Cardiovascular effects of environmental noise: Research in Austria

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Abstract
Cardiovascular effects of noise rank second in terms of disability-adjusted life year (DALYs) after annoyance. Although research during the past decade has consolidated the available data base, the most recent meta-analysis still shows wide confidence intervals — indicating imprecise information for public health risk assessment. The alpine area of Tyrol in the Austrian part of the Alps has experienced a massive increase in car and heavy goods traffic (road and rail) during the last 35 years. Over the past 25 years small-, middle-, and large-sized epidemiological health surveys have been conducted — mostly within the framework of environmental health impact assessments. By design, these studies have emphasized a contextually driven environmental stress perspective and placed the study of noise-related adverse health effects in a broader framework of environmental health, susceptibility, and coping. Furthermore, innovative exposure assessment strategies have been implemented. This article reviews the existing knowledge from these studies over time, and presents the exposure-response curves, with and without interaction assessment, based on standardized re-analyses and discusses it in the light of past and current cardiovascular noise effects research. The findings support relevant moderation by age, gender, and family history in nearly all studies and suggest a strong need for consideration of non-linearity in the exposure-response analyses. On the other hand, air pollution has not played a relevant role as a moderator in the noise-hypertension or the noise-angina pectoris relationship. Finally, different noise modeling procedures can introduce variations in the exposure response curves, with substantive consequences for public health risk assessment of noise exposure.

Keywords: Traffic noise, blood pressure, hypertension, angina pectoris, noise, exposure-response relationship, effect modification

Introduction
Through its geographical position in central Europe, Austria has experienced transit-traffic since Roman times. Since the early seventies the Austrian part of the Alps has experienced a massive increase in car and especially in heavy goods traffic (road and rail). Currently, about 32% of the population is exposed to road noise levels ≥ 60 dBA and 60% ≥ 55 dBA. Even as the increase occurred foremost on the road, the first complaints were issued about highway traffic by the end of the seventies. In 1984, we started a first pilot study in a small community (‘noise village’), which was surrounded by a highway and an associated toll station, to explore the problem.¹⁻² Later, the intentions to move heavy goods transport from road to rail led to an increase of heavy rail traffic during the night, resulting in higher noise levels than through the daytime (+3 dBA). Therefore, multi-community health surveys followed, to study the supposed adverse effects of noise and air pollution in those Alpine valleys where the transit-traffic was on the increase or where large rail infrastructure projects required environmental health impact assessment (EHIA).³⁻⁷ In these studies we emphasized a contextually driven environmental stress perspective⁸ and placed the study of noise-related adverse health effects in a broader framework of environmental health, susceptibility, and coping.⁹⁻¹¹

Cardiovascular effects of noise rank second in terms of disability adjusted life years (DALYs) after annoyance. Although research during the past decade has consolidated the available data base the most recent meta-analysis — based on road traffic noise studies — reveals wide confidence
The conclusions about the effects of road traffic noise rest mainly on the Caerphilly and Speedwell and Berlin studies. Insufficient data are available on the potential cardiovascular effects on account of railway noise exposure.

In earlier articles we have suggested that the large variability of noise effects observed, is partly due to the strong moderation and / or mediation by the context where the noise exposure occurs and partly due to the effectiveness of the coping strategies. Related to this argument, factors such as regional differences in the underlying population morbidity structure (susceptibility and health status) and the overall exposure load (at work, environment, socioeconomic) may in addition be responsible for the often observed heterogeneous results. A specific argument is related to the potential difference in the experienced noise exposure, in the Alpine areas. This may be either related to the perception of noise (perceived exposure contrast, signal-to-noise ratio) or to the inability of classical noise indicators to catch the difference in the meaning of noise exposure, which is known to modify bodily responses. Eventually, since longitudinal studies are sparse and difficult to conduct in a continuously changing world with high mobility, the required latency time for the development of noise-associated cardiovascular effects has not yet been established. Thus, the sampled population experience in the studies may differ in terms of cumulative time to the effect, and reflect only the different power to detect effects, apart from the power provided by the sample size.

This article aims to share and integrate the existing knowledge from the Tyrol studies with a wider audience. The first step would be to make the analysis available, which has not yet been published – or if so – not in English. Second, to summarize the main results observed over a period of 25 years. Third, to add re-analysis based on the existing datasets, which would contribute to some of the still pertinent questions in cardiovascular noise effects research. For this purpose updated models were created to further evaluate the interaction effects and to gain a deeper insight into the meaning of effect modifiers over time.

Methods

Area, sample selection, and recruitment

Both the areas of investigation, the Unterinntal and Wipptal, are located along the most important European North–South-access route for heavy goods, over the Brenner Pass. The heavy goods traffic over the Brenner has tripled within the last 25 years and the fraction of goods moved onto the road has substantially increased (up to two-thirds). The areas consist of small towns and villages, with a mix of industrial, small businesses, tourist, and agricultural activities. The primary noise sources are highway and railway traffic. In addition, densely trafficked main roads are of importance. These roads link the villages and towns and act as access roads to the highway.

Over the years, the sampling strategies have been refined. In the early studies all the people of the representative villages of a certain age range (25 – 65 years or 25 – 75 years) were approached by interviewers. In the later studies a basic phone survey (15 – 20 minutes) was conducted, based on a stratified, random sampling strategy. The address base was typically stratified using GIS (geographical information system) data, based on fixed distances, to the major traffic sources (railway, highway, main road), leaving a common ‘background area’ outside major traffic activities and an area with exposure to more than one traffic source (‘mixed traffic area’). From these five areas, households were randomly selected and replaced in case of non-participation. Entry selection criteria were age range, sufficient hearing, language proficiency, and residency of at least one year at the current address. The participation was higher in the earlier survey (around 60%) and lower in the most recent survey (around 40%).

