Clinical research

Noise burden and the risk of myocardial infarction

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Aims Chronic noise exposure is associated with adverse pathophysiological effects and may contribute to the progression of cardiovascular disease. We, therefore, determined the risk of noise for the incidence of myocardial infarction.

Methods and results In a case–control study, 4115 patients (3054 men, 56 ± 9 years; 1061 women, 58 ± 9 years) consecutively admitted to all 32 major hospitals in Berlin with confirmed diagnosis of acute myocardial infarction were enrolled from 1998 to 2001 in the Noise and Risk of Myocardial Infarction (NaRoMI) study. Controls were matched for gender, age, and hospital. In standardized interviews, information was obtained on environmental and work noise annoyance. The sound levels of environmental and work noise were assessed using traffic noise maps as proxy and international standards for workplaces, respectively. In multivariate logistic regression models, the adjusted odds ratios of noise variables were determined. There was a marginally increased risk of myocardial infarction associated with annoyance by environmental noise in women (adjusted odds ratio 1.47, 95% confidence interval 0.95–2.25, \( P = 0.081 \)) but not in men, and not associated with annoyance by work noise. Environmental sound levels were associated with increased risk in men and women (odds ratios 1.46, 1.02–2.09, \( P = 0.040 \) and 3.36, 1.40–8.06, \( P = 0.007 \)) and work sound levels in men only (1.31, 1.01–1.70, \( P = 0.045 \)).

Conclusion Chronic noise burden is associated with the risk of myocardial infarction. The risk increase appears more closely associated with sound levels than with subjective annoyance. Further investigation of the gender-related risk of noise exposure may aid in improving prevention.

KEYWORDS Noise; Risk factor; Myocardial infarction

Introduction

Cardiovascular diseases cause over 30% of the worldwide mortality.\(^1\) The established cardiovascular risk factors account only for \( \sim 50\% \) of variance in the incidence of myocardial infarction.\(^2,3\) Further potential risk factors of cardiovascular disease include socio-economic variables including environmental and work conditions.\(^4\)

Noise is considered as one of the most severe sources of environmental and work place constraints. In a recent national health survey, 36% of the adult population in Germany reported to be exposed to noise from outside their home.\(^5\) Similarly, noise exposure at the work place is experienced by approximately one-third of the working population during at least 25% of the daily working time, according to European surveys.\(^6\)

Chronic noise exposure is associated with adverse pathophysiological effects and may thus contribute to the progression of cardiovascular disease.\(^7–9\) Most previous studies of the association between chronic noise exposure and myocardial infarction were descriptive, cross-sectional studies.\(^10,11\) In a subsequent meta-analysis, a significant association between work and air traffic noise exposure and hypertension was reported.\(^12\) In addition, there were a few cohort and case–control studies focusing on the association between noise exposure and hypertension or other cardiovascular conditions.\(^13–17\) In the Caerphilly and Speedwell studies, coronary heart disease was not related to the diurnal noise exposure in men.\(^18\) Another cohort study reported a significantly increased risk of myocardial infarction for subjects who experienced their work place as noisy.\(^19\) Finally, a previous smaller case–control study in Berlin\(^20\) suggested a marginally increased risk of myocardial infarction for subjects exposed to street noise at sound levels above 70 decibels.

However, these prior studies have investigated selected sources of noise and most of them were performed in men only. Furthermore, most of the studies had methodological limitations such as failure to sufficiently adjust for possible confounding factors. Therefore, the present NaRoMI (Noise and Risk of Myocardial Infarction) study was designed to determine the association between chronic noise burden and the risk of myocardial infarction in men and in women and to assess the risks of subjective annoyance by noise and objective sound levels of noise in the environment and at the work site, respectively.
Methods

Study design and patients

Using a case–control study design, patients were recruited from all 32 major hospitals in Berlin, characterized by a size of at least 200 beds and the presence of a coronary care unit. Cases were all patients consecutively admitted with a diagnosis of acute myocardial infarction according to the cardiologist-in-charge. Control patients were recruited from the departments of trauma and general surgery with one of the following diagnoses presumably not related to noise exposure: accidents, inguinal hernia, goiter, or colon disorder. A control was sought in the hospital every time a case was admitted. The controls were matched according to hospital, age (within 5 year categories), and gender, with a case–control ratio of 1:1 in men and 1:2 in women; on the basis of the patients’ information, a history of myocardial infarction was excluded in controls. Further inclusion criteria for all study subjects were residency in Berlin since at least the 5 preceding years and for at least 6 months per year, age below 70, and sufficient communication and language skills. Patients with deafness or hearing impairment were excluded from study participation.

