Disturbances of sleep by noise

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ABSTRACT

This paper deals with noise-induced sleep disturbances that are regarded as the most deleterious effects of noise with aftereffects on mood and performance the next day. In the long run noise-induced sleep disturbances are assumed to contribute to the genesis and manifestation of multifactorial diseases. The paper gives a rough overview over the state of the art and of the possibility to prevent or at least to reduce these effects and oulines eventually future research in this area.

INTRODUCTION

Apart from noise-induced hearing loss (NIHL), the world-wide most frequent and irreversible occupational disease, noise causes a great variety of extraaural effects. These effects can be, as depicted in Figure 1, categorized according to the delay of their occurrence relative to the onset of noise exposure as follows:

- **Primary effects** are acute reactions that are restricted to the period of noise exposure. They encompass disturbances of sleep and recreation, disturbances of communication as well as excitations of the autonomic nervous system, the latter showing up as alterations of heart rate, blood pressure and skin resistance or as elevated releases of stress hormones.

- **Secondary effects** are as well observed during noise exposure but may outlast exposure time. These effects concern primarily performance decrements and annoyance.

- **Tertiary or long-term effects** are expected after years or even decades of noise exposure. They are permanent alterations of behaviour or chronic diseases.

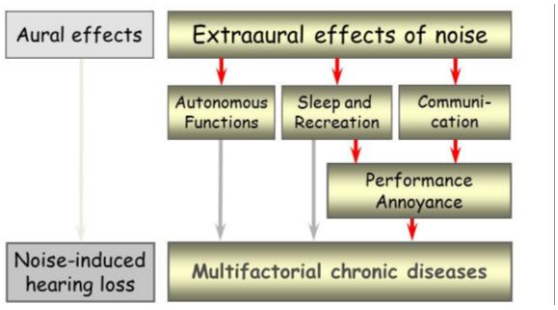

Figure 1. Effects of noise on humans.

Since the middle of the last century a huge number of studies were performed on the various effects of noise on human beings. The majority of these studies focused on annoyance (i.e., the statistically dominating effect). Annoyance is considered as any feeling of resentment, displeasure, discomfort and irritation when noise intrudes into someone's thoughts and moods or interferes with activities. Annoyance causes residents living near noise sources, in particular to transportation or to industrial noise, to complain even officially to the authorities and to form pressure groups, particularly against the noise emitted from aircraft, rail or road traffic, even when it becomes known that new airports, roads or railway tracks are planned. Dose-response curves were most frequently calculated for annoyance. They are characterized by huge variances. Apart from considerably varying individual noise sensitivity these variances are to a considerable degree related to the fact that annoyance is a secondary effect. Annoyance results from disturbances of communication, of sleep and recreation as well as from degraded performance.

NOISE-INDUCED SLEEP DISTURBANCES

Sleep disturbances of whatever reason belong to the most frequently expressed complaints during the consultation hour of the general practitioner. From the viewpoint of medical prevention there are mainly two categories:

- sleep disturbances based on illness that need causal therapy (i.e., the treatment of the underlying disease),

- sleep disturbances that are caused by environmental influences and can be reduced or even prevented by an adequate design of the environment.

The majority of sleep disturbances of the latter category is caused by noise. Noise-induced sleep disturbances are regarded as most deleterious as undisturbed sleep of sufficient length is undoubtedly essential for performance, for well-being and health (WHO 2009, 2011).

Noise-induced sleep disturbances are mainly caused by transportation noise and many publications relate this to the traffic volume in highly industrialized countries. However, complaints about the sleep disturbing effects of transportation noise are found in several documents from the Middle Ages and even from the antiquity. Indeed, it is well comprehensible that the clatter of horse hooves and the noises from wheels driving over cobblestones caused considerable sleep disturbances.

Research in this area began with the solution of basic questions for which primarily artificial sounds (clicks, pink noise etc.) were applied. The majority of studies were then performed in the laboratory where prerecorded noises were

played back. In the next stage residents usually exposed to noise were observed in their familiar environment at home.

Most studies focussed on noise emitted by the three most important means of transportation (aircraft-, railway-, road traffic). This is reasonable not only because of the number of complaints but due to its ubiquitous presence and relative uniformity that allows the development of rather general abatement concepts. Priority was and is still given to aircraft noise, fewer studies were done with road traffic noise and the least with railway noise. This corresponds to the rank order of annoyance as determined in extended social surveys (Miedema & Vos 1998).

Other noises frequently mentioned to cause sleep disturbances but scarcely studied are emitted from industrial plants, entertainment facilities, construction sites and within the last years increasingly often from wind turbines for the provision of renewable energy. They are of rather regional (industry) or transient (construction) significance and they are, due to their variable structure not easy to evaluate. However, basic knowledge gained with studies on transportation noise can be at least partly transferred to these noise sources.

