Acute and chronic endocrine effects of noise: Review of the research conducted at the Institute for water, soil and air hygiene

H Ising, C Braun
Institute for Water, Soil and Air Hygiene, Federal Environmental Agency, Berlin, Germany

Click here for correspondence address and email

Abstract

This is a review of the research into endocrine effects of noise since the early 1980s at the Institute for Water, Soil and Air Hygiene. According to our knowledge, no other group has studied systematically the endocrine effects of acute and chronic noise exposure. Mechanisms of acute noise-induced stress reactions as well as long-term increase of stress hormones in animal and persons under chronic noise exposure were studied. Our theoretical background was Henry's psychophysiological stress model with the two reaction alternatives: (i) The fight-flight reaction, characterised by an increase in adrenalin and noradrenaline (ii) The defeat reaction with increased cortisol. Extremely intense acute noise exposure near the threshold of pain caused an increased release of cortisol from the suprarenal cortex but acute noise exposure with levels between 90 and 100 dB(A) caused an increase of catecholamines. Nonhabituated noise increased primarily the release of adrenalin from the suprarenal medulla, whereas habituated noise caused a chronic increase of noradrenaline from the sympathetic synapses under longterm noise exposure at work. Environmental noise exposure (Leq > 60 dB(A)) caused catecholamine increase if activities such as conversation, concentration, recreation etc were disturbed through noise. In sleeping persons, traffic noise with only Leq > 30 dB (A) and Lmax > 55 dB(A) caused significant acute increase of cortisol, which developed into chronic increase if the noise exposure was repeated consistently. Parallel to cortisol, chronic noradrenaline increase was also observed. Based upon the empirical results, a noise stress model was developed which is a first step forward in the theoretical understanding of endocrine noise effects.

Keywords: noise, stress, adrenalin, noradrenaline, cortisol

How to cite this article:
Ising H, Braun C. Acute and chronic endocrine effects of noise: Review of the research conducted at the Institute for water, soil and air hygiene. Noise Health 2000;2:7-24

How to cite this URL:
It is generally accepted that noise has the potential to act as a non specific stressor. The stress concept was introduced into biological sciences by Selye (1956) and since then modified in various ways. We will use one of these modifications as a basis for the study of noise-induced endocrine reactions, the psychophysiological stress model of Henry and Stephens (1977). [Figure - 1] shows a simplified form of this model. A stimulus is perceived by our ears, eyes, nose or other senses and transmitted to the corresponding parts of the brain, where it is analysed.

One of the purposes of this analysis is for the detection of impending danger in the flood of stimuli to which we are exposed. If the stimulus being detected signals danger then immediately one of the two reaction patterns is chosen: If the danger is threatening control over the situation, the organism is prepared for fight or flight. If the danger is overwhelming and inescapable so that the control of the situation appears to be lost, the defeat reaction will automatically follow. Henry and Stephens found that personal coping pattern, early experiences and genetic pre-disposition are important for the decision between the two types of stress reactions.

According to the stress model of Henry and Stephens it is expected that noise stimuli which carry the information of an approaching danger, have the potential to trigger the fight-flight reaction. This is characterised by a secretion of adrenalin and noradrenaline from the adrenal medulla. If a person is exposed to an unexpected noise with a very high level and a sudden level increase, the defeat reaction will come into play with increased secretion of cortisol from the adrenal cortex. In this case the sound itself appears as an overwhelming power and imminent danger, and will cause sudden earache if the individual level of pain is exceeded.

Quite often the reaction is to throw oneself onto the ground. An example of this was given by a tank commander in the Yom Kippur war. After the elimination of the SAM rocket sites behind the Egyptian lines across the Suez Canal, he and his men were waiting in the desert for more orders. They spotted an Egyptian fighter plane, tried to gun it down and the plane following it, but then realised that the second plane was Israeli. Afterwards they waited again in silence. Suddenly they experienced an extremely loud noise. All the soldiers flung themselves flat onto the sand. After several seconds they looked up and saw this Israeli plane flying back home. They then realised what had happened: The pilot had flown from behind a mock attack on their tanks! Several incidences similar to this have been reported to us during our studies of low flying fighter plane noise effects.