Study organization and data collection

After obtaining the patients’ informed consent to study participation, a computer-assisted (C13) standardized interview was performed at the bedside on general wards, in case patients following the transfer from coronary care units. Medical students were recruited as interviewers and underwent initial standardized training regarding the interview technique and data protection procedures. During the study period, the interviewers were supervised in regular team meetings. Each interviewer covered two to four hospitals, surveying them in person or by telephone on average twice per week to monitor the admission of new patients with acute myocardial infarction.

During the interview, medical and sociodemographic variables were recorded, including family history regarding myocardial infarction (defined as parents or siblings with the disease), history of smoking, education, job situation, physical activity, and income. Noise sensitivity, referring to individual characteristics in reaction to noise, was determined on a scale from 0 (not sensitive) to 5 (very sensitive) according to the standardized 21-item questionnaire by Weinstein. Additional information was retrieved from the patients’ charts, including body weight and height, presence of diabetes mellitus, arterial hypertension, and hyperlipidaemia. Obesity was defined as body mass index ≥30 kg/m².

Subsequently, the data were transferred into a database (Microsoft ACCESS) and checked for plausibility, with subsequent correction by mail or telephone contact with the patient if necessary. In an initial pilot phase, the feasibility of computer-assisted interviews and the patient recruitment procedures were established and the interview questionnaire tested for clarity. The study protocol and data management procedures were approved by the Ethics Committee of the Charité University Medical Centre, Berlin.

Noise exposure assessment

Environmental noise annoyance and sound level assessment

During the interview, the subjects were asked to indicate their annoyance during recent years by environmental noise on a scale from 1 (not annoyed at all) to 5 (extremely annoyed), considering eight potential sources of noise during day (6 a.m. to 10 p.m.) and night: road traffic, aircraft, railway, construction work, commercial, from above apartments, from within the apartment, and other outdoor noise. From this information, a summary score was derived, defined as the sum of annoyance on all individual noise sources divided by 16 (the total number of sources of diurnal and nocturnal noise). The annoyance level was further categorized with respect to exposure during day or night.

Environmental sound level exposure was assessed using the Berlin traffic noise map as proxy. For all addresses of subjects living either in streets with expected traffic of at least 6000 vehicles per day or close to major roads, sound pressure levels are routinely determined in noise maps in accordance with German standards for road and rail traffic. The algorithm of sound pressure levels used for the purpose of this study accounts for the distance between street and building, potential reflections from opposite buildings, and the spatial situation of the apartment vis-à-vis the street with additional adjustment for subjects living close to airports or near railway lines. For those subjects living in streets with less than 6000 vehicles per day and not close to major roads (concerning ~60% of the population), no exact sound pressure levels were available. In the official statistics, these subjects are assumed to live in areas with low sound levels. All decibel values in this article indicate average A-weighted sound pressure levels.

Work place noise annoyance and sound level assessment

During the interview, the annoyance by work place noise was assessed for up to the three most recent jobs during the preceding 10 years. Noise annoyance was assessed again on a scale from 1 to 5 considering three potential noise sources: from outside the workroom, inside self-produced, and inside from other sources. This information was combined to a summary score weighted for the total duration of employment within the 10 years period.

The 10 years work sound level exposure was determined according to the International Organization of Standardization (ISO) norm 9921/1, assessing vocal effort for speech communication required to overcome background noise levels at the work place. In addition, work noise exposure was assessed according to catalogue values for work places and machines with adjustment for ear protection gear.