Noise exposure assessment

The earlier studies (Noise Village study, TRANSIT study) were based on the assessment of noise exposure on a short-term measurement network, with a central long-term recording unit. Subsequently, the individual noise exposure assignment was done in 5 dBA classes, based on these measurements, and local correction by noise expert judgments for each home. No distinction was made between the contributing sources. In the Noise Village study there was a main road and a highway with a toll station. In the TRANSIT study in two of the five communities rail exposure was also of equal importance.

In the lower Inn valley EHIA-studies for the ‘Brenner Eisenbahn Gesellschaft’ (BEG studies: UIT-1, UIT-2) noise exposure (dBA, L eq) was assessed by modeling (utilizing ‘Soundplan™’ software) and the calibration by measurements from 31 sites, according to Austrian guidelines (OAL Nr 28+30, ONORM S 5011). Based on both data sources approximate day–night levels (L eq) were calculated for each respondent and also noise source, to facilitate comparison with the typical dose-response data. Exposure and survey data were then linked via GIS.

In the latest study (ALPNAP study), railway noise emission was extracted from a typical day of noise immission measurements at close distance to the source. For highway traffic the yearly average load (light and heavy vehicles) was
combined with an average diurnal traffic pattern. For main roads, the available traffic frequency data were supplemented with additional traffic counting. Noise emission from road traffic was calculated with the help of the Harmonoise source model. In addition, micro-simulations of the traffic flow were conducted with Paramics (Quadstone, www.paramics-online.com) to obtain optimal individual vehicle characteristics (speed and acceleration). Within the ALPNAP study for the first time two noise calculation procedures were implemented. ‘Bass3’, the propagation model developed by INTEC uses a three-dimensional object precise beam tracer, gradually becoming a stochastic ray tracer at a larger distance from the source, to determine the possible propagation paths. The sound propagation phenomena are included in an ISO9613-2 comparable manner. The model includes up to four reflections and two diffractions sideways. ‘Mithra-Sig’ is the implementation of the French NMPB-Routes-96 procedure by the Centre Scientifique et Technique du Bâtiment, Lyon (CSTB), of the current interim engineering methods recommended by the Environmental Noise Directive (END). It uses a 2.5 dimensional tracing for a visibility check. An extensive noise monitoring campaign was available to check the validity of these simulations. At 38 locations, the sound levels were recorded for over one week during winter (October to January) and during summer (June to August). In addition, the predicted sound pressure levels resulting from parabolic equation (PE)-modeling have been evaluated against these long-term measurements. Indicators of day, evening, night exposure, and \( L_{den} \) were calculated for each source, as also the total exposure at several points on the building facade of the survey participants. In the present analyses, \( L_{den} \) at the façade most exposed was utilized.

**Air pollution exposure assessment**

In the BEG-studies, exposure by air pollution was assessed by a Swiss expert group (OEKOSCIENCE AG, Quellenstrasse 31, CH-8005 Zürich) who had long-term experience in monitoring and calibrating air pollution exposure in the alpine areas, with special consideration of meteorological conditions. An adapted Gaussian propagation modeling procedure was used. In the ALPNAP study, the annual means for NOx, NO\(_2\), and PM\(_{10}\) were calculated for an area 27 km (W–E) \( \times 23 \) km (N–S) east of Innsbruck). For these, air quality assessment of about 300 flow fields were calculated with the meteorological model GRAMM (Graz Mesoscale Model), for each domain. The model system used special algorithms to account for low wind or calm conditions. Traffic emissions were modeled using the network emission model NEMO. For each flow field a dispersion simulation was calculated with the Lagrangian particle model GRAL for horizontal resolutions of 10 \( \times 10 \) m\(^2\) and a vertical resolution of 2 m. The model system used special algorithms to account for low wind or calm conditions. Each run was weighted due to its meteorological classification and frequency. Thereafter, annual, summer and winter means were calculated by post processing and weighting the numerous dispersion calculations. Within the ALPNAP study the simulation results were compared with seven air quality stations located in the Inn Valley. The background values within this study were height corrected according to Seinfeld & Pandis. Calculated NO\(_x\) and PM\(_{10}\) values for each of the participant’s home were assigned by GIS.

**Questionnaire information**

The questionnaire covered the sociodemographic data, housing, satisfaction with the environment, general noise annoyance, attitudes toward transportation, interference in activities, coping with noise, occupational exposures, lifestyle, reported sensitivities, health status, prevalent diseases, and intake of medications. The telephone interview took about 15 – 20 minutes to complete. Education was measured in five grades (basic, skilled, labor, vocational school, A-level, University degree). The last two grades were combined in the category ‘higher education’. Noise sensitivity was queried with a five-point Likert-type question. ‘High sensitivity’ was defined by the two upper points on the scale (4 and 5). Health status was judged on a standard five-grade scale (1 to 5). The three poorest grades were combined as ‘less than good’ in the analysis. Active and emotional coping was assessed by a sum score, based on 13 items. The area characteristics (urban, suburban, and rural) were defined by residential pattern and community size.

**Statistical analysis**

The statistical analyses were carried out with ‘R’ version 2.10. Exposure-effect curves were calculated with extended logistics or ordinary least square regression methods using restricted cubic spline functions to accommodate for non-linear components in the fit, if appropriate. In the results section the \( P \) values were reported for both the linear (‘lin’) and non-linear (‘nlin’) estimates. The non-parametric regression estimate and its 95% confidence intervals (CI) were based on smoothing the binary or continuous responses — in the case of binary response taking the logit transformation of the smoothed estimates — using the contributed R packages ‘Design’ and ‘Hmisc’. The criteria for the statistical consideration of interactions were relaxed, as departure from additivity could be of relevance in a public health context, when involved exposures and outcomes were prevalent. It had also been demonstrated that selected studies could profit in terms of power by raising the Type I error rate from 5 to 20%, to detect interactions that would otherwise remain uncovered. This error rate was applied to report the ‘relevant effect modification’. Table 1 shows the major characteristics of the different studies.