The following experimental studies are described in order to present an empirical basis for a theoretical understanding of noise-induced stress reactions. Firstly some remarks have to be made about the problem of applicability of the results of animal experiments to human beings. It is well known that Selye derived his stress concept from the results of various rat experiments and that his stress concept has been successfully applied to humans. Therefore the general study of stress reactions, especially those directly transmitted reactions to noise, is possible in the animal model. If however, reasoning is of importance in the human's response to noise, animal experiments are useless.

**Exposure to extremely intense noise.**

Twelve male test persons were equipped with probes for multiple sampling of venous blood and exposed to the noise of a military low altitude flight of about 20s duration, reproduced via
loudspeakers inside an audiometric cabin. On the first day of the experiment the maximum level was 105 dB(A) and on the second day 125 dB(A). Blood samples were taken before the noise exposure and 1, 8, 16 and 32 minutes thereafter. Before the experiment the bladder was emptied and urine collected after one hour. The course of the cortisol concentration in the blood relative to the pre-exposure value is shown in [Figure - 2]. The cortisol concentration was significantly increased 8 minutes after the 125 dB(A) noise exposure when compared with the 105 dB exposure and reached a maximum 16 minutes after the exposure. The cortisol difference was found to be significant until the end of the experiment, i.e. 32 minutes after the exposure. Also the excretion of free cortisol was significantly increased after 125 dB(A) as compared with 105 dB(A). The catecholamines showed no significant changes.

In addition to this experiment we exposed 20 rats to military flight noise 12 times during 12 hours. Urine was collected for 12 hours without noise and - starting at the same time the next day - for 12 hours with noise exposure. Blood samples were taken from the exposed animals and additionally from 10 controls. The results are shown in [Table - 1]. After 12 short exposures of 10 seconds duration and a maximum level of 125 dB(A), the cortisol concentration in the blood was increased significantly by 69% while the excretion of noradrenaline and adrenalin showed a non significant tendency to decrease (Ising et al, 1990).

During the study "Health Effects of Military Low-altitude Flight Noise" the excretion of free cortisol and metanephrine - a metabolite of adrenalin and noradrenaline - was measured in the urine sampled for 24hours. A total of 19 male test persons, who live in a control area without flight noise, and 16 males from a highly exposed area with 75m low-altitude flight noise (Lmax = 125 dB(A), Leq*) = 69 dB(A)) participated.

*) The Leq is the level of a continuous noise for the duration of one day which has the same acoustic energy as the sum of all noise stimuli during that day.

The results adjusted for age, body mass and alcohol consumption, are shown in [Table - 2]. The metanephrine and cortisol excretions were increased significantly by 24%. Additionally there existed a significant correlation between the fear the test persons experienced in low-flight situations and the excretion of cortisol (r = 0.36, p<0.05) (Schulte and Otten, 1993).

**Exposure to non habituated noise**
A total of 51 male test persons worked for one day in a quiet room and for one day under the electro-acoustically reproduced noise of car racing; an aggressive type of noise with short periods of 100 dB(A) maximum levels and prolonged quieter intervals. This type of noise was totally unusual for the test persons. Urine was collected during the 7 hours of work (soldering of electronic circuits) and noradrenaline and adrenalin were analysed similarly to the first experiment. The results are shown in [Table - 3]. Under this type of noise adrenalin increased significantly while noradrenaline was nearly constant (Ising et al, 1980 b).