Combined analysis of noise burden

In integrated analyses, both dimensions—environmental and work noise each assessed subjectively by annoyance and objectively by sound levels—were combined to determine the risk simultaneously associated with (i) environmental noise annoyance experienced by the eight potential sources as described previously; (ii) work noise annoyance combining the three potential sources as described previously; (iii) environmental noise comparing all subjects with increased sound levels during day (>60 decibels) and night (>50 decibels) with those with lower levels during day and night; (iv) work noise comparing all subjects with sound levels of >70 decibels with those with ≤55 decibels.

Statistical analysis

Sample size calculation yielded 2000 cases and 2000 controls to demonstrate an overall risk increase of 30% with an expected exposure of 15%, statistical power of 80%, and alpha significance level of 5%. Conditional logistic regression models appropriate for the analysis of matched case–control studies were applied. The models were developed in a stepwise manner. First, the control variables (known cardiac risk factors and other medical and sociodemographic variables as listed in Table 1) but not the exposure variables were included. This analysis was performed to find out whether the study was sensitive enough to significantly demonstrate the risk associations known from the literature and to identify potential sources of bias that should be controlled for in the subsequent analyses. Subsequently, a fixed set of confounders was defined, which was used for all of the following adjusted analyses. In a second step, noise exposure variables were studied one-by-one univariately as well as after adjustment. In particular, the "dose–response" function, i.e. the functional form of risk association such as linear vs. categorical vs. threshold model was established. This was done by comparing the model fit (as measured
by the likelihood ratio statistic) of a linear model with the fit of a model using a pre-defined categorized version of the noise exposure variable. Finally, a model combining a selection of exposure variables representing different sources of noise was analysed. Although continuous sound level measurements were available for a part of the patients, for analysis purposes, the sound levels had to be categorized to include patients with not-quantified low sound levels or missing values. For better comparability, the quantitative determinations were always categorized into three classes with pre-defined cut-offs taken from the literature. All analyses were performed for men and women separately because substantial gender differences in exposure patterns were anticipated. Calculations were performed using LOGXACT 4.0 for Windows.25 For variables with more than two categories, overall likelihood ratio tests were performed in order to control the type I error over categories. Subsequently, categorywise case/control comparisons were performed without adjustment for multiplicity. All reported P-values are two-sided.

Results

Characteristics of cases and controls

From June 1998 until March 2001, a total of 4115 patients were included into the NaRoMi study; of those were 3054 (74%) men with a mean of 56±9 years of age and 1061 (26%) women with 58±9 years. Further, 715 patients had been found eligible for, but did not consent to study participation, with similar rates in cases and controls. The prevalence of cardiovascular risk factors and important sociodemographic variables of cases and controls and the corresponding odds ratios are demonstrated for men and women in Table 1. As expected, most of the established cardiovascular risk factors were associated with a two- to three-fold odds ratio of myocardial infarction (with the exception of hyperlipidaemia with odds ratios of 5 and 6). Furthermore, some of the sociodemographic variables were associated with increased or decreased odds ratios. Using continuous variables for confounder adjustment yielded similar results. The noise sensitivity was similar in male cases and controls (both 2.8 ± 0.7) and in female cases and controls (both 2.9 ± 0.7). Sixteen per cent of the cases had a history of myocardial infarction; subgroup analyses yielded similar effects of noise in patients with or without prior myocardial infarction.

Noise annoyance

The annoyance levels by environmental and work noise for cases and controls and the corresponding odds ratios are demonstrated in Table 2. In men, neither environmental nor work noise annoyance was associated with myocardial infarction. In women, only annoyance by diurnal environmental noise was associated with a mildly increased risk of myocardial infarction that was not statistically significant after adjustment. As expected, the mean annoyance by environmental noise was higher during the day than during the night; however, the corresponding odds ratios were similar (Table 2).

Sound levels

The frequency of environmental noise exposure by sound level assessment for cases and controls and the corresponding odds ratios are demonstrated in Table 3. In men, the adjusted odds ratios in different noise categories ranged from 1.43 to 1.74 compared to the reference group of those exposed to
levels ≤60 decibels during the day and from 1.12 to 1.54 compared to the reference group of those exposed to levels ≤50 decibels during the night. In women, the respective odds ratios ranged from 1.24 to 2.22 during the day and from 2.73 to 3.50 during the night. In both gender groups, there was a step-up in risk above the reference group but no significant dose effect trend among the higher decibel categories. In those subjects without environmental noise level assessment, the odds ratios were significantly increased compared with the reference groups.