**Results**

Exposure-response relationship without consideration of effect modification.
### Table 1: Study characteristics

<table>
<thead>
<tr>
<th>Study</th>
<th>Year / season</th>
<th>Areas</th>
<th>Traffic sources</th>
<th>Age range</th>
<th>N</th>
<th>Methods</th>
<th>Participation</th>
<th>Design / Sampling</th>
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<td>Highway</td>
<td>25 – 64 years</td>
<td>197</td>
<td>Interview</td>
<td>77</td>
<td>Cross-section of the whole Community</td>
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<td>1 full community</td>
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<td>25 – 69 years</td>
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<td>Interview</td>
<td>62</td>
<td>Cross-sectional cohort (Schönberg i-ii)</td>
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<td>25 – 64 years</td>
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<td>Interview; environment, Coping, behavior, Health, medications</td>
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<td>Cross-sectional</td>
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<td>Highway</td>
<td>18 – 74 years</td>
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<td>Interview; environment, Coping, anthropometry 4 blood pressure readings</td>
<td>51</td>
<td>Cluster sampling: 500 m Sampling radius around</td>
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<td>Highway</td>
<td>20 – 74 years</td>
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Statistically significant straight noise-effect relationships with basic adjustment of relevant confounders (no interactions) were only observed in selected analyses, with cardiovascular endpoints. In most analyses the noise-effect relation was only statistically significant in subgroups or in those with a predefined combination of susceptibility factors (mainly gender, age, family history of disease, and behavioral risk factors). To illustrate this point, first, only the exposure response relations of all studies that resulted from regression models with adjustment for standard factors without IA-terms were described. Note: The graphs show predicted probabilities based on modeled — not observed data.

In both the Noise Village study and the TRANSIT study no relevant relationship (main effect = ME) between noise and systolic blood pressure (SBP) could be observed. The UIT-1 study showed a slight linear relationship of hypertension with sound level, mainly in the older group [Figure 1]. The UIT-2 study exhibited a relationship of SBP with noise only in men at age 60. In the ALPNAP study, in both the hypertension and angina models, without interaction terms, a slight curve leveling off is visible around 60 dBA, L_{den} [Figure 2]. Only in the UIT-1 study (basic hypertension model) the sound level increase between 50 and 60 dBA, L_{den}, was significant (OR = 1.38, CI = 1.03 – 1.86). Furthermore, distance to the main road was a significant factor (P = 0.007). The companion models considering interactions are described a little later in the text, under the specific moderation heading. Interactions (IA) that were not significant in classical statistical terms were labeled as relevant effect modifications. In some studies we also described the relationship of distance to a relevant source. Note – the meaning of the air pollution models did not change when interaction terms were included.

**Exposure-effect relationship with effect modification**

**a) Noise annoyance**

It has been argued that subjective reports of actual perceived exposure are needed, in addition to objective indicators of noise, to better assess the potential adverse effects.\(^{12,34,35}\) Due to the established noise-stress-CVD hypothesis of action, it would also seem reasonable to find noise-CVD associations, particularly among those who showed a particular disturbance or interference by noise, either during the day (impairment of concentration or performance) or night time (impairment of sleep). Only a few studies have tested these hypotheses.\(^{14,36}\) Overall, our data did not reveal any significant support to the simple hypothesis that higher noise annoyance is associated with higher cardiovascular disease outcome. To the contrary, from our early work in the Noise Village study we consistently observed the opposite in our SBP or hypertension relationships with traffic noise. Reporting higher annoyance (very much versus not at all) was significantly linked with lower SBP (-5.83, CI = -8.99 to -2.68 mmHg), adjusting for age, sex, body mass index (BMI), education, cholesterol, family history, and window behavior in the TRANSIT study.\(^{3}\) Likewise in the Noise Village study, the prevalence of hypertension was higher [Figure 3] in those reporting less interference by noise in their daily life (IA noise*interference \(P = 0.06\)).

We explained this finding – which was unexpected at first glance – with the much higher adaptive efforts that higher annoyed subjects invested to reduce noise exposure compared with less annoyed subjects.\(^{5}\) This supports a protective effect of certain active behavioral coping strategies – induced by higher annoyance. In the later studies, however, these associations of both coping activities and annoyance with blood pressure were weaker or no longer statistically significant. It remains to be speculated whether

\[\text{Figure 1: Hypertension diagnosis/treatment: Exposure-response for overall sound exposure (road and rail traffic) by age and sex. Adjusted for health status, weather and noise sensitivity, work noise and vibration, distance to highway and rail – [UIT-1 study, 1998]}\]

\[\text{Figure 2: Angina pectoris: Exposure-response for highway sound exposure by age. Adjusted for BMI, family history, known hypertension, education, health status, depression, smoking, occupation, coping, anger, PM}_{10} - [ALPNAP study, 2006 (MITHRA)]\]
the health gain of active coping fades away over time when the troubling noise exposure situation persists. Alternatively, it could be that annoyance reporting habits changed over time or coping became more common. Thus, the power to detect health gains of protective behavior diminished over time.

