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Traffic Noise and Risk of Myocardial Infarction

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Background: The biologic plausibility for noise stress-related cardiovascular responses is well established. Epidemiologic studies on the relationship between transportation noise and ischemic heart disease suggest a higher risk of myocardial infarction in subjects exposed to high levels of traffic noise.

Methods: To determine the risk of road traffic noise for the incidence of myocardial infarction (MI), we carried out a hospital-based case-control study in the city of Berlin. We enrolled consecutive patients ($n = 1881$), age 20–69 years, with confirmed diagnosis of MI from 1998 through 2001. Controls ($n = 2234$) were matched according to sex, age, and hospital. Outdoor traffic noise level was determined for each study subject based on noise maps of the city. Standardized interviews were conducted to assess possible confounding factors and the annoyance from various noise sources.

Results: The adjusted odds ratio for men exposed to sound levels of more than 70 dB(A) during the day was 1.3 (95% confidence interval = 0.88–1.8) compared with those where the sound level did not exceed 60 dB(A). In the subsample of men who lived for at least 10 years at their present address, the odds ratio was 1.8 (1.0–3.2). Noise-exposed women were not at higher risk.

Conclusions: The results support the hypothesis that chronic exposure to high levels of traffic noise increases the risk for cardiovascular diseases.

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Epidemiologic studies on the relationship between transportation noise and ischemic heart disease suggest a higher risk of myocardial infarction in subjects exposed to high levels of traffic noise.^{1,2} Although the findings in these studies seem to be reasonably consistent, many of the individual studies have low statistical power. Expert groups have rated the evidence of the association from “limited” to “sufficient.”^{3,4} The existing data on the relationship between road traffic noise and ischemic heart disease suggest an average A-weighted sound pressure level of 65 to 70 dB(A) during the day as a possible threshold of effect. (The unit of sound measurement is decibels [dB]. “A-weighting” refers to a filter, which is used in sound meters to account for differences in hearing sensitivity at different sound frequencies; “dB(A),” is the common unit for the assessment of community noise and occupational noise.)

A previous population-based case-control study carried out in the area formerly known as West Berlin found an odds ratio (OR) for myocardial infarction of 1.32 (95% confidence interval = 0.89–1.96) in men who had lived for at least 15 years on streets with average A-weighted sound levels (6–22 hours) of more than 70 dB(A) compared with subjects who lived on streets with sound levels up to 60 dB(A).⁵ The Noise and Risk of Myocardial Infarction (NaRoMI) study is a replication of the previous one using the same test hypothesis on a new sample. It includes a larger sample size, uses improved methods of exposure assessment, and considers a larger set of potentially confounding factors. It is a hospital-based case-control study covering the entire city of Berlin.

METHODS

Selection of Cases and Controls

To determine the potential risk of noise for the incidence of myocardial infarction (MI), a matched case-control study was carried out. Patients consecutively admitted to 32 major hospitals in Berlin with confirmed diagnosis of acute MI or survivors of sudden cardiac arrest (International Classification of Diseases, 9th revision code 410), age 20–69 years, were enrolled over a prospective period of 3 years from 1998 to 2001. The diagnostic criteria followed the World Health Organization definitions, including ischemic changes in the electrocardiogram, clinical symptoms, and enzymatic changes.

Hospital controls were individually matched according to sex, age (5-year categories), and hospital. Because of the lower incidence rate of MI in women, a case:control ratio of 1:1 for men and 1:2 for women was applied to increase the statistical power for women. Control patients were admitted to the same hospitals for diagnoses that were presumably not related to noise, including hernia, goiter, colon or rectum problems, and accidents. Study participants were informed about the objective of the study and gave their written consent. The study was approved by the ethical commission of the Medical Faculty of the Humboldt, University of Berlin.

Covariates

After subjects were moved from intensive care, standardized interviews were conducted on the wards to assess the home environment, sociodemographic features, and potentially confounding factors. Data included family history of MI, smoking, education level, marital status, employment status, working hours (employment) >40 hours/week in any job during the past 10 years, shift work, second job or activity >5 hours per week, and Weinstein noise sensitivity.⁶ We obtained clinical information on diagnoses regarding diabetes mellitus, hypertension, hyperlipidemia, and body mass index from clinical records. To account for possible confounding, all variables were included in the statistical models. As a result of possible incomplete assessment in controls, hyperlipidemia was considered only in sensitivity analyses.

