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Cardiovascular effects of noise

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Introduction

It is well understood that noise levels below the hearing damaging criterion cause annoyance, sleep disturbance, cognitive impairment, physiological stress reactions, endocrine imbalance, and cardiovascular disorders. Public health policies rely on quantitative risk assessment to set environmental quality standards and to regulate the noise exposure that is generated by environmental noise sources in the communities. According to the European Environmental Noise Directive (END) the member states are currently assessing and documenting (noise maps) the noise exposure from environmental noise sources in their countries, including road, rail, aircraft, and industrial noise. [1] The END (Annex III) advises using dose-effect relations to assess the effects of noise on populations. Noise from transportation is by far the most widespread source of noise exposure, causing most annoyance and public health concerns. [2] With respect to noise mitigation measures, the avoidance and prevention of physical health effects plays an exceptional role in public health, besides other aspects of the quality of life that may be affected by noise. The cardiovascular effects of noise have been the source of growing interest in recent years. This is because - on the one hand - evidence has increased that noise affects cardiovascular health. High blood pressure and ischemic heart diseases (including myocardial infarction) - on the other hand - have a high prevalence in industrialized countries and are a major cause of death. [3],[4] The question at present is no longer whether noise causes cardiovascular effects, it is rather: what is the magnitude of the effect in terms of the exposure-response relationship (slope) and the onset or possible threshold

(intercept) of the increase in risk. Effect estimates tend to be larger when the exposure and effect modifying factors are considered. This is important for causal reasoning and inferential statistics (significance testing). For the calculation of population attributable risks the weaker effect estimates that refer to the total population are relevant, because detailed information about such modifying factors are often not available on a large scale and are not considered in noise mapping. Noise mapping data according to the END, however, will be increasingly used, not only for action planning, but also for health impact studies. The available data needs to be critically viewed.

In this issue of Noise and Health well-known protagonists of noise effects' research give an overview about the major research that has been carried out in their countries in the field of cardiovascular effects of noise. Most environmental epidemiological noise studies been carried out in The Netherlands, Sweden, The United Kingdom, Serbia, and Germany.

Biological Background and Hypothesis

Although the topic that noise affects cardiovascular health has garnered a lot of attention, particularly, in recent years, [5],[6],[7],[8],[9] it is important to realize that it has a long history; experiments on animals and humans had already been carried out in the post-war period, where the basic biological mechanisms were investigated. [10] The general stress theory is the rationale for the hypothesis that noise affects the autonomic nervous system and the endocrine system, which in turn affects the homeostasis of the human organism. [11],[12],[13] Persistent changes in endogenous risk factors due to noise-induced dysregulation and disturbed metabolic function, promote the development of chronic disorders such as atherosclerosis, hypertension, and ischemic heart diseases in the long run. It is important to note that non-auditory noise effects do not follow the toxicological principle of dosage. This means that it is not simply the accumulated sound energy that causes the adverse effects (dealing with decibels is not like summing up micrograms as we do for chemical exposures). Instead, the individual situation and disturbed activity need to be taken into account (time activity patterns). It may be very well that 80 decibels at work cause less of an effect than 65 decibels when carrying out mental tasks at home or 50 decibels when being asleep. In this respect, the evening hours, when people come home from work for relaxation and the night time when the organism physically recovers from daytime load and brain restoration takes place may be particularly important with respect to noise-induced health effects. Sleep is an important modulator of cardiovascular function. Noise-disturbed sleep, in this respect, must be considered as a particular potential pathway for the development of cardiovascular disorders. [14],[15]

Epidemiological Reasoning

Coherence (biological plausibility), consistency (among studies using different methods and design), the presence of an exposure-response relationship, and the magnitude of effect are important criteria when judging the causation of an empirical association. [16],[17],[18] The biological plausibility of the hypothesis that long-term exposure to environmental noise causes cardiovascular diseases is supported by a couple of findings and facts:

