail or road traffic noise.

Paradigms of occupational and environmental noise effects

The legal basis on noise protection at the workplace (cf. Regulations on the Prevention of Accidents, 1990) contain regulations both about preventive medical measures and the right to claim damage compensation. Because these two cases require different levels of evidence for noise-induced health impairments, they may briefly be discussed at this point. With regard to damage compensation, clear evidence is required of a causative connection between defined noise exposure and the postulated health impairment. In the case of preventive health protection, however, any reasonable assumption of a possible health hazard justifies protective measures. From the point of view of preventive medicine the quality of the evidence connecting noise exposure and health hazards is usually classified in one of three categories:

- sufficient
- limited
- inadequate

Up to now, the only noise induced occupational disease acknowledged with sufficient evidence is noise induced hearing loss.

According to ISO 1999, occupational noise induced hearing damage does not occur below immission levels of L_{eq} = 80 dB(A) with reference to 40 working hours per week. Higher exposure will increase the risk of permanent hearing threshold shifts – also for listeners to loud music. An analogy is observed between noise-induced hearing impairment and the damaging effects of exogenous toxic substances. The effects of toxins and of loud noise on the hearing capacity is proportional to the total amount absorbed and to the total sound energy immitted respectively. For all extraaural noise effects no analogy is found to toxic substances.

The majority of studies on extraaural work noise effects were based upon the paradigms of aural noise effects and have erroneously used persons with noise exposure below 85 dB(A) as "non-exposed" control groups. Additionally, ear protection - which is normally only partly used - was rarely taken into account. This has lead to severely underestimated noise effects resulting from exposure misclassifications of up to 30 dB and bereaving such studies of any relevant contribution to the question of work noise-induced extraaural health impairments. Therefore the conclusions being drawn from these studies are false (Babisch, 1998). There are only a few studies, in which such incorrect methods were avoided. These studies, however, have revealed a significant increase in cardiovascular diseases as well as increased mortality rates following long-term work noise exposure (Zhao et al.1991; Ising et al. 1999; Melamed et al. 1999).

There is a major need, therefore, to abolish such paradigmatic errors of the past, and not to draw misleading conclusions from earlier methodically incorrect occupational noise studies.

Table 2. Review of recent studies on the relationship between traffic noise disturbance and increases in stress hormones.

+ : significant increase; = : no change; ø : not measured

First author	Year	Noise type (test nights/yrs) n	acute/ chronic noise	L_{eq}[dB(A)] (L_{max})	Test persons n	Recor-dings n	Adrenaline	Nor-adrenaline	free cortisol
Maschke	1992	Flight noise 8 nights	acute	**29-55** (55-75)	8	64	+	=	ø
Maschke	1995	Flight noise (8 nights)	acute	**29-45** (55-65)	28	224	+	=	+
Evans	1998	Flight noise (1.5 years)	chronic	**53/62 ***	217	217	+	+	ø ***
Harder	1999	Flight noise (40 nights)	acute + chronic	**42** (65)	15	600	=	=	+
Ising	1999	Flight noise (1-3 x 10s)	acute	(90-100) *	68	272	=	=	=
Carter	1994	Road noise (2 nights)	acute	**32** (65-72)	9	18	=	=	ø
Babisch	1996	Road noise (years)	chronic	**45-75***	200	200	=	+	ø
Braun	1999	Road noise (years / 2 nights)	acute + chronic	**<45/ 53-69 ***	26	152	=	+	+
Evans	2001	Road noise (years)	chronic	**46/ 62 *** **L_{dn}****	115	115	=	=	+
Ising	2001	Road noise (years)	chronic	L_{max}:30/42 (at night)	56	56	ø	ø	+ **

* outside level ** 1st half of night *** only total cortisol measured **** day and night level

On the other hand, Jansen and Notbohm (1994) have come to the conclusion that the risk of cardiovascular disease can scarcely be increased by traffic noise, *since the noise effect research has failed to provide unambiguous findings in spite of essentially higher noise exposure*. Even more extraordinary is the assessment quoted by the above authors, based solely on the noise level, as "healthy / indifferent / unhealthy or disturbing / substantially disturbing / hazardous". Moderator variables such as situative factors are not taken into account.