b) Bedroom location
In the Tyrol studies we did not consistently observe the improved exposure effect relationships by either introducing bedroom location or an indicator of sleep disturbance as independent factors in the regression models. However, some models did improve. For example, participants with bedrooms facing toward a quiet yard [Figure 4] did show a clear trend toward a reduction in hypertension diagnoses in the ALPNAP-study (OR = 0.78, CI = 0.59 – 1.05). In the UIT-2 study a relevant interaction (IA) with bedroom location (IA: \( P = 0.18, \) ME\(_{\text{bedroom}}\): OR = 2.01(1.09, 3.78)) was observed when the distance to the main road was considered as an additional source parameter (ME\(_{\text{distance}}\): \( P = 0.02 \)) in a non-significant rail noise model [Figure 5]. The interaction of bedroom location in the highway model was similar, but statistically not relevant (IA: \( P = 0.31 \)) and also the single main effect (ME) of the bedroom location was less precise (ME\(_{\text{bedroom}}\): OR = 1.77, CI = 0.72, 4.39, ME\(_{\text{distance}}\): \( P = 0.08 \)). In addition, the presence of night disturbance by rail did exhibit a further main effect in both the rail (OR = 2.24, CI = 1.21 – 4.17) and highway models (OR = 1.98, CI = 1.08 – 3.62).

c) Length of exposure
Duration of living at the current home may be another candidate variable representing a more homogeneous group with longer latency times for potential health effects. Bluhm \textit{et al.}, observed a stronger association between noise and hypertension in subjects with a longer period of residence (> 10 years).\[^{[37]}\] We found no significant effect of longer duration on the overall noise-disease association in the ALPNAP-study, in the regression model. The duration of living was, however, strongly associated with older age (IA: \( P = 0.12 \)) and house type (≥ 20 years in single homes 52%, versus 21% in apartment blocks). Thus, it is difficult to disentangle, especially when a large proportion of the sample has such a record of longer living (66%) or single housing (56%). However, when an extreme comparison was made with a strong family history of hypertension in the model adjustments, duration of living for ≥30 years at the present address was significantly associated with hypertension (OR = 1.68, CI = 1.07 – 2.66) against < 8 years of living at that address. The comparison of living for ≥ 30 years versus < 30 years in the UIT-1 study revealed quite clear results supporting the theory that the length of exposure was an important variable [Figure 6]. In these analyses, the distance to the road was considered as exposure and heart problems as the outcome. When distance was replaced by the overall sound level, duration of living at the current home was significant (\( P = 0.04 \)). However, no sign of effect modification by noise level was evident.

d) Age
Hypertension: The re-analyses of the Tyrol health studies revealed substantial evidence for effect modification by age and gender on the relationship between noise and indicators of hypertension. Previously, in the small noise village study, we observed supporting evidence for a noise effect only in those at a higher age compared to participants at a lower age, with both dichotomous and continuous blood pressure outcomes. The interaction with noise level was statistically significant (IA: \( P = 0.01 \text{lin}, \, P = 0.02 \text{nonlin} \)). In the TRANSIT study, the age-noise level interaction on treated hypertension was only significant in men (IA\(_{\text{treat}}\): \( P = 0.03 \)) [Figure 7]. In the

\[\text{Figure 3: Systolic blood pressure > 160 mmHg: Exposure-response for highway sound exposure by annoyance rating. Adjusted for age, sex, BMI, education, occupational noise, window behaviour - [Noise Village study, 1984]}\]

\[\text{Figure 4: Hypertension: Exposure-response for highway sound exposure by bedroom location. Adjusted for age, sex, BMI, family history, education, health status, duration of living, IA level*age, level*history, level*health status – [ALPNAP study, 2006]}\]
Figure 5: Hypertension/treatment: Exposure-response with distance to main road (rail model) by bedroom location. Adjusted for age, sex, BMI, family history, health, health worry, education, weather sensitivity, work noise, nightshift, heart medication, heart rate, night disturbance rail, level rail – IA distance*bedroom, health*worry*, weather*heart rate, work noise*nightshift – [UIT-2 study, 1998]

Figure 6: Heart problems: Exposure-response with distance to highway by duration of living at age 60. Adjusted for sex, education, hypertension, weather and noise sensitivity, coping, region, NO₂, overall sound level – [UIT-Istudy, 1998]

Figure 7: Hypertension: Exposure-response for overall sound exposure (road and rail traffic) by age in men. Adjusted for BMI, family history, cholesterol, education, noise sensitivity, IA sound level*age, sound level*sensitivity – [TRANSIT study, 1989]

Figure 8: Angina pectoris: Exposure-response for overall sound exposure (road and rail traffic) by age. Adjusted for sex, BMI, family history, cholesterol, education, health, smoking, fat intake, exercise, nightshift, community – [TRANSIT study, 1989]
2 study, interaction patterns due to gender \((IA_{sex}; P = 0.18\text{ lin}, 0.398\text{ nonlin})\) occurred when adjustment for known hypertension was included. In the ALPNAP-study the effect of modification due to age was stronger – the noise-sex interaction, however, was of minor importance. In summary, we observed a stronger, although not always a significant effect of the noise level in men compared to women. In addition effect modification due to age was present and often enhanced the overall effect.