Recording and evaluation of sleep

Systematic research on noise-induced sleep disturbances started at the end of the 60s, after the electrophysiological registration of sleep became available outside clinical facilities. Up to now, polysomnography, i.e. the simultaneous recording of brain waves (electroencephalogram, EEG), of eye movements (electrooculogram, EOG) and of the muscle tone (electromyogram, EMG), is the only measure that allows an unequivocal differentiation between the states awake and sleep and the documentation of the duration and distribution of the various sleep stages over the night. This then very expensive and time-consuming method was implemented at only a very few institutions. Though the equipment became due to tremendous subsequent developments in the electronic sector much lighter and cheaper and despite the acceleration of the evaluation process by the use of evaluation software, the recording of the polysomnogram is still (relative) expensive and time consuming. This led to the development and application of alternative methods that, however, focus on (assumed indicators of) awakening. Very simple is the recording of behavioural awakening, where the investigated person is asked to signal each awakening, e.g. by pressing a button. This method leads, however, to an underestimation of the number of awakenings. A favoured method is the recording of body movements with devices that are worn like wrist watches. The resulting actigram causes, however, a considerable overestimation of awakenings. A recently developed algorithm for automatic evaluation of the electrocardiogram and detection of cardiac arousals could be in connection with the recording of body movements a promising approach for future research (Basner et al. 2008b).

The significance of noise-induced sleep disturbances

In the 70s the leading scientists in this area developed a complex hypothesis that was then and is still generally accepted. It shows the suggested process from acute noise-induced sleep disturbances over the impairment of subjectively evaluated sleep quality and the impairment of mental performance up to irreversible health disorders (Griefahn & Muzet, 1978). According to this hypothesis, depicted in Figure 2, noise causes in the acute situation autonomic, motoric and cortical arousals and awakening reactions. Depending on the frequency and duration of these event-related responses, alterations of sleep structure, of the duration and distribution of each single sleep stage including intermittent wakefulness are expected. The aftereffects are, depending on noise-exposure and the degree of sleep disturbance, reduced subjective sleep quality, sleepiness, as well as impairments of mood and performance. Chronic alterations, that are expected after years or even decades of permanent or frequently repeated noise exposure concern behaviour and health. Chronic alterations of behaviour might be monocausally related to noise, in contrast to health impairments where this is unlikely. Noise is rather expected to contribute to the genesis and the manifestation of multifactorial diseases. The likelihood and the speed of these developments and the extent of these effects are determined by the physical parameters of noise, on the one hand, and by the actual environmental conditions and by the features of the concerned individual, on the other hand.

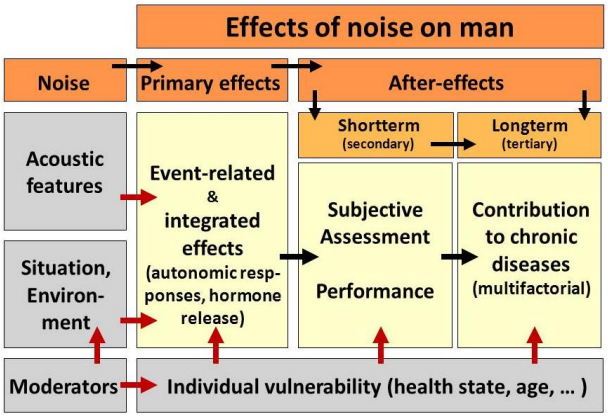

Figure 2. Hypothesis on noise-induced sleep disturbances. Primary and aftereffects.

Approaches to study noise-induced sleep disturbances

Concerning the methodological approach the great number of studies completed since the end of the 60s can be categorised in principle as follows:

- Experimental studies in the laboratory where environmental influences are controlled/minimized and where defined noises are applied (e.g. Basner et al., 2011, Griefahn et al., 2006).
- Experimental studies in the field where in addition to the noise a person is usually exposed to further defined noises are delivered into the sleeping room (Harder et al., 1999).
- Field studies where residents living in different distances to a noise source are studied and (optionally) compared with non-exposed persons (control group) (Basner et al., 2006).
- Intervention studies where the installation of noise attenuating measures (e.g. double glazing, sound barriers) or the implementation of new noise sources (opening of airports etc.) are observed (Fidell et al., 1998).
- Social surveys that concern the subjective evaluation of sleep and the again subjectively evaluated disturbance by noise.

- Epidemiological studies where particular behaviours or diseases are related to a noise source (Jarup et al., 2008).