Their model, which led to this overestimation of the noise level, is valid only for direct noise effects such as hearing damage.

extraaural noise effects i.e. physiological, psychological and mental, are believed to be in analogy with aural noise effects. The authors argue as follows: *The lower the sound intensity measured the greater is the variation in the reactions observed in terms of individual and situational influences, with the result that any scientific statements on extraaural noise effects are subject to considerable limitations.*

The first part of this statement is correct, but the conclusive part seems rather preposterous. It is true that in laboratory tests with very high sound levels these are closely correlated with the noise effects, but they yield no information on the effects of environmental noise. In the sound level range of environmental noises, the moderators play a decisive part (Deutsche Forschungsgemeinschaft, 1974). Therefore, environmental noise effects must be analyzed using adequate methods.

Environmental noise effects cannot be extrapolated from short-term laboratory findings, as has been shown in a study: "Stress reactions and health hazards induced by traffic noise exposure, comparison of methods between field and laboratory trials" (Ising, 1983). Several hours of exposure to road noise under field conditions at level L_m = 60 dB(A) caused greater blood pressure reactions in self-estimated noise sensitive persons than in those who were noise insensitive. Short-term sound exposure in the laboratory with intermittent noise at L =100 dB(A) showed opposite results. No correlation between blood pressure reactions

under field conditions with hours of exposure and laboratory studies with a duration of several minutes could be established.

This shows that results of short-term laboratory tests cannot be used as a model of long-term effects caused by environmental noise exposure. Therefore, the dose-response diagram in Table 1 is not apt to be used as a basis for assessing environmental noise effects. As a consequence, such paradigmatic errors of the past ought to be recognized as such and be eliminated.

Serious mistakes were also made when establishing limiting levels for environmental noise immission (Maschke et al. 2001a,b). This was a result of inadequate interdisciplinary cooperation. A prerogative of any competent future studies in the area of noise effect research should therefore consist of requesting and planning a close cooperation between the physico-technical, the socio-psychological and medical as well as the epidemiological disciplines. Positive examples today are the interdisciplinary working group " Problems of Noise Effects" of the Federal Environmental Agency (UBA) and among the larger research projects the flight noise study of the German Research Association (Deutsche Forschungsgemeinschaft, 1974) as well as the studies on the health effects of military low-flight noise conducted on behalf of the UBA (Curio and Ising 1986, Ising et al. 1991) and the Caerphilly-Speedwell cardiovascular studies (Babisch et al. 1999).

Psycho-social noise effects

A major result of the DFG-Study was the finding that noise disturbances can only be predicted to a maximum of one third by acoustic measures such as noise level, exposure time, frequency range etc. Non-acoustical variables, such as situative and individual moderators, exert a considerable influence on noise processing while remaining unchanged under noise exposures (Guski 2001).

Evidence of disturbances resulting from environmental noise has been designated as definitely sufficient and assigned an initial threshold L_{dn} = 42dB(A) (outside) in the "Noise and Health" report of the Health Council of the Netherlands (1994). Jansen and Notbohm quote L_m = 45–55 dB(A) as the range of the threshold for reactions by the population (based on a disturbed contingent between 0 and 20%) (Jansen and Notbohm, 1994). Ortscheid and Wende (2000), in their assessment of flight noise based on currently available literature, come to the conclusion that the boundary to substantial disturbance is reached with a flight noise of 55 dB(A) in the daytime and 45 dB(A) at night (outside).

In their report the two types of noise effects "disturbance" and "health impairment" play a central part in their objective to develop protective measures. In its special assessment "Environment and Health", the panel of experts for environmental questions (1999) adopted the following viewpoint on questions of disturbance by environmental noise: *In Western Europe the trend has become apparent that the number of citizens suffering from serious disturbance is decreasing, but those subject to less serious disturbance is increasing. The main source of disturbance is road traffic noise. In the "old" Lands of the Federal Republic 68% of the population are disturbed, in the "new" Lands the rate is 83%. Approximately 50% of the population are disturbed by flight noise and 20% each by rail and industrial noise. Under constant noise exposure the degree of disturbance remains unchanged. There are no indications as to people habituating to noise. If the disturbance persists over longer periods of time this strain is to be classified as negative stress (distress).*

Noise-induced sleep disturbances and endocrine reactions

In the past an arousal reaction was considered as being the only relevant health effect of nocturnal noise. In the above mentioned expertise "Environment and Health" it is quoted, however: According to (Maschke, 1998), merely considering the arousal reaction does not take into account either the derangement of the physiological sleep

structure, nor the interference with the normal sequence of the sleep stages and the detrimental effects of compensation.