Heart disease: Neither in the TRANSIT nor in the ALPNAP study did we find an indication of relevant effect modification due to gender on the relationship between the noise level and heart disease \((IA: P = 0.6)\). In both the studies, the prevalence of angina pectoris was slightly higher among men across noise levels.

g) Education

Education was associated with both dichotomous and continuous blood pressure outcomes in the Noise Village study. The interaction of education with the noise level was evident in both sexes – but only relevant in the older age group (\(\geq 45\) years) \((IA: P = 0.20)\). The power \((N = 174)\) was limited to test interactions. In the TRANSIT and the UIT-1 studies, education was not significant overall, due to social differences between the studied communities. In the UIT-2 study, a significant effect of education on systolic blood pressure was found (lower SBP in subjects with higher education compared to lower education: mean adjusted difference: -3.90, CI = -7.79 to -0.01 mmHg) – with no relevant signs of interaction with noise level. In the ALPNAP study, this trend was reversed – but there hypertension diagnosis or treatment was the outcome. This might be due to differences in the detection and treatment of hypertension in general practice.

h) Hypertension

In the TRANSIT study \([Figure 11]\) we observed a highly significant impact of known hypertension on the prevalence of angina pectoris \((P \leq 0.0001)\). However, no relevant interaction with the noise level was evident. Similar results were obtained in the ALPNAP study with respect to angina pectoris.\(^7\) Subjects with pre-existing hypertension did exhibit a steeper increase in prevalence between noise levels

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**Figure 9:** Systolic blood pressure: Exposure-response for highway sound exposure by age and sex. Adjusted for BMI, education, occupational noise, noise annoyance, IA sound level* age, level*sex, level*annoyance, level*occ. noise – [Noise Village study, 1984]

**Figure 10:** Hypertension treatment: Exposure-response for highway sound exposure at age 60 yrs by family history. Adjusted for age, sex, BMI, education, health status, duration of living, house type, IA sound level*age, level*history, level*health status, age*duration of living – [ALPNAP study, 2006 (sound propagation: MITHRA)]
of 50 and 60 dBA (OR = 2.23, CI = 1.10 – 4.52), but no statistically relevant effect modification of hypertension on the relationship between noise and angina pectoris was observed.

i) Depression
In the TRANSIT study a borderline significant association between the prevalence of depression and the prevalence of angina pectoris was found. No interaction with the noise level was present. Although there was no association with blood pressure or hypertension in the ALPNAP study, we found a significant difference between people who suffered from depression and the probability of an angina pectoris diagnosis (OR = 2.06, CI = 1.08 – 3.94). There was, likewise, no relevant interaction with the noise level.

j) Air pollution
Hypertension: Support comes neither from the BEG studies nor from the ALPNAP study (not shown) for a significant positive effect of NO$_2$ or PM$_{10}$ on blood pressure or hypertension. Rather, opposite trends were observed. In addition no relevant signs of interaction could be found.

Angina pectoris: As in the case of hypertension, air pollution also did not affect the noise angina pectoris relation in the ALPNAP study. The observed inverse association is fully determined by the noise level.

k) Health status
A prospective study reported a stronger relation between annoyance and ischemic heart disease in middle-aged men with no prior disease at entry point.$^{[38]}$ A similar effect modification ($P = 0.16$) was observed in the presence of a strong noise*age interaction ($P = 0.02$) in the UIT-1 study, with respect to hypertension [Figure 12]. Only in those subjects with a good or very good health status a significant exposure effect relation was observed with regard to hypertension diagnosis or treatment. Also with respect to the three health status categories in the ALPNAP study, only for those with very good health status, a relation with noise level was found in men (IA: $P = 0.18$), but not in women. The ALPNAP study could not confirm such an effect modification of health status on the association between noise level and angina pectoris – although health status was a relevant predictor of disease when persons with excellent versus poor health status were compared (OR = 0.50, CI = 0.24 – 1.01).

l) Combination of risk factors
Hypertension: Using the final model (adjusted for the other factors) of the ALPNAP study, simulations were carried out to demonstrate the relevant effect modification of the most important risk factors (age, family history, health status) on the relationship between the noise level and hypertension, when the factors were varied, in terms of extreme group comparisons [Figure 13].

Heart disease: Likewise, we calculated the effect of two significant risk factors in the ALPNAP study, namely, hypertension and depression, on the probability of angina pectoris due to noise exposure (highway), for subjects of age 40 and 60 years, respectively [Figure 14]. A strong, effect modifying impact of the prevalence of these two diseases on the association between the noise level and the probability of developing angina pectoris was evident. However, the wide confidence intervals indicated the limitation when combinations with small subgroups were investigated.

m) Noise sensitivity
Hypertension: With respect to hypertension, in none of the studies carried out in Tyrol were positive relations with noise

Figure 11: Angina pectoris: Exposure-response for overall sound exposure (road and rail traffic) by age and hypertension. Adjusted for sex, BMI, family history, cholesterol, education, health, noise sensitivity, community, sound level*age – [TRANSIT study, 1989]

Figure 12: Hypertension diagnosis/treatment: Exposure-response for overall sound exposure (road and rail traffic) by age and health status. Adjusted for sex, annoyance, weather and noise sensitivity, distance to highway, main road, rail, IA sound level*age, sound level*health status – [UIT-1 study, 1998]
Lercher, et al.: CVD-effects of environmental noise: Austria

Heart disease: Different results were obtained for heart disease. In the TRANSIT study, angina pectoris showed a non-significant association with noise sensitivity ($P = 0.11$), but there was neither any interaction with sex (IA: $P = 0.98$) nor with the noise level (IA: $P = 0.72$). In the UIT-1 study noise sensitivity was not a significant predictor either. Instead weather sensitivity exhibited a strong, effect modifying impact (IA: $P = 0.11$) on the relationship between the distance to the highway and the prevalence of angina pectoris [Figure 16]. In the ALPNAP study a different pattern was found [Figure 17]. A strong interaction of sensitivity with sex (IA: $P = 0.01$) was found on the non-linear relationship between the sound level (highway) and the prevalence of angina pectoris (IA: $P = 0.16$). The sex-sensitivity-interaction showed a deviating pattern: although sensitive males consistently showed the highest disease rates with varying noise levels, sensitive females exhibited the lowest rates of angina pectoris. Note – the confidence intervals are wide.