The probability with which a causal relation can be stated depends, among others, on the temporal gap between the onset of noise exposure and an alteration thereafter. Alterations that occur immediately after the onset of noise exposure are most likely evoked by noise (event-related acute reactions). Alterations of the sleep structure, i.e. of the amounts and distributions of sleep stages and arousals over a whole night results, however, not only from the impact of noise but from the influence of various qualitatively and quantitatively different stressors. Thus the contribution of noise to the total reaction cannot be quantified yet. The determination of a causal relation becomes even more difficult for the aftereffects following noise-induced sleep disturbances as e.g. the actual situation when filling out questionnaires or when performance tests are done may influence the results. Concerning long-term health effects evidence for noise as a possible contributor has increased in the recent past, but high quality non-cross-sectional studies are rare and the role of nocturnal noise exposure is still unclear. This implies that the prognostic significance of the various event-related acute reactions is with regard to possible health detriments unclear.

STATE OF THE ART

The following section deals with the various effects that noise causes during sleep and during the subsequent time awake.

Event-related reactions

Autonomic arousals. The first physiological reactions to acoustic stimuli consist during both the awake state as well as during sleep of transient excitations of the autonomic nervous system. These excitations manifest as alterations of breathing frequency, of systolic and diastolic blood pressure, of the peripheral resistance and an increased release of stress hormones but are most frequently indicated by the alterations of heart rate (Sforza et al., 2000, Muzet & Michel, 1977). The patterns of these cardiac arousals depend, as shown in Figure 3, on the state of consciousness. When awake aircraft noise causes an initial decrease followed by an increase of heart rates. The decrease and the following increase is markedly stronger when the observed person performs a strenuous task. During sleep, however, the same noise is associated with a slow increase of heart rates followed by a decrease below the baseline and a slow increase towards the prestimulus values.

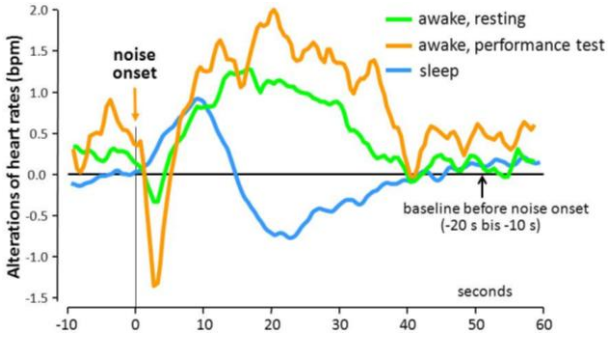

Figure 3. Cardiac arousals – alterations of heart rates due to aircraft noise when awake while resting (green line) or doing a performance test (orange line) and during sleep (blue line).

During sleep these autonomic (or vegetative) arousals can occur alone but they are usually the first responses in a cascade of subsequent alterations. As shown in Figure 4 cardiac arousals have, as far as the intruding noise does not wake the person up, a distinct biphasic pattern. In case, however, that the noise causes the person to wake up the courses of post-stimulus heart rates become monophasic (Griefahn et al., 2008b).

Cardiac arousals without concomitant awakenings are characterised by an initial increase of heart rate by on average 10 beats per minute (bpm) followed by a steep decrease below baseline values. After a slow increase the cardiac response is ceased after 30-40 s. The increase of heart rate corresponds to a vagal inhibition, the subsequent decrease to a counter regulation with an increase of vagal tension and a simultaneous inhibition of sympathetic excitation (Griefahn, 1975, 1989). These autonomic excitations are usually accompanied by cortical arousals of 3 to 10 s, i.e. excitations to be measured with the electroencephalogram (Sforza et al., 2004).

The patterns and the extents of these responses are, however, not specific for acoustic stimuli. They are evoked by numerous other environmental factors, but they occur as well spontaneously without noticable external cause. These arousals do not habituate as demonstrated by numerous studies; they increase during the course of single nights and are evoked even in longterm residents living in the vicinity of noise emitting facilities (airports, railway tracks, roads) (Basner et al., 2011). The autonomic arousals are therefore graded as potentially pathogentic and stress therefore the necessity of the development and implementation of noise abatement measures.

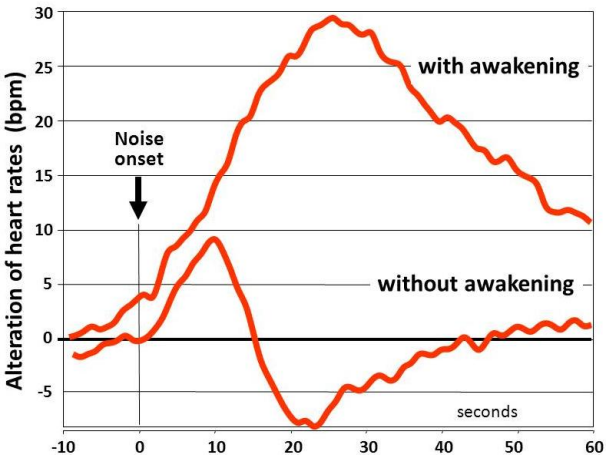

Figure 4. Cardiac arousals – alterations of heart rate due to noise, monophasic response in case of awakening, biphasic alteration without awakening.