Although there is no proof yet as to whether and to what extent prolonged noise exposure with ensuing sleep disorders will cause health detriments as described by Maschke, the Environmental Council give their opinion as follows: *From our point of view it cannot be excluded that the observed sleep disturbances may adversely influence health and performance capacity in the long term.*

Therefore for reasons of medical prevention it is necessary principally to avoid noise-induced impairments even when below the arousal threshold.

Acute and chronic stress hormone increases during sleep have been measured even at relatively low sound levels. In a prospective interventional study on children, Evans et al. (1998) found significant increases in adrenaline and noradrenaline excretion after a new airport had been opened. Total cortisol showed a tendential increase, but free cortisol was not measured.

In a field survey on persons living in the neighbourhood of the Berlin Tegel Airport, using recorded simulated night flight noise, an increased adrenaline excretion was measured after the first two test nights. In comparison, an increase in cortisol excretion was found after the third and fourth test nights. As few as 16 overflights at maximum levels of 55 dB(A) – the mean level during test nights being 30 dB(A) – induced significant stress hormone increases and a distinctly deteriorated subjective sleep quality (Maschke et al., 1995). In this study, however, the question on the influence of habituation to night-flight noise remained unanswered.

Harder et al. (1999), therefore, measured free cortisol excretions during three test nights without noise exposure and 37 test nights with simulated flight noise played-back into the bedrooms via loudspeakers. The mean values in the test group showed an acute increase in cortisol excretion only after flight noise test nights two and three. In the following, the mean cortisol excretion values went back to normal, merely superimposed by slight alterations in a seven day rhythm. The most impressive result was a significant increase in cortisol excretion with values above the normal range during the last two weeks under night flight conditions (Maschke et al., 2002). This study has shown that long-term nocturnal noise exposures may lead, in persons liable to be stressed by noise, to permanently increased cortisol concentrations above the normal range.

As part of a survey on "Traffic and health in densely populated Berlin areas" the catecholamine excretion of 200 women was measured (Babisch et al. 1996). Women whose bedrooms were orientated towards streets with rather high noise levels (mean levels at night L_m > 57 dB(A) (outside)) showed significantly increased excretion values of catecholamines compared to those of women living in relatively quiet homes (L_m < 52 dB(A). The results remained stable after controlling covariables (smoking, alcohol, social status etc.).

Additionally, stress hormone excretions were measured in test persons who lived in noisy streets and were asked to leave their bedroom windows open in order to further increase the noise level. These test persons had for several years been exposed to nocturnal mean traffic noise levels between 53 and 69 dB(A) (outside). Acute noise level increases of 9 to 18 dB(A) through leaving the windows open, resulted in a mean increase of free cortisol excretion by one third. A comparison with a control group living in quieter surroundings (L_m < 45 dB(A) showed that the noradrenaline and cortisol excretions in the heavy traffic noise group was higher in concentration even when their windows were kept closed. This finding gives evidence of persistent stress hormone increases as a result of long years of nocturnal noise exposure (Braun, 1999).

Evans et al. (2001) examined children exposed to moderate road traffic noises (outside daytime level L_m > 60 dB(A)). Their night-time urine

contained increased concentrations of free cortisol and cortisol metabolites compared to those of children living in quieter areas (outside daytime level < 50 dB(A)), whereas no differences in catecholamines were observed.

Also, in a study on children with high traffic noise exposures day and night, mainly caused by heavy goods vehicles (Ising H. and Ising M., 2002), a significant increase in excretion of free cortisol and cortisol metabolites was found in the first half of the night, but not in the second half. A comparison of these findings to those of children living in quieter surroundings clearly illustrates an interference of nocturnal noise exposure with the spontaneous circadian rhythm of normal cortisol release.

A review of recent studies on the relationship between traffic noise levels and stress hormone increases is given in Table 2. In only two out of ten studies no stress hormone increases under traffic noise conditions were found. Among five flight noise studies only one (Ising et al. 1999) failed to find stress hormone increases, the reason being that in this case noise exposures were too short in duration and incidences only rare. These were caused by occasional overflights of military aircraft in the late evening hours. The negative results of a road traffic noise study (Carter et al. 1994) has been caused by a methodological error, as stress hormone concentrations were presented rather than hormone excretions or concentrations related to creatinine.