The frequency of work noise exposure by sound level assessment in cases and controls is also demonstrated in Table 3. In men, the adjusted odds ratios in different noise categories ranged from 1.19 to 1.25 compared to the reference group of those exposed to levels ≤50 decibels. In women, the respective odds ratios ranged from 0.88 to 1.11. Again, there was no significant dose effect relationship among the decibel categories. Using the assessment of sound levels according to catalogue values for the typical noise of work places and machines, the adjusted odds ratios in different noise categories ranged from 0.72 (95% confidence interval 0.57–0.90, P = 0.005) to 0.74 (0.57–0.95, P = 0.019) in men and from 1.11 (0.75–1.64, P = 0.608) to 1.20 (0.75–1.91, P = 0.608) in women.

### Discussion

The present results demonstrate that chronic noise exposure is associated with a mildly to moderately increased risk of myocardial infarction. In addition, the findings indicate gender-related differences associated with different noise sources.

To overcome the focus of previous smaller studies on selected noise sources only, the NaRoMI study was designed to assess and analyze simultaneously the subjective annoyance and the objective sound level both in environmental noise and in work noise. The validity of the present case-control design is supported in that the results of odds ratios associated with known cardiovascular risk factors were within the range of previous epidemiological studies. The high odds ratio associated with hyperlipidaemia is most likely based on underreporting of lipid disorders in control patients; therefore, this parameter was not included in statistical modelling.

To limit potential selection bias, we prospectively included patients in a population-based approach. In addition, hospital control patients were recruited with disorders occurring presumably independent of their status to noise exposure. To compensate for potential recall bias, the information obtained from the patients was complemented with noise parameters assessed independently from the interview.

Several findings of our study appear of particular interest. First, women were found relatively more susceptible to the risks of environmental noise annoyance when compared with men. One possible explanation is that women in our study may have spent more time at home when compared with men as suggested by the difference in working status. However, gender-specific reaction and pathophysiological response towards noise exposure also seem possible and should be investigated. Secondly, the risk of objective noise exposure appears to dominate the risk of subjective noise annoyance. Furthermore, the adjustment for individual noise sensitivity did not essentially alter the results. Finally, the lack of a consistent dose-response relationship in the noise parameters reported here suggests that noise burden is associated either with a threshold phenomenon regarding cardiovascular risks or perhaps with an initial monotonous dose-response relationship.
Table 3  Exposure to environmental and work sound levels in cases and controls, corresponding odds ratio, and overall tests (see statistical analysis for details)

<table>
<thead>
<tr>
<th>Men (n = 3.054)</th>
<th>Women (n = 1.061)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cases/controls (%)</td>
</tr>
<tr>
<td><strong>Environmental noise</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Day</strong></td>
<td></td>
</tr>
<tr>
<td>≤60 decibels (reference)</td>
<td>12/16</td>
</tr>
<tr>
<td>&gt;60–65 decibels</td>
<td>12/12</td>
</tr>
<tr>
<td>&gt;65–70 decibels</td>
<td>10/10</td>
</tr>
<tr>
<td>&gt;70 decibels</td>
<td>6/5</td>
</tr>
<tr>
<td>Not assessed</td>
<td>61/57</td>
</tr>
<tr>
<td><strong>Night</strong></td>
<td></td>
</tr>
<tr>
<td>≤50 decibels (reference)</td>
<td>5/7</td>
</tr>
<tr>
<td>&gt;50–55 decibels</td>
<td>11/12</td>
</tr>
<tr>
<td>&gt;55–60 decibels</td>
<td>10/11</td>
</tr>
<tr>
<td>&gt;60 decibels</td>
<td>13/12</td>
</tr>
<tr>
<td>Not assessed</td>
<td>61/57</td>
</tr>
<tr>
<td><strong>Overall test environmental noise</strong></td>
<td></td>
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<tr>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Work noise</strong></td>
<td></td>
</tr>
<tr>
<td>≤55 decibels (reference)</td>
<td>57/59</td>
</tr>
<tr>
<td>&gt;55–70 decibels</td>
<td>19/17</td>
</tr>
<tr>
<td>&gt;70 decibels</td>
<td>15/12</td>
</tr>
<tr>
<td>Not assessed</td>
<td>10/12</td>
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<tr>
<td><strong>Overall test work noise</strong></td>
<td></td>
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Decibel values indicate average A-weighted sound pressure levels. Odds ratio (OR) and 95% confidence interval (95% CI), univariate and adjusted for the variables presented in Table 1.
with subsequent saturation among categories of higher noise exposure.