The patterns and the extents of the biphasic responses that occur entirely in the absence of consciousness are determined by the physical parameters of the intruding noises. This gives valuable hints for the design of technical noise abatement measures. An example for this assertion is given in Figure 5, left panel. It shows the impact of the slope of rise (the increase of the noise levels per second) for railway noises (but similar effects are found for other noises as well). Steep increases of noise levels that result e.g. from high speed cause dramatic heart rate increases followed by a correspondingly steep decrease of heart rates. More moderate increases of noise levels (slow trains) cause less extreme alterations of heart rate. The significance of the rise times is probably the reason for significant differences between the different transportation modes. Railway noise causes the steepest increase of heart rate to the highest maximum with a similar steep

decrease far below the baseline value. Reactions caused by road traffic noise are similar but the steepness of the increase and the maxima are smaller. Corresponding to the slow increase of the noise levels of flyover noises evoke slow increases in heart rate to later and less pronounced maxima (see Figure 3). Finally, also the maximum noise level has a - though moderate - influence on the course of heart rates (Griefahn et al., 2008b).

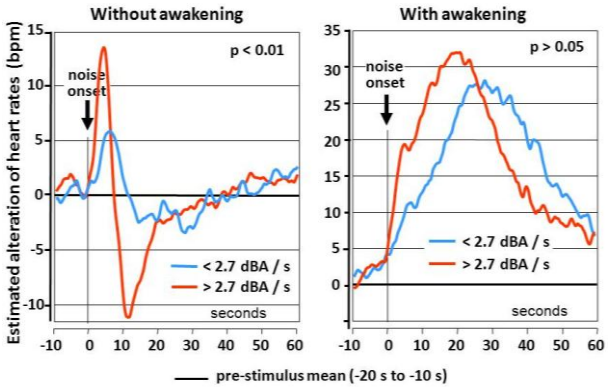

Figure 5. The effect of the slope of rise (speed) of railway noises on the pattern and extent of cardiac arousals with and without awakenings.

If, however, an acoustic stimulus wakes the person up, then the response becomes monophasic (Figure 4, Figure 5, right panel). The change in heart rate reveals a long lasting increase over at least 30 s by on average 30 bpm; the subsequent decrease is slow and the baseline is not regained within a minute after noise onset. In contrast to the biphasic alterations when no awakening occurs, the physical parameters of the noise have only minor or no impact on the extent and on the course of this response. Instead, this response is almost exclusively determined by the excitation that characteristically accompanies any awakening. The probability with which a monophasic cardiac arousal can be expected is therefore similar if not identical with the probability of an awakening (Basner et al. 2008b). The slope of rise seems to have some effect (see Figure 5, right panel) that is, however, not significant. In addition there is no differentiation between various types of transportation noise as it was ascertained for the biphasic arousals.

Cortical arousals/awakenings. Autonomic arousals may occur alone but they are usually accompanied by cortical arousals. These are transient accelerations of the frequencies in the electroencephalogram and in the electromyogram. These arousals last at least 3 s. They are defined as awakenings if they last for at least 15 s (Rechtschaffen & Kales, 1968). Several studies have shown that the probability of being woken up is significantly related to the maximum level of the intruding noises. Figure 6 gives an example for these dose-response relations. It shows the impact of the maximum level on the probability of polysomnographically verified awakenings. Similar dose-response curves have been shown for recalled awakenings, for behavioural (signalled) awakening, and for body movements (Basner et al., 2006; FICAN, 2003; Passchier-Vermeer, 2003; Passchier-Vermeer et al., 2002).

As shown by Figure 6 the likelihood of an awakening response depends on the type of noise (Griefahn et al., 2006). The probability to be awakened is significantly greater for railway noise as compared to aircraft and road traffic noise. Similar results were recently published by Basner et al. (2011). It is therefore reasonable to reconsider the railway bonus that allows a higher equivalent sound pressure level than at busy roads in several countries. Further influences on the probability of an awakening are the slope of rise, the spectral composition of the noise event (Basner et al. 2011), the duration of a flyover or pass-by, the duration of the previous noise-free interval (the longer the interval, the more likely is an awakening) and the time of night, where the gradually decreasing sleep depth plays a decisive role. Age and subjective noise sensitivity have, however, no influence.