It should be added, however, that - although mostly stress hormone increases were observed - in some rare cases decreased excretions of stress hormones were found under nocturnal noise conditions, (Ising und Braun 2000, Harder et al. 1999).

The Health Council of the Netherlands (1994) classified the evidence of biochemical noise effects as limited. Yet, the results of the presented studies demonstrate that noise exposures over time periods of years may induce, in a certain percentage of exposed persons, permanent changes of the stress hormone regulation, along with possible consequences in terms of functional and organic damages. A decisive factor in the assessment of noise-induced health effects are persistent stress

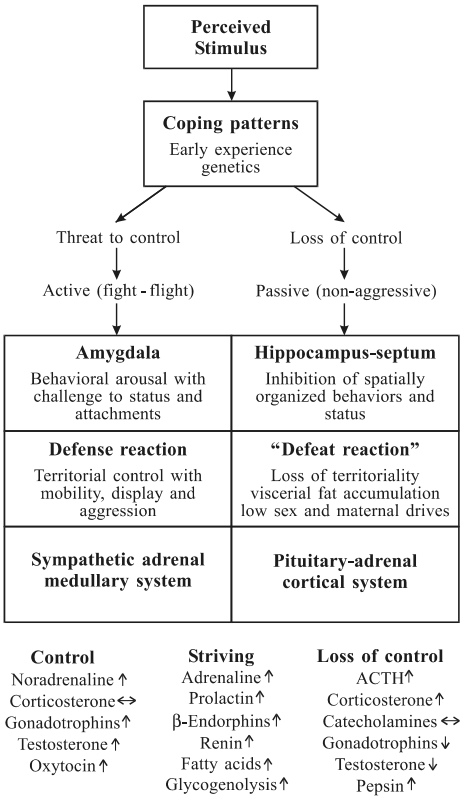

Figure 1. Psychophysiological stress model according to Henry (1992)

"Defens reaction is activated when organism is challenged but remains in control. With loss of control there is activation of the hypothalamopituitary adrenal axis, and the gonadotrophic species preservative system shuts down. Viseral fat accumulates with a Cushingoid distribution, and there is a shift from active defense to a passive nonaggressive coping style." (Henry, 1992).

reactions. Up till now, the majority of studies investigating noise stress effects were based on measurements of the catecholamines adrenaline and noradrenaline and of cortisol.

In terms of the psycho-physiological stress model of Henry (1992), displayed in Figure 1, these stress hormones may be viewed as "guiding substances" for the identification of stress reaction types described there. An increase in cortisol for example shows activation of the hypothalamus, pituitary and adrenal cortex system (HPA system). The consequences of long term activation of the HPA system, may among other things, be insulin resistance, stress-ulcers and cardiovascular diseases.

Environmental noise and cardiovascular risk

The hypothesis of an increased risk of cardiovascular diseases is derived from the stress concepts (Selye 1956, Henry 1992, Björntorp 1997). As shown above, noise exposure may lead to acute and chronic changes of the physiological stress hormone regulation. The different types of stress reactions may lead to derangement of normal neuro-vegetative and hormonal processes and exert an adverse influence on the equilibrium of vital body functions. These include cardiovascular parameters such as blood pressure, cardiac function, serum cholesterol, triglycerides, and free fatty acids, hemostatic factors (fibrinogen) impeding the blood flow in terms of increased plasma viscosity (Friedman and Rosenman 1975), and presumably blood sugar concentration as well. Pathological changes of these parameters may be caused by a variety of endogenous and exogenous factors representing the classical risk factors of cardiovascular diseases. In this context, disturbing noise as well as stress inducing noise at night time is to be classified as an exogenous risk factor in the development of