In the literature plenty of evidence is given for fight/flight reactions of animals under non-habitual noise exposure. But what will happen after the habituation process, when the animals have learnt that the type of noise they are exposed to, is not signalising any danger? In many cases the stress reactions will disappear. There are several examples in our own animal experiments which prove this (Ising, 1993), but these experiments also demonstrate that under certain conditions a third alternative of noiseinduced stress reaction will occur.
Exposure to habitual noise

We studied endocrine effects in brewery workers, who had been in the same job for several years and were all fully habituated to the noise. All test persons worked for one day without ear protectors. According to estimations of the security engineer less than 1/3 of the workers with Leq > 90 dB(A) normally used ear protectors. Therefore the situation without ear protectors was approximately the normal work situation. A total of 47 workers participated and their urine was collected during 8 hours of work. Their work noise levels ranged from Leq = 71 dB(A) to 102 dB(A). The group was subdivided into three subgroups with Leq < 86 dB(A), 86 - 94 dB(A) and 95 - 102 dB(A). The subgroups did not significantly differ in age, body mass index or in a number of biochemical and anamnestic parameters which were assessed in a general health check up of all the test persons. The only significant differences were an increased total protein in the lowest and an increased hearing level in the highest exposure group as compared with the other subgroups.

The results of catecholamine measurement during work are shown in Table 4. A higher work noise exposure of 10 dB(A) or more lead to a significantly higher excretion of noradrenaline but not of adrenalin (Ising et al, 1980 a, results of re-analysis of published raw data).

This study shows

1.) a dependency of noradrenaline excretion on the level of habitual work noise.

2.) no dependency of adrenalin upon habitual work noise.

3.) an indication that the threshold level for noradrenaline increase under work noise is approximately 95 dB(A).

These results indicate the possibility of chronic increase of noradrenaline under chronic exposure to habitual noise.

The question whether long term noise exposure leads to a chronic increase of noradrenaline was also studied in a rat experiment of 3 months duration. After a habituation period of 3 weeks the noise exposed rats showed a chronic and significant increase of noradrenaline but adrenalin was constant (Gunther et al, 1978).

The noise stress model

From the results of our noise exposure experiments, the noise stress model was derived on the basis of the psychophysiological stress model of Henry and Stephens [Figure - 3]. It shows three reaction alternatives of humans and animals to noise at different habituation levels of the exposed person or animal and for different intensities of the noise (Ising et al 1990).

Exposure to loud and habituated noise e.g. working noise with levels above 90 dB(A) leads to a sympathetic activation and an increased release of noradrenaline from the sympathetic synapses.

Non-habitual loud noise, however, stimulates the fight/flight reaction with an increased release of adrenalin (and noradrenaline) from the suprarenal medulla.
Extremely intensive noise near or above the threshold of pain (Lmax > 120 dB(A)) has the potential to trigger the defeat reaction which is connected with a stimulation of the hypothalamic pituitary adrenocortical system and an increase of cortisol in the blood.

The results of work noise induced stress hormone increase, especially of catecholamines were studied by several other groups. Manninen and Aro (1979) found a work noise dependent increase of noradrenaline only, whereas Cavatorta et al. (1987) found a work noise dependence for both adrenalin and noradrenaline. Sudo et al. (1996) showed that the use of ear plugs reduced the excretion of adrenalin, noradrenaline and cortisol. Melamed and Bruchis (1996) found a decrease of cortisol excretion only in the afternoon, when workers, exposed to ambient noise levels > 85 dB(A), wore ear muffs. These results show that also under field conditions, noise dependent increase of adrenalin, noradrenaline and cortisol have been found. However, according to our knowledge, no other group tried to differentiate between the conditions that lead to different types of noise induced hormone increase.

Taking into account the high noise levels, which are necessary to trigger directly one of these alternative reactions we have to ask if this has any relevance for environmental noise with levels clearly below 90 dB(A).

**Stress reactions induced by environmental noise**

In an experiment 18 male test persons were performing a type of work which demanded constant concentration - assembling of small gear boxes. They worked for one day (8h) each under control conditions (Leq < 50 dBA)), low frequency noise exposure (12-24 Hz octave band noise, Leq = 60 dB(A)) and road traffic noise exposure (Leq = 75 dB(A)).

The results are shown in [Table - 5]. Under the nonhabitual low frequency noise, the adrenalin increased significantly with noradrenaline remaining almost constant. Under the habitual traffic noise exposure noradrenaline increased although the level was only Leq = 75 dB(A) (Ising et al 1983 b).

