Systematic reviews in noise epidemiology.
Limitations and chances from a Public Health view
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ABSTRACT
Systematic reviews can provide valuable evidence to health care and health policy, especially, when clinically important effects of similar magnitude are observed. Whether these strong requirements are met in noise epidemiology is questionable. The medical model does not fully fit with the stress and context driven causal pathways through which the health effects are determined. Heterogeneity of effects is expected due to different background prevalence of vulnerabilities, health and disease. Exposure assessment is more demanding than in related areas (air pollution) due to the need of accounting for perceptual accuracy and sound control options. Furthermore, the applied A-weighted sound level indicators do not correlate in the same way with the actual nervous system arousal for all sound sources. Eventually, most research is of observational nature and randomization and placebo control is largely not feasible. Nevertheless, systematic reviews are indispensable for the further development of noise epidemiology. Furthermore, in order to compare the potential size of the adverse effects of noise on humans at the policy level the calculation of disability adjusted life years (DALYs) is required and dependent on high quality systematic reviews. Examples of limitations will be outlined in detail and suggestions are made for future improvements.

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1. INTRODUCTION
There is no doubt that the development and use of systematic reviews and meta-analyses to judge the relative importance of certain health determinants has a large potential to lead to a rationally guided public health decision making. It should, however, not be forgotten that these tools of evidence-based science were developed for randomized controlled trials and for making recommendations in clinical medicine. Whether this paradigm could also be applied to environmental factors was less clear.

With the establishment of the Environmental Burden of Disease (BoD) concept the WHO has made an enormous effort to support its claim that a relevant fraction (~25%) of the global disease burden results from environmental causes (1,2). However, while related environmental factors such as air pollution were included in the assessment – noise was not among the factors considered initially. Eventually, in 2011 the WHO published the burden of environmental noise assessment (3) and in the just updated Environmental Burden of Disease assessment noise is included (4).

Why it has taken so long to include noise in such a BoD assessment and why is a valid assessment still an ongoing challenge?
The first and main reason is that the medical model does not fully fit with the stress and context driven causal pathways through which the expected health effects of noise are channeled. Second, the physical description of the noise is only a surrogate indicator of the perceived psychosocial stress moderated or mediated by the personal vulnerability under the specific environmental conditions and coping options of the human receptor in the respective area of concern. Following this accepted stress conceptualization of potential health effects in noise epidemiology (5–8) – a much larger heterogeneity is expected to result from population studies across various geopolitical and socio-cultural backgrounds compared with air pollution effects which follow closer the medical model with its toxicology based physiological mechanisms (9). Furthermore, additional heterogeneity of the noise effects is expected due to different environments, building structures and housing, coping opportunities and background prevalence of health and disease in the respective study populations.

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The WHO-Bonn has decided to make the necessary update of the noise guidelines from 1999 and to base the update on systematic reviews of selected sound exposure types and health outcomes – although the majority of data still come from cross-sectional and less from prospective studies.

From the view of public health administrators at regional and local levels there is that the average results of a systematic review may be difficult to apply in contextually different local circumstances, where environmental health impact assessment are conducted. On the other hand, the results represent the best available knowledge across different environments and are a proper basis for BoD assessment at larger (national or supra-national) scales – necessary for make informed decision making at the policy level.

It is the main aim of this paper to point to critical underlying conceptual issues and quote examples from literature or own work to illustrate potential ways how the requirements at the various policy levels could be met by including in the guidelines necessary information in addition to the results of systematic review to allow valid health impact assessments of noise exposures also at lower levels of administration where the preventive actions are actually translated into potential health gains.

2. THE IMPORTANCE OF THE UNDERLYING NOISE-HEALTH MODEL

2.1 The basic model

The old Henle-Koch causal model (“epidemiologic triad” is well suited to explain the major difference between a toxicology versus a stress driven model of potential health effects. The crucial distinction, graphically outlined in Figure 1, is that the size of the environmental determination of health effects is much larger in a stress based model of noise effects compared with air pollution that follows toxicology principles. While the contribution of the agent and host part of the causal equation may be approximatively of similar size for air and noise pollution – the environmental part plays a much larger role for triggering the health effects of noise.

![Figure 1. The „epidemiologic triad“ as guide to integrated assessment](image)

If we consider only the agent and the host in our analyses we will overlook the environmental (and social) determinants of noise effects which are not only responsible for the size and severity of the adverse effects but also may be amenable to preventive action.

2.2 The more complex model

A more advanced path model of potential health effects of sound does include not only the crucial environmental partitions but does also integrate the subjective part of the exposure (perceived noise stress) in addition to the single physical indicator of the sound exposure (Figure 2). Only a few noise epidemiology studies dealing with cardiovascular outcomes have included some information on bedroom location to be used in meta-analyses (10–13) but not provided extended information on housing, building type, neighborhood and/or community characteristics.