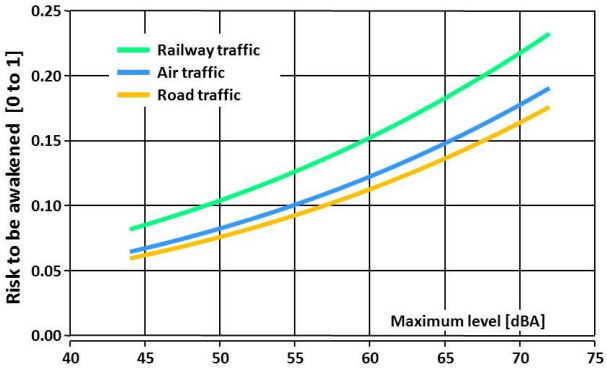

Figure 6. Probability of awakening responses in relation to the maximum levels of the three main sources of transportation noise (unadjusted probabilities are shown).

Alterations of sleep structure

It is generally expected that repeated arousals and awakenings finally result in an alteration of sleep structure (i.e., of sleep onset latency, of total sleep time and of the duration and distribution of intermittent wakefulness and of the various sleep stages). Although these alterations, in particular a prolongation of sleep onset time and of intermittent wakefulness and a shortening of slow-wave sleep (SWS) and of REM sleep were frequently observed and reported, the extent of these effects are as shown by numerous publications surprisingly low and reveal only occasionally dose-response relations (Basner et al., 2004, 2011, Griefahn et al., 2006). Figure 7 shows this for example for a study with 72 subjects, 18-71 years of age, who slept 9 nights each in the laboratory while exposed to aircraft, railway, and road traffic noise (Basner et al., 2011). The increase of the frequency of nocturnal passbys and overflights caused only moderate alterations of sleep structure with gradual increases only for the

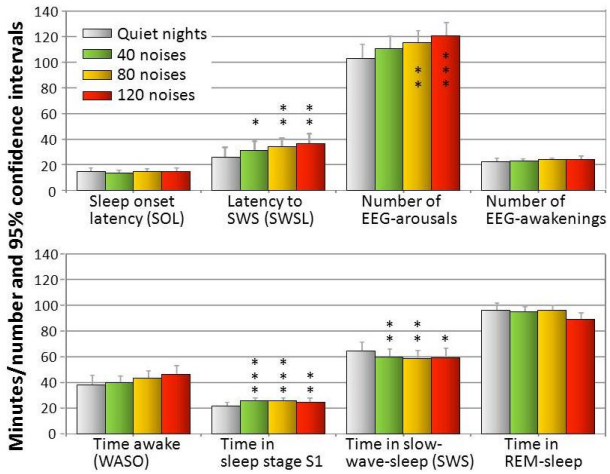

Figure 7. Alterations of sleep structure related to the number of noise events (transportation noise) during sleep.

latency to deep sleep and for the number of cortical arousals. The reason for this is that the number of evoked awakenings increases where the number of spontaneous awakenings decrease meaning that the temporal distribution of awakenings is altered.

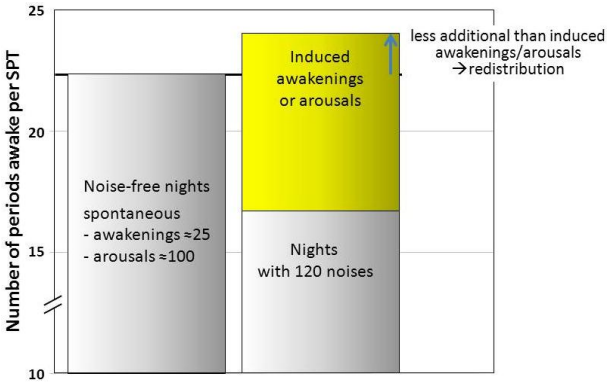

Figure 8. Spontaneous awakenings in noise-free nights. Total number of awakenings in noisy nights is less than the sum of spontaneous and event-related awakenings suggesting a redistribution of awakenings (after Basner et al. 2011).

Aftereffects

The aftereffects are subdivided into the shortterm effects that are ascertained during the wake period after noise-induced sleep disturbances and into longterm effects (i.e., effects on behaviour and health).

Short-term after-effects

Shortterm after effects show up as a decrease of subjectively evaluated sleep quality, as increased sleepiness as well as a decrease of mood and performance (after Basner et al. 2011).