In another experiment with 41 men we studied the effect of noise-induced disturbance of communication. The test-persons took part in a seminar for several days and were observed for one day under control conditions and one day with road traffic noise exposure with Leq = 60 dB(A). Under this noise exposure the noradrenaline excretion increased significantly by 10% while adrenalin remained constant. (Ising et al, 1983 a).

During sleep the organism reacts especially sensitively to noise exposure. This was confirmed in a field study with 29 test persons who slept four nights under control conditions and 4 nights under flight noise exposure with a mean Leq = 40 dB(A), Lmax = 55 and 65 dB(A), number of overflights 16 and 64.

The mean excretion (sd) of adrenalin and cortisol with or without flight noise is shown in [Figure - 4]. There was a significant increase of adrenalin, in the first two nights with noise exposure as compared to the mean value of the control nights. In the following two nights adrenalin returned to the control value. Cortisol, however, was almost unchanged in the first two nights and increased in the third and fourth night significantly. In this experiment an acute noise-induced increase of adrenalin was counterbalanced by a delayed increase of cortisol (Maschke et al, 1997). Because of this result, the question arose as to whether long
term environmental noise exposure may lead to chronic alteration of stress hormones. This question was dealt with in the following studies.

Catecholamine levels were measured in the night time urine of ca. 200 female subjects, aged 30 to 45 years, who lived on streets with different traffic noise levels ranging between Leq = 45 and 75 dB(A) during the night. (The indoor levels are approximately 10 dB lower than outside when a window is open and 20-30 dB lower with windows closed.) A significant (p < 0.05) model adjusted increase in renal noradrenaline excretion of 0.61 (µg/g creatine) per tenfold increase in traffic volume was found. This corresponds approximately to an increase of the Leq by 10 dB(A). The noradrenaline effect was only found when the exposed room was the bedroom, not the living room. Subjectively disturbed women had significantly higher noradrenaline concentrations than less disturbed women (Babisch et al 1996).

In a road traffic noise stress study, both acute and chronic stress effects of noise were demonstrated (Ising et al, 1998, Braun, 1999). In a group of 26 male and female test persons living on noisy streets, the night time noise exposure was experimentally changed by keeping the bedroom window open or closed for 2 nights each. Because of the high outside noise level most of these test persons were used to sleeping with closed windows. Additionally we tested a control group of 18 male and female test persons who lived on quiet roads and were used to sleeping mostly with open windows. During the experiment urine was collected from both groups and questionnaires concerning the sleep quality were completed in the morning. [Table - 6] shows the noise levels at night time outside the windows as well as the number of test persons and nights of the study.

The resulting excretion of adrenalin, noradrenaline and free cortisol are shown in [Figure - 5] as bar graphs - the medians, the 25 % and 75% values as well as the 5% and 95% values. Some outside values are additionally indicated.

There was no change of the adrenalin excretion for the three noise conditions. In contrast to that there was a significant increase of noradrenaline in the noise group as compared to the controls but no difference between sleeping with closed or open windows. Since the controls and the noise group lived for several years under unchanged noise conditions, the increase of noradrenaline reflects a chronic effect. The additional acute level increase of 9 - 19 dB(A) by opening the window had no acute effect on noradrenaline but on cortisol which also showed a chronic increase under chronic night time noise exposure. Both effects of cortisol, the chronic and the acute effect, were significant. Extrapolation of the night time cortisol excretion for 24 hours opens up the possibility to compare the results with the normal medical range. This comparison reveals that with closed windows about one third of the individual cortisol excretions exceeded the normal range in the noise group when sleeping with closed windows. This result doubled with open windows. In the control group only 5% transgressed from the normal range.

In [Figure - 6] it is shown that the acute cortisol effect is noise level dependent. In this figure the total group is divided into subgroups with level differences of 9 -14 dB(A) (left) and 16 - 19 dB(A) (right). In the latter subgroup the acute cortisol increase is more than fourfold higher than in the subgroup with the smaller level increase.