Discussion

Exposure modifiers

The results from literature and the results of our studies over time suggest that there are important modifiers that may partly be responsible for the large variations found in the noise health effects research. Bluhm et al. suggest exposure misclassification as the main culprit. Specifically, their findings of stronger associations in persons with a longer length of exposure (years of residence) at the same address, not having triple-glazed windows, bedroom windows directly facing a road or living in single houses, do support this suggestion of potential over- and underestimation of true exposure. Caution is warranted, as effect modification due to the length of exposure may actually be caused by older age, which is typically confounded with it.

On the other hand a longer duration of time spent living at the same address may also indicate a certain time of exposure required to exert an effect. Therefore, studies with an insufficient proportion of people living longer at the same address (> 10 years) may lack the power to detect noise effects. In the Swedish study this proportion is high (44.5%). In the ALPNAP study, 50% had lived for at least 16 years and 25% at least 30 years at the same address. From meta-analysis of annoyance studies we know that confounding with age is a serious problem. In the Swedish study the age range went up to 80 years, which is an unusually high age range, with inclusion of a large proportion of elderly people.

Figure 13: Hypertension diagnosis: Exposure-response for highway sound exposure at age 60 yrs in poor health with a strong family history. Adjusted for sex, BMI, education, house type, annoyance, occup. noise, area, IA sound level*age, level*history, level*health status – [ALPNAP study, 2006 (sound propagation: INTEC)]

Figure 14: Angina pectoris: Exposure-response for highway sound exposure at age 60 yrs with hypertension and depression by sex. Adjusted for BMI, family history, education, smoking, sensitivity, coping, anger, NO$_2$ level, IA sound level*age, level*anger, level*hypertension, level*smoking, sex*sensitivity – [ALPNAP study, 2006 (MITHRA)]
very likely to have lived longer at the same address. Another Swedish study with a sample age range up to 75 years also found a stronger association with > 10 years latency – but only for men – while the previous study observed a stronger association in women.\cite{37}

We have seen protective effects (closing windows during night) as an additional modifier of exposure over and above the fact of having tightly fitted windows.\cite{5} Tightly fitted windows or closing windows during daytime alone did not show up as significant variables.

Selander et al. found an elevated association between road traffic noise and myocardial infarction in participants reporting noise annoyance, mostly in their bedrooms.\cite{36}

These findings can be interpreted in different causal pathway directions. First, in general, bedroom exposure is a better exposure indicator by reducing exposure misclassification, as most participants (nightshift workers as an exception) are actually in bed, while daytime exposure can vary substantially due to activity pattern and work exposure. Second, bedroom exposure is a causally relevant exposure, as sleep is affected and impaired sleep is a known risk factor for myocardial infarction in men and women.\cite{39-42}

Our studies support bedroom location or night disturbance as a potential moderator especially when additional noise sources contribute to the overall noise exposure. The effect seems, however, to depend also on the kind of source combination (rail – highway – main road). Therefore, bedroom location should be considered in the analysis design – but high variation is possible due to the actual feature of the specific source combinations.

Effect modification: Socio-demographic factors

a) Gender and age

Several studies observed differences in the effect of noise on cardiovascular outcomes by gender.\cite{37,43-49} Unfortunately, the found associations are not uniform – thus casting a doubt on their reliability and validity. Similar to what has been argued in air pollution studies – that effects found only in women may be related to their longer duration of exposure at daytime – thus asserting that this issue is related to exposure assessment rather than implying a different vulnerability. However, there is evidence of a gender difference of psychophysiological reactions toward stress. Generally, males are more susceptible to cardiovascular disease\cite{50} and women show greater resistance to stress between puberty and menopause.\cite{51,52}
In accordance with these findings the Tyrol studies did not provide support for a stronger effect in women. Instead, more often men did exhibit stronger effects in interactions with noise exposure and older age. When no effect modification by gender was observed, disparities in health care could be at work, like in hypertension treatment or angina pectoris diagnosis.\textsuperscript{[53-57]} The extra-large studies of de Kluizenaar \textit{et al.} and Bodin \textit{et al.} used their power to test whether certain age ranges did exhibit stronger associations between noise and cardiovascular outcomes.\textsuperscript{[58,59]} The findings of these studies suggested the middle age ranges (40 – 60 years) to be associated with hypertension, but not other ages. As the findings reported so far concerned middle-aged people, explanations were targeted to explain this finding. In view of the results from the Tyrol studies, where the elderly were consistently more affected, other explanations were necessary and equally plausible. As noise was viewed as a subtle, but chronic stressor, longer latency periods might be necessary to observe the effects. In a recent, large, semi-ecological medication study in the same study area, we reported very significant findings for an age group above 70 years.\textsuperscript{[60]}

The observed moderating effects of age or gender must be reviewed with caution, especially when only category-specific effects are reported and no exposure-effect relation is presented. Contrary to the argument of Bodin \textit{et al.}\textsuperscript{[59]} it can be stated that cohort analyses have shown that some classical cardiovascular risk factors lose their importance to predict cardiovascular mortality due to the survivor effect, and age gains importance.\textsuperscript{[61]} Hence, the longer stress-related risks can exhibit their subtle effects, they can gain importance with age. Eventually, the support for a positive association between noise annoyance and cardiovascular health is weak. At least in the Tyrol studies, more often, the opposite effect was observed.