Unfortunately, we are currently not yet well prepared to integrate such a model in a methodologically sound way by considering potential moderation and mediation of environmental factors – besides bedroom location - which play a decisive role in triggering or avoiding the effects.
3. EXPOSURE (“soundscape”) ASSESSMENT

3.1 The need for critical evaluation of noise propagation modeling

Typically, engineering methods and the resulting noise maps are validated against long term noise measurements in “simple” open area propagation conditions and rarely in complex residential settings where most people actually live (15). It is likewise rare to evaluate noise propagation maps produced by different methods. It has been shown, however, that the variation between models can be substantial (16–19) and this needs to be seriously taken into account (20,21).

However, in the new area of big data this step of validation is usually omitted but critical (22,23). The available noise maps are simply taken as they are. In addition, epidemiologists arriving from the field of air pollution use now often simple land use regression methods to assign noise exposure values to the subject’s residential address. Also these methods are usually not validated against noise effect indicators. In a recent study on annoyance a valid exposure response could only be demonstrated with road traffic but neither with aircraft nor rail traffic (24). This may be due to too simple assumptions about the relation between noise and air pollution (25). As these large-scale studies receive a lot of weight in systematic reviews caution is needed and the assessment criteria for noise assignments need to be strengthened.

As there is great progress among some noise propagation modeling groups (26–29) these new developments should be evaluated not only against annoyance but also against more serious health effects such as CVD.

3.2 From exposure modeling validation to exposure effect modeling evaluation

In the ALPNAP study we employed two (in the case of highway noise even three) noise propagation methods which opened the unique opportunity to evaluate the modeling in the framework of the actual noise – health relations. Thus the effects of different noise modeling techniques on the estimation of noise associated health impacts could be directly assessed. We have initially observed larger differences with annoyance (30) compared with hypertension (31).

However, recent analyses for the WHO-evidence review on CHD effects of noise revealed larger differences concerning angina pectoris exposure response relationships (Figure 3, Figure 4) when two different noise propagation methods were used to model highway noise.
Figure 3. Angina ever (harmonoise modeling) 
Figure 4. Angina ever (ISO-variant modeling) 
Adjustments made for age, sex, education, occupation, area, health status, smoking, coping, anger, depression, hypertension, family history of hypertension, sleep quality, PM10

On the other hand – using MITHRA for main roads sound modeling Lden and Lnight exposure response curves did agree quite well (Figure 5 and Figure 6). An important aspect of this case is that the specific effect threshold is lower for the night exposure than for the Lden model. Separate modeling of the nighttime is required to get insight into the most vulnerable period of noise exposure.

While there are a few studies finding some evidence for the importance of night-time exposure in hypertension (32,33) not much data about sensitive times for ischemic heart disease is available. Separation of the night-time exposure window is important as experimental and field evidence for short-term night effects on blood pressure (34), dipping (35) and endothelial function (36) exist.

3.3 The next steps: consideration of special noise exposure characteristics

Fluctuation and emergence in sound exposure is a topic investigated only in relation to annoyance. From this research we know that the threshold level where human response is triggered is lowered by strong fluctuations or emergence (37). In this context the amount of background noise needs to be considered (38). Recently, a Swiss publication proposed a simple “intermittency” indicator which could be helpful in noise studies to characterize and differentiate between sources (39).

Another issue hitherto neglected is consideration of sound sources with high low frequency content. A study in the Netherlands has shown that even along motorways with a higher percentage of heavy vehicles this should not be neglected (40). The study found for 59% of the households adjacent to the motorways dBC-A levels ≥ 15 dB. Note: in such cases the A-weighted noise indices do no longer
show a valid picture in terms of affectedness reported by exposed subjects (41,42) as noise abatement actions (walls, windows, building structures) are not as effective as for higher frequencies (43). Audible rattle around airports is a well known example (44). Modulated sounds are an additional problem (45). Psychoacoustic measures (loudness etc.) provide more appropriate means to resemble the human hearing and response (46–49).

3.4 The next steps: consideration of combined noise exposure (mixed source exposure)

In most studies single sources are studied and typically not much information about the other sound sources is available. In reality we are surrounded by more than one sound source. Recent data from the environmental agency in Germany revealed 33% of people are exposed to 2 or 3 sources – 11% even to 4 or 5 sound sources. From research into annoyance it is known that in cases where one sound source clearly dominates it may be sufficient taking into account the dominant source only.

Again, this research topic is investigated only in relation to annoyance (50,51).

3.5 The next steps: consideration of noise exposure in combination with other agents

Several reviews have made clear that in transportation research on effects of noise you have to consider in addition air pollution, vibration and general deterioration of the environment (52,53).

Recently, Sørensen found the strongest association with ischemic stroke for a combination of high noise and high NO2: notable: interactions were not statistically significant and fatal stroke was positively associated only with air pollution but not with traffic noise (54).

This means first: adjustment for air pollution is not the right strategy to detect combined effects and second: it is not enough to explore combined effects only by classical multiplicative interactions but analyses should also screen for lower type interactions.

4. MODERATION BY PERSON AND CONTEXT

4.1 Personal factors (“psych- and physioscape”)

In CVD noise epidemiology the basic factors mostly considered in adjustments are age, sex, family history, noise sensitivity. In only a few studies additional information is available about occupational noise and risk factors, other lifestyle factors (smoking, physical activity etc.), health status or subjective annoyance due to the investigated sound sources.