The present findings are consistent with the hypothesis of an association between long-term noise exposure and risk of cardiovascular disease based on conceptual stress modelling.7,8 Sound pressure levels and/or annoyance by noise may enhance psychological stress and anger and lead to impaired physiological factors such as increasing catecholamine levels associated with increased blood pressure and plasma lipids.8–10 Such mechanisms may be further modified by situational and personal parameters (e.g. time pressure, smoking). Accordingly, chronic noise exposure would be the equivalent of an exogenous risk factor contributing to the development of atherosclerosis and cardiovascular disease.28 Previous observations of a dose-response relationship between nightly sound exposure level and odds ratio for the prevalence of treatment for high blood pressure are consistent with our finding that objective sound exposure may be responsible for disease.29 Finally, the present findings are also consistent with previous observations of possible triggers of acute myocardial infarction, including stress, anger, and exposure to traffic.30–32 On the basis of the conceptual relationship between loud noise and stress, noise exposure may perhaps precede the onset of acute myocardial infarction. Furthermore, noise exposure and air pollution may be related or even interact in increasing risk of pulmonary and cardiovascular disease.30

The mildly increased risk of myocardial infarction in men exposed to increased sound levels is consistent with the results of earlier studies.18,20 However, the observation of a markedly increased risk (even though with wide confidence interval) in women is new. As in most previous studies women were not separately analysed or not even included, this finding needs to be confirmed or refuted in future studies. In the adjusted analyses, the subjects exposed to streets with over 60 decibels during the day experienced an increased risk of myocardial infarction when compared with the subjects with exposure below 60 decibels according to the algorithm used in the present study. During the analyses, it became apparent that the large group of subjects without further street sound level assessment, generally regarded as living in areas less exposed to noise, demonstrated an increased risk of myocardial infarction also. As such pattern lacks any potential explanation by biological plausibility, it may be perhaps based on other yet unknown factors. Probably, the assumption of relationship between vehicle numbers and sound levels underestimates noise exposure in those areas. The overall results of subjective noise exposure showed no trend.

The results of work sound level assessment yielded apparently inconsistent results according to the two different methods used. Although noise assessment based on vocal effort was associated with an increased risk of myocardial infarction, noise assessment based on catalogue values was not. We believe that the first method is more adequate because the information was obtained individually from all study subjects and reflects the actual work situation with fluctuating noise levels, whereas the catalogue values represent average peak values. Furthermore, after including both categories into a multivariate model, the vocal effort results remained significant. The results emphasize the need to reassess the importance, in general, and the adequate thresholds, in particular, of wearing ear protection at work places. The currently used threshold of 85 decibels33 may protect sufficiently from hearing damage but not from cardiovascular risk.

Some general limitations of the NaRomi study should be pointed out. Although the complexity of subjective and objective noise exposure was integrated better than in earlier studies, there are unresolved methodological issues. Transitional noise exposure, such as while driving to work or during leisure and sports activities, was not evaluated. During analysis, the noise exposure was categorized and the outcome validated using partially the same data set. The recruitment of hospital instead of population-based controls was chosen mainly for logistic reasons and implies potential selection bias. Finally, patients with age >70, those experiencing fatal myocardial infarction, and those living in rural areas were not included in our study. Therefore, the present results can be considered representative only for non-fatal myocardial infarction and city populations excluding the elderly.

Noise is a frequent burden of our daily life particularly in metropolitan areas and at industrial work sites. The present results may provide a basis for gaining further insight into pathophysiological mechanisms associated with noise. The results should also aid in improving preventive strategies particularly for subjects at increased risk of developing cardiovascular disease.

**Acknowledgements**

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**Conflict of interest:** none declared.
Appendix

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