\textit{b) Education}

The results show that when measured blood pressure was considered, lower education was consistently associated with higher blood pressure and higher prevalence of hypertension, based on the standard cut-off points. When reported hypertension was used, persons with a higher education exhibited a higher prevalence. This suggested a differential effect of health care on education. However, no significant effect modification with noise level was observed in any of the studies.

\textbf{Effect modification: Vulnerability factors}

\textit{a) Family history}

Family history of hypertension is an established major risk factor for the development of hypertension.\textsuperscript{[62-64]} In all studies (not available in UIT-1) family history was a significant contributor to either continuous or dichotomized blood pressure outcomes or treatment. Significant or a public health-relevant effect modification was observed with age and also with noise level. This supported the idea of higher vulnerability of people with a family history to noise exposure with a certain latency time. As more than one-third of the adult population in the ALPNAP study (41\%) showed some degree of family history (one parent) effect modification should be evaluated in all noise – hypertension studies.

\textit{b) Hypertension}

High blood pressure is a proven risk factor for cardiovascular diseases.\textsuperscript{[65]} Selander \textit{et al.} found a stronger association between road traffic noise and myocardial infarction in those with hypertension.\textsuperscript{[66]} Earlier or recent hypertension was also a significant contributor to angina pectoris in the ALPNAP and the TERW-89 studies. The moderation with noise level did not become significant.

\textit{c) Depression}

Depression is a known risk marker for cardiovascular diseases.\textsuperscript{[67]} Most studies have found depression to be significantly associated with mortality and / or cardiac morbidity – although the mechanisms underlying this relationship remain unclear.\textsuperscript{[66,67]} As dysregulation of the autonomic nervous system is a plausible pathway to disease, chronic exposure to noise is a possible candidate for effect modification.\textsuperscript{[68]} Both in the TRANSIT and the ALPNAP studies depressive symptoms or depression diagnosis were significant contributors in an angina pectoris regression model. Although some interaction between the noise level and the state of depression was visible in the figures, the power was too low to gain significance. However, the presence of both depression and hypertension showed a higher prevalence of angina at higher noise levels. We are not aware of other studies having evaluated depression as a possible moderator of the noise angina relationship.

\textit{d) Health status}

Health status is a general and reliable predictor of future morbidity and mortality.\textsuperscript{[69-72]} In all the studies (not available in Noise village and TERW-89) health status has made a significant contribution to the cardiovascular outcomes studied. Consistently, persons with a poor health status have shown higher starting levels of morbidity, and typically also, stronger slopes in the exposure response analysis. However, due to the generally lower disease levels, sometimes only people with an excellent or good health status exhibit a significant increase of either hypertension or angina pectoris with increasing noise levels in a dose-response fashion. Therefore, effect modification has not always been significant at classical error rates ($P < 0.05$), but still relevant in terms of potential public health significance ($P < 0.20$). We are only aware of one study having applied a similar approach, by using the disease status as a possible moderator of the noise exposure disease relationship.\textsuperscript{[68]}
e) Noise sensitivity

Noise sensitivity is known to be associated with higher symptom rates and medication consumption and is also a predictor of noise annoyance.[5,73] In recent times, work based on data from the Finnish Twin Cohort study reported an association of noise sensitivity with hypertension, after adjustment for noise exposure and other factors, in a multivariate model.[74] In a further study, a relation between self-reported noise exposure and cardiovascular mortality was observed in noise sensitive women – but not among men.[75] On the other hand, we consistently observed a negative relationship between noise sensitivity and hypertension as a health endpoint. This was in total contrast to the results of the Finnish studies (overview in Heinonen 2009[76]) showing several associations of noise sensitivity with hypertension and heart disease (morbidity and mortality) in noise-exposed female subjects. Overall, there was a non-significant trend in noise-sensitive subjects to show a higher prevalence of angina pectoris at higher noise exposure – but due to a significant interaction (sex*sensitivity, $P = 0.01$) this was not true in women. Thus, there was no good evidence for a relation of noise sensitivity in women in this study. The power to detect weaker associations was low in the ALPNAP study. However, the pooled Caerphilly and Speedwell analyses (only men) also did not observe a significant association (OR = 0.9) with noise sensitivity in a larger sample.[77] Note, the Finnish studies differed methodologically, as both noise exposure and noise sensitivity was obtained subjectively. Such a procedure was vulnerable to the known subjective bias from stress research.[78,79]

Measures of hypertension

In the noise literature, the clear diagnosis of hypertension (from medical sources or patient-remembered doctor diagnoses) is used in the analyses. Women are expected to show a lower prevalence of hypertension till the end of the fifth decade.[90] As medication use or type has not been confirmed in our studies, misclassification may be introduced by other unknown medications that may lower blood pressure. Furthermore, true awareness and control rates cannot be determined with the kind of data available. The literature reports awareness rates to be around 70%. Treatment and control rates are found to be around 60 and 30%, respectively, with lower rates in the elderly.[90,91] The experience with other surrogate measures of hypertension in our studies show the following characteristics:

- Blood pressure readings are less often significantly related to noise levels
- Dichotomizing blood pressure readings at higher cut-off levels (160 / 95 mmHg) are more likely significantly associated with noise exposure
- Treated hypertension is not a better indicator than doctor diagnosis or known hypertension
- When using treated hypertension we have not observed a gender difference in prevalence. This gender difference is consistently present when using remembered diagnosis or personal readings of blood pressure – indicating a lower prevalence of treatment among men than women. These findings are confirmed by large population surveys – but it seems that the male population is catching up.[81]

Time effects and latency to the effect

As these are series of cross-sectional studies over time, it is difficult to comment on time factors. However, there are some findings that contribute to the current scarce knowledge:

1. In the studies where we had two time frames in the retrospective question available (e.g., ‘hypertension diagnosis ever’ and ‘hypertension diagnosis during past 12 months’) the precise time framing ‘past 12 months’ did exhibit a stronger relation with noise than the more loose time framing ‘ever’. This finding may be explained by the concurrent measurement of exposure and outcome, and thus, reflect a higher precision in both. Alternatively, it could also give hints for time windows, where a certain proportion of the study population may exhibit noise-related effects.