A differentiation according to gender revealed that the noise-induced acute and chronic cortisol increase was significantly stronger in males as compared to females [Figure - 7].
[Figure - 8] shows the individual trends of the mean cortisol excretion under lower and higher night time noise exposure. It is obvious that in most cases the cortisol excretion increase with acutely increasing noise exposure. However, there are four persons with the opposite trend.

In this study, the increase of cortisol obviously dominates and it cannot be decided whether noise exposure may also result in acute and/or chronic decreases of cortisol.

An overview of the available field studies since 1987 on endocrine effects of occupational and environmental noise is given in [Table - 7]. Three of the 12 studies used occupational noise with indoor levels of Leq = 56 - 100 dB(A). Four of the nine studies on environmental noise effects used experimental noise exposure in the bedrooms with the indoor levels given in the Table. In the remaining five studies the effects of existing traffic noise with the indicated*) outdoor levels were studied, mostly in comparison to less exposed controls.

Discussion

A noise stress model was derived on the basis of the psychophysiological stress model of Henry and Stephens. They distinguish between two types of stress reactions, the fight/flight reaction with increased release of mainly adrenalin from the suprarenal medulla and the defeat reaction with increased release of cortisol from the suprarenal cortex. Both types of stress reactions can also be triggered by noise. When the noise is interpreted as a warning signal of impending danger the fight/flight reaction will follow. Extremely intensive noise, however, is an overwhelming danger in itself, especially if the threshold of pain is exceeded. If such a noise occurs unexpectedly and the noise level increases suddenly so that there is no time for reasoning, it will trigger the defeat reaction.

Additionally to these well-known stress reactions, the noise stress model adds a third type of stress reaction i.e. the increased release of noradrenaline from the synapses of the sympathetic nervous system under the exposure to loud habitual noise.

The first two types of stress reactions have the purpose to enable the organism to cope optimally with a dangerous situation. If the organism, however, has learned by experience, that a certain type of noise is not related to danger, the fight/flight reaction is not at all adequate. We would expect that stress reactions would cease with habituation. Many experimental results confirm this (see for instance Nawroth,1984). However, there are important exceptions. Loud noise itself has the potential to cause an endocrine stress reaction similar to a physical load. During physical exercise noradrenaline is being released from the synapses of the sympathetic nervous system. A similar increase of noradrenaline was observed under exposure to loud habitual work noise with Lmax > 90 dB(A). This noradrenaline increase cannot be explained by an increased release of catecholamines from the suprarenal medulla, since in humans (as well as in rats) the suprarenal medulla contains ca.85% adrenalin and only 15% noradrenaline. In contrast to this the synapses of the sympathetic nervous system contain no adrenalin but only noradrenaline.

However, there is at least one important difference in the reaction of the organism to physical load and to noise exposure. The total peripheral resistance is decreased during physical load and increased during noise exposure. During an experimental increase of the catecholamine concentration in the blood the peripheral resistance is also increased (Ising et al,1992).
There is empirical evidence that noradrenaline may be increased chronically even under exposure to traffic noise, but noise-induced increase of adrenalin is not expected to occur over prolonged periods. However, the results of Evans et al (1998) demonstrate a chronic increase of both adrenalin and noradrenaline in children under persistent flight noise exposure over a period of 18 months. In this study Evans did not find a chronic increase of cortisol. But Evans measured total cortisol and not the biologically active fraction of it, the free cortisol. Therefore, this result does not contradict our results or that of Maschke et al. (1997). According to these studies, long term exposure to road traffic noise as well as civil flight noise has the potential to cause chronic increase of free cortisol which are further increased when there is an acute increase of the night time noise level.

Keeping in mind the noise stress model, increase of cortisol under exposure to environmental noise with levels below $L_{max} = 70$ dB(A) seem to be most unlikely. However, this model was developed on the basis of experimental results obtained with active test persons. Detecting danger and choosing an adequate reaction to it has the purpose to save life, in all life threatening situations. Since hearing is our most important warning system it must work independently of the momentary state of arousal. We are not equipped - neither by nature nor by the Creator with devices like eyelids to lower the input sensitivity of our ears. For these reasons it is not unlikely that stress effects in sleeping persons are triggered by lower noise levels than in active persons.