Noise Assessment

The objective traffic noise exposure (sound level) of the subjects was assessed using noise maps from the city authorities and standardized questionnaires. We calculated traffic noise levels (12 months average A-weighted sound pressure levels as determined from noise maps) according to ANSI S1.4 and ISO 1996/1 with reference to the most affected facades of the dwellings for day (6–22 hours) and night (6–22 hours), taking seasonal variations into consideration. The noise maps were established in accordance with German standards for road (RLS 90) and rail traffic (SCHALL 03) and accounted for reflections from the buildings opposite. All main roads with more than 6000 vehicles per day were assessed by the traffic authorities, and exact noise levels at the facades of the houses were calculated for more than 6300 street segments (parts between intersections).⁷ Streets with lower traffic volume (side streets) were categorized as “quiet.” No exact sound levels can be given for these streets. However, the cutoff criterion of traffic volume refers to average A-weighted sound levels during the day of approximately 60 dB(A) and approximately 50 dB(A) during the night at a distance of 25 m from the streets (maximum speed 50 km/h, 5% heavy vehicles). The traffic noise exposure in side streets was validated using data of 4 of 12 Berlin District Councils that assessed the noise levels in all the side streets of

their parts of the city (more than 5800 street segments). The speed limit in 85% of all the side streets was 30 km/h and 50 km/h in all other streets. The group of subjects living in side streets served as the reference group in the statistical analyses, which was in accordance with the test hypothesis and previous noise studies.

All subjects' houses were categorized in 5-dB(A) categories (as usually applied in noise regulations) according to the sound levels given in the traffic noise map. In the first step, we made the assessment for the home address (in most cases the street closest to the buildings). In the second step, all addresses were checked for noise from streets other than the home address. Using high-resolution GIS information, the distances to all main roads were measured for each house. When this sound level was higher than the one for the street of the address, we reallocated subjects into the higher sound-level category; otherwise, subjects remained in their initial category. All noise calculations were made separately with respect to the front (facing the street of the address) and back of the house.

To account for transportation noise other than from streets, dichotomous variables were created so that residence within the 60 dB(A) contours around airports or railway lines could be evaluated. These calculations were made according to the German aircraft noise regulations considering an exchange rate (ISO 1996/1) of 3 dB(A), the train noise module of the Berlin noise map, and the measured distance of houses from railway lines. The 2 variables (aircraft noise and train noise) were considered as covariates in the statistical analyses.

The 10-year worknoise exposure (sound level) was determined according to ISO 9921/1 assessing vocal effort for speech communication and according to catalogs for workplaces and machines, allowing for the use of ear protection. For the present analyses, we controlled for possible confounding by occupational noise exposure (in dB(A), corrected for use of ear protection: ≤55, 56–70, 71–85, >85, or no data or no job). Replacing this with other work noise indicators did not considerably change the effect estimates of the traffic noise factor.

The subjective experience of noise exposure (“annoyance”) was assessed using a standardized questionnaire. Personal interviews were carried out in the hospitals. Environmental noise annoyance was determined using a 5-point scale (“Considering recent years, how much were you disturbed by noise at home? 1 = not disturbed at all, 5 = very disturbed”). We considered 8 noise sources around and in the subject's home: road traffic noise, aircraft noise, railway noise (excluding tram), noise from construction works, commercial noise (including noise from industries), other outdoor noise, impact noise, and other indoor noise. Annoyance during the day and the night was evaluated separately. To control for annoyance from occupational noise, we created an indicator variable (annoyance level: high/fairly high, fairly low/low, or no data/no job during the past

10 years). This variable was based on information taken from the noise questionnaire referring to noise from outside of the work room, from the subject's own machines or appliances, and from machines or appliances used by colleagues (sum score of annoyance, weighted by duration of employment).⁸