2. Consistently, we found that persons at a higher age (>60 years) showed a firmer relation with noise than those at a lower age (~40 years). This relation was typically enhanced in the presence of an additional risk factor for the outcome under investigation (especially family history of hypertension). These findings suggest longer latency times and the need for other risk factors to be present in order to develop noise-related effects. The findings from our semi-ecological study, where significant relations with noise (antihypertensive prescriptions) were only found at an older age (>70 years), indirectly support longer latency times in general.[60]

3. Duration of living may not be a good approximation of the length of exposure, as it is strongly associated with age, housing factors, and education in our studies. From the social science literature it is long known and recently confirmed that people moving around less are better off in the light of various health outcomes and health-related behavior.[82-85] At least in our studies we have observed no significant difference in subset analyses with 10, 20 or 30 years of living at the same address. Although, utilizing the length of residence as a continuous variable with adjustments of age, housing, education, and health status does show a small increase in the odds for hypertension development when the contrast in duration is stretched (<8 years versus >30 years), it is not clear whether this indeed represents an independent finding. However, it also supports longer latency times, similar to the conclusions from the Speedwell and Caerphilly studies (>15 years).

Air pollution

Both noise and air pollution are often emitted by the same source, namely motorized traffic, and depending on the propagation conditions a wide range of correlations is
Such conditions open the possibility of confounding and make it difficult to disentangle the associated effects statistically. A large number of studies have shown stable associations of ambient air pollution with morbidity and mortality of cardiopulmonary disease. A smaller number of studies have reported associations with blood pressure or hypertension. As noise exposure is also associated with coronary heart disease (CHD) and hypertension, and only a few recent studies have actually considered both pollutants in the regression models, it remains an open question as to what contribution is made by which pollutant to which health outcome.

In both the UIT studies and the ALPNAP study high quality air and noise pollution propagation data were available for individual assignment. In none of the investigated health endpoints (angina, blood pressure / hypertension) a relevant or consistent relation with the studied range of air pollutants (NO$_2$, PM$_{10}$) nor a relevant moderation could be established. The large population-based Oslo Health Study (N = 18,770) was also unable to find a relation between the indicators of air pollution exposure and blood pressure. As we had two noise assignment options in the ALPNAP study, of which the ISO-assignments showed very low correlations with NO$_2$ (r = 0.12) and PM$_{10}$ (r = 0.09), confounding was highly unlikely to be of importance in this study. Although the MITHRA-assignments showed higher correlations (NO$_2$: r = 0.48 and PM$_{10}$: r = 0.39), the statistical importance of both the air pollutants and the noise variables did not change. In the BEG studies the highway noise to air correlations were stronger (NO$_2$: r = 0.63 and PM$_{10}$: r = 0.61).

**Methodological issues**

*a) Interaction assessment and non-linearity*

The investigation of moderation of the noise health relationship by public health relevant factors is a necessary requirement for the better understanding of the processes that determine the person-environment-health relationship. A single reporting of the average risk effects or associations from an entire population can often conceal the substantial variation that may occur in important subgroups (the elderly, women, and persons with positive family history of cardiovascular diseases, including high blood pressure). This deviation from the average risk can be even more pronounced when risk combinations are considered. If significant interactions are present, the meaning of the main effects becomes questionable. Unfortunately, most studies do not have the power to evaluate effect modification and interaction tests, in general, lack power. The relaxation of the significance criteria can sometimes help. The use of P values as the sole criterion is discouraged. However, caution is needed as additional mediation or residual confounding may distort the results or make it difficult to interpret. Therefore, only a biological plausible effect modification (based on prior knowledge) must be tested and a step-by-step procedure is advised — followed by detailed sensitivity analyses, to safeguard the conclusions. Eventually, there is a strong need to examine non-linear components in the exposure-response analyses. Substantial over- and underestimations may result without consideration.

*b) 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 not in complex residential settings, where most people actually live. The availability of having two (in the case of highway noise, three) noise propagation methods in the ALPNAP study opened the unique opportunity to evaluate the modeling in the framework of actual noise – health relations. Thus, the effects of noise modeling techniques on the estimation of noise-associated health impacts could be directly assessed. Although sometimes only marginal differences were noted even with complex effect modification, in other cases (e.g., angina pectoris) with only one method a significant exposure-response relationship was established, but not with the second method. This leaves behind a substantial amount of uncertainty. Therefore, a move from mere exposure modeling to exposure effect modeling is required to minimize the bias in public health risk assessment of the effects of sound on humans.

**Conclusions**

Because noise is not a strong risk factor per se, the specific context of the exposure, health predispositions, and the adaptability to this person-environment configuration determines whether effects occur. Specifically, the coping opportunities are of importance. If active coping (closing windows, bedroom on quiet site) is not feasible noise persists as a chronic stressor and with advancing age the effects may surface. As the effects of age and gender observed in noise effects research can only be prevented by reducing the intensity and the duration of exposure overall, residential areas should be considered as sensitive areas and the noise here must not exceed 55 dBA. This is in accordance with the results of the most recent studies. Finally, from the reported studies we have not been able to find support for a relevant role of air pollution. Neither with hypertension nor with heart disease a statistical significant association did come in reach. Eventually, no signs for a relevant moderation of the noise health relation by air pollution could be observed in the Tyrol studies.

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