During sleep - when there is no influence of reasoning - the noise of an approaching lorry may be interpreted by the brain as an approaching danger, but an awake person hears the same and would feel completely safe if in bed but not if on the road. Therefore we may expect that a type of noise which is related to danger from our own experience may trigger a defeat reaction during sleep. For the same reason it is easy to understand that when our partner snores this noise is classified by the brain to be less dangerous than the noise of several lorries per night - even if their Leq is 20 dB less than that of the snoring partner. This example demonstrates that the noise level cannot be expected to be a predictor of stress reactions if different types of noise are under consideration.

From this point of view, the information a sound stimulus is carrying seems to be more important than the sound level. However, since decreasing distance from the source of danger e.g. an approaching thunderstorm, is related to increasing sound levels, it is expected that the sound level of one type of noise will be related to the intensity of stress reactions. Additionally it follows that the momentary level and not the energy equivalent level (Leq) is directly related to stress reactions. But - on the other hand - if only one type of noise is under consideration e.g. flight noise, the Leq is closely correlated to the maximum level and the frequency of its occurrence and is therefore a good predictor of noise-induced stress reactions of this type of noise.

Beside this we all know from our experience that the same type of noise causes completely different degrees of disturbance when we are engaged in different activities e.g. physical work like riding a bike - or concentrating in order to understand a difficult discussion - or if we are trying to sleep near a noisy road.

These considerations lead to the conclusion, that a dose-response dependency between sound level and stress reactions can only be expected if considering one type of noise exposure during one type of activity.
Therefore in the range of environmental noise levels, the measurable sound parameters are less important for the effect of the noise than the information carried by the sound or attached to it by the exposed person, taking into account his momentary activities.

In contrast to this, noise with levels above 90 dB(A) has the potential to act directly as a stressor. In the noise effect model in [Figure - 9] we distinguish therefore between a direct and an indirect transmission path in which a sound stimulus influences physiological and psychological processes. Individual parameters are moderators of both direct and indirect effects of noise.

1. coping potential: the ability not to hear the noise, or to close the window etc.

2. experience with the noise source: i.e. the increasing noise level of a lorry signals an approaching danger on the road,

3. the momentary noise sensitivity : the degree of noise-induced distress is dependent along other influences upon the total amount and duration of stressors one has to cope with. Not included in this simplified model is among other things the influence of reasoning.

From these considerations we concluded that cortisol increase can be expected to be triggered by environmental noise. Most astonishing, however, was the amount of cortisol increases and that these increases became chronic under persistent night time noise exposure. Sapolsky (1984) offered an explanation for such chronic cortisol increase. He compared cortisol increase of young and old rats during and after immobilisation stress. Both young and old rats reacted to immobilisation with a 4 - 5 fold increase of cortisol. But while the young rats showed a normal recovery, the older ones recovered only partially and showed a second increase 3 hours after the end of the immobilisation. Sapolsky discusses carefully the specific differences in the species, rats and humans and concluded, that in both species high cortisol concentrations will cause partial destruction of cortisol receptors in the brain. From such observations and considerations he derived his hypothesis of an interaction between acute cortisol increases and cortisol receptor damage leading to chronic cortisol increase. Among the long term side effects of chronic cortisol increase exceeding the physiological normal range, he mentioned arteriosclerosis and immuno suppression.

Further studies on the amount of noise induced chronic dysregulation of cortisol and its long term health effects are necessary. The predominant effect of chronic noise exposure seems to be the development of hypercortisolism. However, the other possibility of a chronic noise-induced dysregulation is the development of hypocortisolism. In connection with this effect, the observation of an inhibition of stress induced cortisol increase in children with allergic diseases is important (BuskeKirschbaum et al,1997). Therefore in future research a possible relationship between the risk of allergic diseases and noise-induced dysregulation of stress hormones especially of hyper- and hypocortisolism should be studied.[27]

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