Subjective evaluation of sleep quality has been ascertained with short questionnaires in almost every study performed in the laboratory and in the field. Due to a meta analysis that includes 24 studies the impairment of subjectively evaluated sleep quality is significantly related to the equivalent sound pressure level (Miedema & Vos, 2007). In addition, this analysis revealed some differences between the various transportation noises that correspond with annoyance during the day, i.e. the strongest effect of aircraft and the least effect of railway noise. As this contrasts with the physiological measurements it is supposed that the actual situation when filling in the questionnaire plays a decisive role. Moreover, night time noise annoyance that was ascertained after sleep in a few studies showed a dose-response relation with the nocturnal noise load (it is, however, not justified to ascertain annoyance as a surrogate for sleep quality). Eventually, dose-response relations were ascertained for sleepiness which can be objectified with the pupillary unrest index (Basner et al., 2008a).

Concerning performance there have been contradictory reports. A detailed analysis shows, however, that the consolidation of memory, for which deep sleep in the first hours of sleep in connection with low cortisol levels is decisive, is impaired and that the quantitative parameters of performances (reaction times) and those performances that rely on the shortterm memory are impaired after sleep in noise (Elmenhorst et al., 2010).

These shortterm aftereffects were almost exclusively ascertained during a short time period after awakening. In a recently completed study where 48 subjects slept 4 subsequent nights and 4 subsequent days in the laboratory and did thereafter experimental 8-h work shifts has shown that sleepiness was during the entire work shifts (day shifts and night shifts) greater and some performance parameters (e.g., reaction time) were impaired.

Longterm aftereffects. The hypothesis presented in Figure 2 presupposes that permanently lasting noise exposure finally impairs health. Monocausal relations (i.e., diseases only caused by noise) are, however, not likely. The assumption that noise contributes to the genesis and premature manifestation of multifactorial diseases bases on the following observations:

- First, the acute autonomic responses to noise (e.g. elevations of heart rate and of blood pressure, reduced variation of blood pressure during sleep in noise (Griefahn et al., 2008b; Haralabidis et al., 2008)) are not specific to noise. Instead these reactions are evoked by numerous other extrinsic but also by intrinsic stimuli.

- Second, these reactions do not, as already mentioned, habituate. They are still observed in residents living years or even decades in the vicinity of noise emitting facilities (airports, railway lines, busy roads, industry).

Both, the fact that the genesis of those diseases to which noise is assumed to contribute to takes years and even decades and that a great number of risk factors have been identified for all these diseases is the reason for the still limited evidence. At the same time, up to now there are only a few studies that concerned explicitly the longterm health effects due to nocturnal noise.

The assumed mechanisms of the well founded hypothesis of a causal relation were summarised in corresponding models (Babisch, 2000; Fyhri & Aasvang 2010). They show the process from permanent or repeated noise exposure, over the acute physiological and hormonal reactions up to the manifestation of diseases. A meta analysis based on 4 extended studies in Berlin on the connection between the incidents of myocardial infarctions and noise load by road traffic resulted in the calculation of a plausible dose-response relation (Babisch, 2006). Though the contributing studies did not focus on sleep, nocturnal noise load seems to be decisive and the model proposed by Babisch (2006) is at least a founded hypothesis for further directed studies.

A hint to the health relevance of noise-induced sleep disturbances results from the comparison of sleep under exposure to noise with age-related alterations of sleep. A suitable indicator of physiological sleep quality is the sleep disturbance index (SDI, Griefahn et al., 2008a). This index includes 7 weighted parameters derived from the polysomnogram. It increases as shown in Figure 9 steadily and significantly with age indicating gradual decreases in sleep quality. The index calculated for 18-20 year old persons who were during sleep exposed to transportation noise with equivalent sound pressure levels between 39 and 50 dBA corresponds with that calculated for about 20 years older persons sleeping in quiet environments.

The SDI calculated for persons sleeping in a noisy environment are markedly lower than calculated for persons suffering from obstructive sleep apnea (Cassel et al., 2008). Another comparison on the basis of performance tests shows that the prolongation of reaction times after noise-induced sleep disturbances are significantly less than after partial sleep

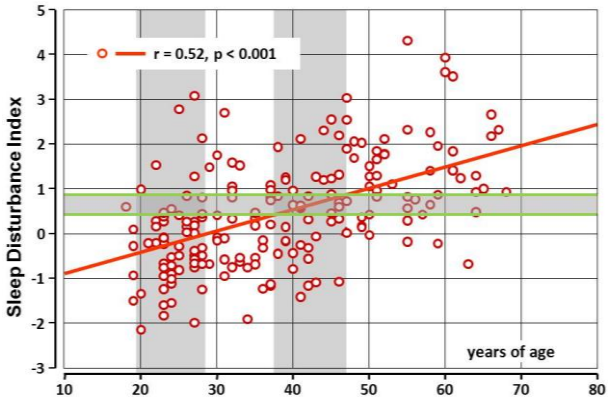

Figure 9. Sleep disturbance indes during lifetime and in 18-30 year old persons sleeping under noise exposure.

deprivation by 3 h/night and less than in a situation where oxygen concentrations or alcohol concentrations in blood are elevated (Elmenhorst et al., 2010). Thus the effects of nocturnal noise on health are rather subtle.