Short-term laboratory studies carried out on humans have shown that the exposure to noise affects the sympathetic and endocrine system, resulting in acute unspecific physiological responses (e.g., heart rate, blood pressure, vasoconstriction, stress hormones, electrocardiogram (ECG) changes). [10],[19],[20],[21],[22] At moderate environmental noise levels such reactions were found, particularly when the noise interfered with the activities of the individual (e.g., learning, concentration, attention).Noise-induced, instantaneous, autonomic

responses do not occur only in the waking hours, but also in sleeping subjects, even when no electroencephalogram (EEG) awakening is present. [23],[24] The responses do not adapt on a long-term basis, although a clear subjective habituation occurs after a few nights. [25],[26] Subjects who had lived for several years in a noisy environment still respond to acute noise stimuli. Repeated arousal from sleep is associated with a sustained increase in daytime blood pressure. [27]The long-term effects of chronic noise exposure at high noise levels have been studied in animals, showing permanent vascular changes and alterations in the heart muscle, which indicate an increased risk of cardiovascular mortality. [28]Although the effects tend to be diluted in the occupational studies due to the 'healthy worker effect', epidemiological studies carried out in the occupational field have shown that employees working in high noise environments are at a higher risk for high blood pressure and myocardial infarction. [29],[30],[31],[32],[33],[34]

Higher risks of high blood pressure due to aircraft and road traffic noise and of myocardial infarction due to road traffic noise were found in a number of community noise studies, showing a reasonable consistency, particularly, as the new studies have entered the pool in recent years (see the articles in this issue). Even though the results of individual studies are sometimes not statistically significant, they can contribute to the meta-analyses to overcome the problem of small numbers. Meta-analyses have been carried out to develop exposure-response curves that can be used for quantitative health impact assessment. [29],[35],[36],[37] In the recently published World Health Organization (WHO) Night Noise Guidelines for Europe, it has concluded in its recommendations for health protection that there is 'limited' evidence that the risk of cardiovascular diseases increases for night noise levels (L night,outside) above 55 dB(A). [15] 'Limited' evidence, because not many studies are available where the exposure of the bedroom is explicitly related to the night noise level. The night noise criterion is deduced from studies looking at daytime noise (L Aeq,16hrs) or 24-hour exposure levels (L dn of L A24hrs), for which the evidence is regarded 'sufficient' by most experts, for noise levels greater than > 65 dB (A), by assuming a 10 dB (A) difference of average day and night noise levels for road noise in urban settings. With regard to the magnitude of effect, the effect estimates (odds ratios) of the relative risk derived from the meta-analyses range approximately between 1 and 1.7 across the relevant exposure range. However, small relative risks - in general - are subject to a confounding bias due to unknown factors, or residual confounding due to incomplete adjustment of factors in the analysis, which the study design, data assessment, and statistical analysis must account for.

The magnitude of an effect seems to vary depending on whether the outcome and / or exposure is assessed subjectively or objectively. For example, hypertension can be defined as the prevalence of self-reported, doctor-diagnosed, high blood pressure, on the intake of anti-hypertensive drugs and ambulatory blood pressure measurements. Reporting bias cannot be excluded depending on the sequence of data assessment and the awareness of the subjects of the study objectives. Conversely, the reporting of exposure or exposure-related factors (e.g., noise annoyance, which may also serve as a determinant of health effects) may be biased. Any such differential reporting may result in unknown under- or overestimation of the true effect. Particularly, when exposure and outcome are assessed at the same point in time on a subjective basis, results must be interpreted with caution. Selection bias is very likely to be present in noise studies. Noise sensitive or annoyed subjects or subjects with serious health problems may have moved out of the exposed areas. It is often observed in social surveys as well as in epidemiological studies that the relative risk drops for subjects in the highest noise category. Self-selection may be the reason for it. On the other hand, the formal objection against cross-sectional studies of an unknown cause-effect relationship ('egg or hen first?') seems to be less of a problem in noise studies. It is more likely that subjects move from noisy to quiet areas rather than the opposite. Noise effects found in cross-sectional studies, in this respect, tend to be underestimated, which would not discard an association as such.