But what if these factors interact with the exposure? The majority of studies do not test for potential effect modification. Even for basic factors like age and sex it would make sense to test systematically. Therefore, in systematic reviews it is mostly impossible to include the few studies in pooled analyses and information is lost.

A rare exception is the large longitudinal study on the relation of traffic noise with stroke (55). A statistically significant interaction with age was found: cases over 64.5 years showed a significant association with stroke while no association was observed for those below 64.5 yrs.

Another candidate is family history of hypertension. It is well known from early experimental and field studies that persons with a family history of hypertension react stronger to noise exposure (56,57).

In a reanalysis of data from the ALPNAP-study for the evidence review we tested for interaction in a traditional fashion: no statistical significance was observed. However, when we analyzed the sample separately with and without family history of hypertension the following results were observed (Figure 7 & Figure 8).
Adjustments made for age, sex, education, occupational noise, area, health status, noise sensitivity, annoyance, and distance to highway (no change with PM10).

In the context of another not well supported relation of noise and measured blood pressure a few questions come up after 2 cross-sectional studies showed significant effects of traffic noise on hypotension (58,59). What, if a significant fraction of the population experience a reduction of blood pressure under noise exposure? (see Figure 9) Obviously, a lot of misclassification would enter the noise blood pressure analyses. In fact, there is reasonable evidence to assume that this may be the case.

Although, earlier experimental studies on the effects of noise on blood pressure revealed often inconsistent results. While some studies found increases, others reported no change or even decreases. However, note: these studies used high sound intensities (>75 dBA). Interestingly, the observed discrepancies were never subject of serious discussions in the literature. A carefully conducted field experiment at the Environmental Agency in Berlin under naturalistic working conditions with ecologically valid traffic noise exposure (60 dBA vs 50 dBA) of longer duration (6:30 hrs) was the only study, where the full distribution of noise effects on blood pressure was reported (60). We used this information to generate the graphic from the original Table (Figure 9).

In the graph you recognize the full distribution of the blood pressure response of all participants. On the X-axis you see the mean difference of systolic (left) and diastolic blood pressure (right) between the noise exposure day and the control day. The spread is substantial on both sides of the
Null (the no response) and a large portion of the participants show lower blood pressure on the noise exposure day.

4.2 Contextual factors

At Internoise 2012 we have discussed the importance considering contextual factors for the correct application of exposure response curves in annoyance assessment at smaller scales (61).

4.2.1 The ecological level (“enviroscape”)

This scale is concerned with the potential effect of structural differences at a regional level. This covers diverse indicators such as population density, mesh size of traffic in cities, area layout, climate, nature and topography, land use variables, economic activities and work structure on the exposure side. On the health outcome side differences in the prevalence of diseases, health care quality and access. These indicators show often great variation at regional/local or at the level of the work or living environment.

4.2.1.2 Occupational environment

An ideal example for the work environment is the case-control study by Selander about the joint effects of road traffic and occupation noise and job strain revealed a stronger relationship with 2 (OR 1.57, 95% CI 1.24–1.98) or 3 factors (OR 2.27, 95% CI 1.41–3.64) compared with only one exposure (OR 1.16, 95% CI 0.97–1.40). It demonstrates also the importance of interactions in the field.

4.2.1.1 Community level

Figures 10 and 11 show substantial differences in the prevalence of cardiovascular conditions at the community level. Methodologically speaking: the health outcome opportunities are quite different across the participating communities. The noise exposure distribution across the communities also differed. Likewise varied the hypertension treatment prevalence and the occupational noise exposure significantly in these communities. It is therefore interesting that we did not find a significant noise hypertension relationship – but surprisingly found a relation with hypotension.

![Hypertension prevalence by community](image1.jpg)

Fig. 10. Hypertension prevalence by community

![Hypotension prevalence by community](image2.jpg)

Fig. 11. Hypotension prevalence by community

4.2.2 Housing, bedroom position and window behavior

For many studies this important information is not available. Missing information on these critical data can contribute substantially to exposure misclassification as the difference in exposure between front- and rear-side exposure may be up to 20 dBA. Therefore, it is not surprising to see studies having considered this information show significant (or stronger) relationships with hypertension (10,13,62,63).

4.3 Other factors to be considered in future studies

In a reasonable way we need to include also the important variables from social (64,65) and ecological epidemiology (66) in future studies. The social network and support studies (67,68), the
positive effect studies from landscapes with high quality green and blue space (69,70) and good soundscapes (71) providing restorative resources to buffer adverse environmental conditions (14,72,73) All rival with the “noise space” as potential contributors to health.

5. CONCLUSIONS

To compare the potential size of the adverse effects of noise on humans at the policy level the calculation of disability adjusted life years (DALYs) is required and dependent on high quality systematic reviews.

To allow valid health impact assessments of noise exposures also at lower levels of administration (regional or local) the inclusion of context information on both sides of the equation (exposure and health related) at the structural, the ecological and the social level is required to deliver unbiased health impact assessment with sustainable effects on health.

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