The significance noise has in the long run on health needs to be investigated using epidemiological approaches. Up to now only a few epidemiological studies were done. The assumed relation between elevated blood pressure and the prevalence of hypertension and nocturnal noise emitted by aircraft and road traffic has been explored in the HYENA project (Jarup et al., 2008). Residents living in the vicinity of airports in Athens, London, Milan and Stockholm were investigated and statistically significant associations were found. Moreover, measures of blood pressure revealed that single noises cause acute increases of blood pressure and that the natural dip of blood pressure during the night is damped when sleeping in noisy environments (Haralabidis et al., 2008, 2010; Jarup et al., 2008).

The epidemiological studies performed by Greiser et al. (2007, 2009, 2010) focused on residents living near the airport Cologne/Bonn. This airport is characterised by a high number of nocturnal flights. The studies focused on the prescriptions for the treatment of hypertension and of cardiovascular diseases and on the prescription of tranquilizers, sedatives and hypnotics. For these prescriptions an association was found with noise load between 3 am and 5 am and this was stronger in women than in men. Concerning cardiovascular diseases there was a statistical association with the equivalent sound pressure level calculated over 24 hours, particularly for persons who cannot claim noise abatement measures financed by the airport. Finally, the authors found an increased risk of diseases in women for breast cancer, Non-Hodgkin lymphomas and leukemia, where the given explanations are, however, not convincing.

NOISE ABATEMENT

Noise abatement was even practised in the Middle Ages and in the antiquity where approaches were realized that are even today used. A major measure was the ban of noise emitting facilities from the cities or the ban of transportation means for certain time periods, particularly for the night.

According to the studies performed so far noise-induced sleep disturbances are to be considered as a health risk. The majority of noise-induced sleep disturbances is undoubtedly caused by transportation noise and most studies focussed accordingly on these noises. As the extents and patterns of the respective effects were related to various acoustic features (such as maximum levels, rise times, duration, noise-free intervals, frequency spectra) as well as to situational conditions (such as time of night) it is possible to transfer some knowledge to other noises that are not or less intensively studied. These are industrial noises that are significant on a rather regional level and construction noises with a rather temporal significance. Rather uniform noises are known to disturb sleep as well, namely church bells and wind turbines. With the increasing use of renewable energy the latter become more important and need to be investigated more intensively. A considerable increase is also expected for transportation noise. As traffic volume will increase further on but as the transportation facilities (roads, railway tracks, flight paths) are not accordingly extended, traffic will gradually invade into the shoulder hours and into the night.

Thus, noise abatement is an essential element of public health care. Directed noise abatement presupposes, however, to set limits. None of the limits suggested so far can protect everybody from the deleterious effects of noise. There are always persons who will awake by the lowest noises and others who are not affected even by the loudest noises. Thus all limits where in case of excession noise abatement measures have to be provided/financed by the operator of a noise emittent facility are compromises (and not seldom determined/influenced by financial possibilities).

Traditionally, limits consider exclusively the acoustic load and are expressed for single events (by maximum noise levels, SEL etc) or for complex scenarios (by integrated measures such as the equivalent noise level). Of course, the lower the limit is set, the fewer persons will be affected. However, as various scenarios calculate to the same equivalent noise level one and the same Leq does not provide the same protection.

A protection concept that solely concerns the effects of noise was developed for the residents living in the vicinity of airports (Basner et al. 2006). As a criterion event-related awakenings were chosen as awakenings represent the strongest form of activation and are as shown above associated with strong excitations of the autonomic nervous system.