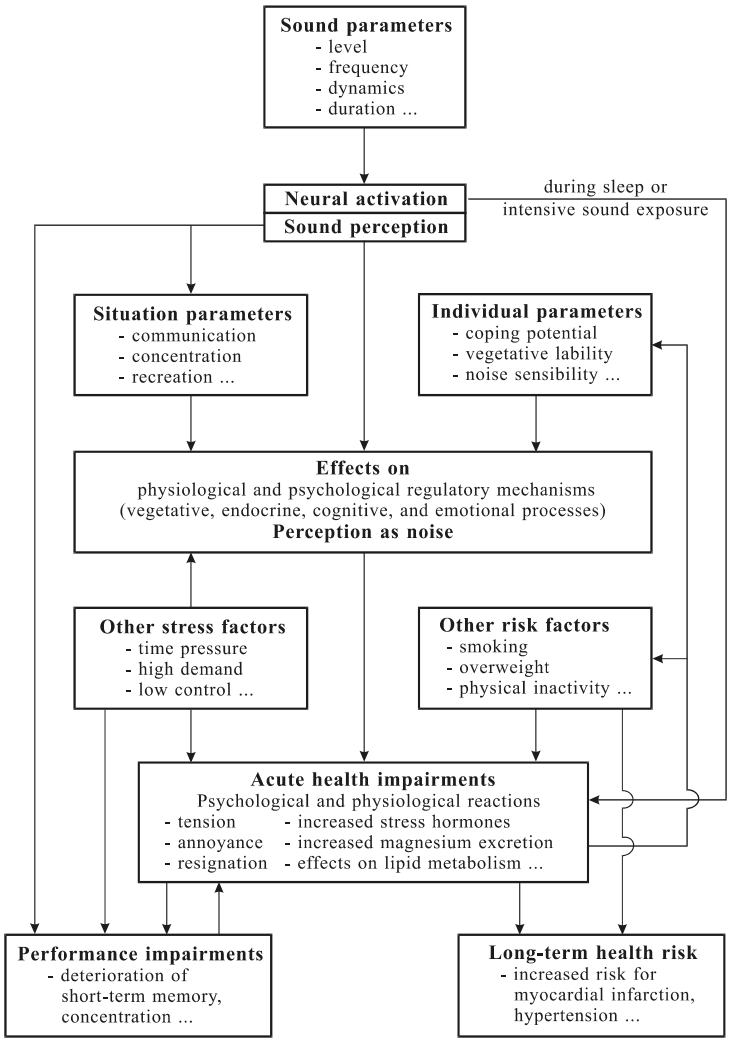

Figure 2. Model of noise perception and psychophysiological effects of noise, risk factors and cardiovascular diseases.

atheriosclerosis, hypertension, ischemic heart disease and myocardial infarction (VDI-Richtlinie 3722, 1988; Babisch 2001; 2002).

Maschke et al (2003) assessed the traffic noise exposure at day and night time separately and found a dose dependent and significant increas of lifetime prevalence of hypertension in persons with $L_{eq\ night} > 50$ dB(A) but not with daytime noise exposure.

In Figure 2, these effects are depicted in a diagram. Sound or noise immissions are processed via central pathways and activate the neuro-endocrinological systems either by inducing direct effects as in the case of work noise, or in the case of relatively low environmental noise levels or during sleep, through instant signal processing in the amygdala which is itself linked with cortical, limbic and hypothalamic centres (Spreng 2000) – or inducing indirect stress effects like disturbances of communication and concentration.

Concerning noise and cardiovascular risk the expertise "Health and Environment" states as follows: Noise, when acting as a stress factor, may enhance the pathogenesis of several health disorders. This is the case with cardiovascular diseases.

The long-term consequences of noise induced increase of stress hormones have to be investigated in epidemiological studies. Studies on the relationship between road traffic noise and coronary heart diseases are briefly described with the following conclusion:. ... "*The studies presented are lacking in test power on account of too few cases in groups with higher noise exposure; the results are statistically insignificant ...*
Nevertheless, the Environmental Council is of the opinion that the results show a consistent trend. The threshold level for possible noise-induced risk of myocardial infarction has been established at a daytime immission level of 65 dB(A)."

With mean road traffic noise exposure levels of more than 55 / 65 dB(A) (daytime / night time) (outside), however, an increase in the risk of myocardial infarction by 20% is to be expected. According to the evaluation of the Health Council of the Netherlands (1994), evidence of an increased risk of cardiovascular disease induced by traffic noise exposures above L_m = 70 dB(A) is considered as being sufficient.

The concurring tendencies and the basic consistency of the traffic noise studies known so far yield sufficient scientific reasons for preventive protection measures to be taken against noise-induced risk increases of cardiovascular disease.

Correspondence Address
H. Ising
Rheinstr. 69, D-14612 Falkensee, Germany
Email: hmising1@aol.com

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