Considerations for Future Noise Research

The assessment and adequate control of confounding factors is essential in epidemiological noise research. However, their treatment in the statistical analysis needs to be carefully considered. For example, does it make sense to control for intermediate factors which - according to the reaction model - play a role on the pathway from exposure to disease, for example, biological risk factors such as blood lipids and high blood pressure? Or, is it useful to control the current behavioral risk factors that may have changed as a consequence of the prevalence of the disease of interest? Both categorical data analyses (relative risk of different noise categories with reference to the lowest) as well as continuous data analyses (relative risk per dB-increase, based on regression models) give useful information and should be carried out simultaneously. Although the latter is best for assessing trends, the first can help to detect non-linear relationships and possible higher risks of exposure groups where the number of subjects is small compared to the many data points that determine a trend. Not only the significant, but also the insignificant results (odds ratio and confidence interval) must be reported in publications, to enable the consideration of the data in meta-analyses. For example, sometimes results are explicitly given for one sex, but not for the other, only stating that the results are not significant. However, the data can contribute to the pooled estimation of the exposure-response curves.

The exposure assessment can be improved when room orientation and shielding is taken more into consideration in noise studies. Noise indicators usually refer to the most exposed façade of buildings. In such cases day and night noise exposure are often highly correlated, particularly, for road traffic noise. However, the relevant rooms and related activities, for example, sleep, might not be affected - thus lowering the effect estimates due to differential exposure misclassification, by making them more susceptible to other small confounding factors. An improved and standardized noise propagation and mapping software is used a lot more, to create detailed noise maps that show calculated noise levels for all facades of the houses. An increase in the availability of data can be expected in the near future due to the obligatory END noise mapping. The data, however, must be critically viewed, because the façade levels may refer to major trunk roads in the distance, but not to the local traffic next to the building, when only the primary road network has been considered for noise mapping. Another way of handling room orientation is to consider the variable as an effect (exposure) modifier in the statistical analyses, by using interaction terms or stratification.

Noise annoyance, the prevalence of chronic health problems, years of residence, and window opening habits may be other interesting exposure and effect modifiers to look at. Some studies showed higher noise level related risks in annoyed subgroups or in subgroups with long residence time. The latter makes sense with respect to the longer induction periods that manifest cardiovascular health outcomes. Some studies suggest gender differences in the effects of noise. However, the direction (higher risk in males or females) is not yet clear; the results are contradictory and may be due to random variation or incomplete control of the female hormonal status.

The effects of combined exposures due to multiple noise sources (e.g., different transportation noise sources or transportation noise and occupational noise) on health are of interest in future noise research. The same applies to the combination of noise with other life-stressors, to identify certain risk groups that might need higher protection. The competing risks of noise and air pollution are a challenge for future noise research. As far as road traffic is concerned, both refer largely to the same source (excluding long-range transport or air pollutants). Study designs accounting for differential impacts of shielding could help to disentangle the effects and to assess possible synergistic effects of combined exposures. Distance to the road has been used as a determinant of traffic-related exposure to air pollutants in studies. However, proximity alone is not an exclusive indicator of exposure to air pollution from road traffic, but also a major determinant of noise emission and immission.

Railway noise is causing increasing concern due to high speed trains and increasing freight train traffic during the night. Almost nothing is known about cardiovascular effects of railway noise. Annoyance studies suggest that railway noise is less annoying (at the same average noise level). The question is whether the 'railway bonus' also applies to physical health. As transportation policies aim at moving traffic from the street onto the railtrack, research is needed in this field; particularly, because of the rapidly increasing freight train traffic during the night.

Children are often considered as a vulnerable risk group, because they have less control over the environment than adults. [38],[39],[40] The studies show (primarily systolic) blood pressure increases in children exposed to aircraft and road traffic noise. However, the studies are not always consistent. Longitudinal studies could answer the question whether the observed effects are transient or whether the noise exposure at a young age has consequences on their health in the later years.

Networking

Networking among noise effects' researchers has been a powerful tool to strengthen the research in this field. This refers to the formulation of test hypotheses, the study design, the sharing of costs, and the dissemination of results. The European-funded projects PAN ('Protection Against Noise'), NOPHER ('NOise Pollution Health Effects Reduction'), PINCHE ('Policy Interpretation Network on Children's Health and Environment'), RANCH ('Road traffic and Aircraft Noise exposure and Children's cognition and Health'), HYENA ('HYpertension and Exposure to Noise near Airports'), and the ongoing ENNAH ('European Network on Noise and Health') project have been good examples, just to mention a few. The health effects of noise are considered as an emerging environmental burden of disease issue by the World Health Organization (WHO) and the European Environmental Agency (EEA). [6],[9] The quantitative assessment of the impact of environmental noise on cardiovascular health and other health endpoints is still high on the agenda.

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