The respective limits were deduced from the highly significant dose-response relation between event-related awakenings with the maximum sound pressure levels of the corresponding overflights determined in a field study with 64 residents that were investigated for 9 subsequent nights. This allowed the calculation of a curve where the risk to be woken up is the same. The fewer event-related awakenings are allowed the greater is the area around a noise source, e.g. the runways of an airport. The risk was set to a single additional awakening as averaged over a year. This corresponds to an increase of the total number of awakenings by about 4 % as compared to 24 spontaneous awakenings that occur during an 8-hour noisefree night. The calculation of this curve of equal risk considers also the fact that the probability to be woken up increases – due to gradually lighening sleep – during the course of the night. To consider further subjectively evaluated sleep quality the concept allows not more than a single recallable awakening, i.e. longer lasting wake periods that are likely evoked by noises with maximum levels of at least 65 dBA. The so defined protection area is shown in Figure 10 together with the isocontours for equivalent noise levels of 50 and 55 dBA for Frankfurt airport. The area defined by the risk of a single awakening corresponds nearly with the area that is defined by the equivalent noise level of 50 dBA but the effect-oriented areas are greater where many but not very loud landings occur and smaller in areas with

only a few but very loud starts. This effect-oriented concept has already been realized at the airport Leipzig-Halle with a great number of nocturnal flights.

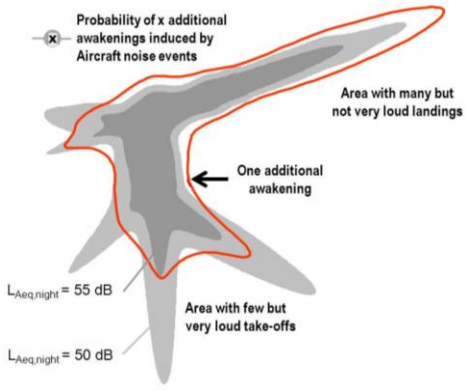

Figure 10. Acoustically defined (grey areas) and effect-oriented (red line) isocontours calculated for Frankfurt airport (flight data from 1997).

Curfews. A rather common measure to reduce the noise load for residents near noise emitting facilities is the implementation of curfews. The disadvantage of these curfews that do not encompass the whole night but e.g. 4-5 hours to the traffic volume then increases during the remaining hours of the night. Studies where curfews at the beginning and at the end of the night were compared (Figure 11) have shown that

- curfews in the early night (left panel of Figure 11, quiet from 11pm to 3am or from 11pm to 5am) are less beneficial than curfews in the early morning (left panel, quiet from 3am to 7am) as deep sleep usually occurring within the first hours of sleep is less affected by noise than lighter sleep that prevails in the second half of the night,

- the benefit is, particularly for curfews at the end of night, greater for early and for normal sleepers than for late sleepers because they have the chance to finish their sleep in quiet, whereas the late sleepers are at the end of their sleep always exposed to noise.

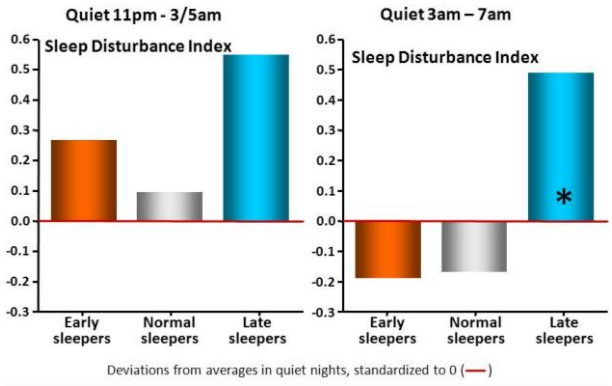

Figure 11. Effects of traffic curfews: Quiet from 11pm to 3am or 5am (left), quiet from 3am to 7am (right). Reduction of Sleep Disturbance Index (better sleep) only for curfews during late night and only for early and normal sleepers.

FUTURE RESEARCH

During the last decades significant knowledge about protection of health against the deleterious effects of noise was gathered. Nevertheless, there are still considerable gaps in knowledge that have to be closed in future studies. Basically three fields of study have to be mentioned:

- Epidemiological studies. The approach with epidemiological studies has to be intensified where supplementary experimental studies are necessary to clarify the mechanisms of noise processing.

- Individual vulnerability. Most of the experimental and field studies performed so far that served the derivation of limits were exclusively done with healthy persons. Persons at higher age are characterised by an increased vulnerability against environmental noise and these persons react stronger to nocturnal noises than healthy persons. Another vulnerable group are probably children who as a rule sleep during times with high traffic density and thereby high noise load.

- Development of methods. Another goal is the development of methods that can be applied in field studies instead of the polysomnogram that are, however, able to indicate the awakening reactions with the same accuracy. A suitable indicator of awakening reactions is certainly the strong monophasic alteration of heart rate as described above and shown in Figures 4 and 5 (Griefahn et al. 2008b). This presupposes the recording of the electrocardiogram. The arousals can be automatically detected by a special algorithm (Basner et al. 2008b). The accuracy of this procedure can be increased by the simultaneous recording of the actigram. As both, the electrodes for the recording of the electrocardiogram and the device for the recording of the actigram can be self-applied this method provides the possibility to perform even extended field studies at relatively moderate costs.

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