Statistical Analysis

Conditional logistic regression analyses were carried out (matched analyses) to calculate OR and CI, and to adjust the results for a set of potentially confounding factors. Because most of the previous noise studies were carried out in men, separate models were calculated for men and women. We calculated nonparametric regression coefficients to assess associations between the determinants of noise exposure. Associations between noise level and MI incidence were analyzed in the total sample

and in a subsample of subjects who had been living at least for 10 years in their present homes. This enabled us to account for chronic noise stress conditions and the long induction period of the disease under study. The cutpoint of 10 years was determined on the basis of the distribution of the residence time on the one hand and on pragmatic grounds of sample size and statistical power on the other. To ensure that effect estimates obtained from the subsample were stable, other criteria were also applied (eg, 15 years).

RESULTS

Table 1 shows characteristics of the cases and the controls, including the number of subjects, the prevalence of risk factors, and the distribution of other covariates. The total

TABLE 1. Characteristics of Study Subjects, and Association Between Covariates and Myocardial Infarction

Variable*	Men		Women		Men (n = 3054) OR [†] (95% CI)	Women (n = 1061) OR [†] (95% CI)
	Cases (n = 1527)	Controls (n = 1527)	Cases (n = 354)	Controls (n = 707)		
Age (years); mean ± SD*	56 ± 8	56 ± 9	58 ± 9	58 ± 9		
Diabetes mellitus	17	10	25	11	1.84 (1.43–2.38)	3.00 (1.95–4.62)
Hypertension	43	25	48	31	2.24 (1.87–2.70)	1.99 (1.45–2.74)
Family history of myocardial infarction	31	17	37	22	2.11 (1.73–2.57)	2.00 (1.45–2.76)
Smoking status						
Current	54	45	48	29	2.69 (2.11–3.43)	3.85 (2.64–5.61)
Former	32	32	22	22	1.80 (1.41–2.30)	1.97 (1.31–2.96)
Never [‡]	14	23	30	49	1.0	1.0
Body mass index (kg/m ²)						
<25 [‡]	37	45	38	46	1.0	1.0
25–29	46	39	35	34	1.22 (1.02–1.46)	1.14 (0.80–1.62)
≥30	15	16	27	20	0.89 (0.70–1.13)	1.42 (0.95–2.13)
Unknown	1	0	1	1	5.42 (1.93–15.2)	1.56 (0.23–10.5)
Current employment status						
Unemployed	11	13	10	7	0.74 (0.57–0.97)	1.09 (0.60–1.96)
Not in working for other reasons	42	46	61	64	0.57 (0.45–0.72)	0.52 (0.33–0.83)
Employed >10 hrs/wk	47	42	29	29	1.0	1.0
Employment >40 hrs/wk during past 10 yr	54	48	25	231	1.14 (0.97–1.35)	1.02 (0.71–1.46)
Second job >5 hrs/wk	19	17	17	140	1.11 (0.89–1.37)	1.23 (0.81–1.85)
Shift work	26	25	19	15	1.05 (0.87–1.27)	1.08 (0.71–1.65)
Living without partner	20	31	34	42	0.55 (0.45–0.67)	0.60 (0.44–0.83)
<12 yr at school	74.7	73.5	87.9	78.2	1.11 (0.91–1.36)	1.68 (1.07–2.62)
Noise sensitivity score (6-point scale); mean ± SD* [§]	2.8 ± 0.7	2.8 ± 0.7	2.9 ± 0.7	2.9 ± 0.7	1.14 (1.01–1.29)	1.05 (0.85–1.30)

*All characteristics are expressed in percent, unless otherwise indicated.

[†]Multivariate model, adjusted for all other variables in the table.

[‡]Reference category is the absence of the condition, except where indicated.

[§]Odds ratios are per unit of a 6-point scale.

number of 4115 study participants (response rate 86%) was made up of 3054 men (age mean \pm standard deviation [SD]: 56 ± 8.5 years) and 1061 women (age mean \pm SD: 58 ± 8.7 years).

Table 1 also shows adjusted risk estimates for the relationships between the covariates and the incidence of MI as derived from the multiple logistic models, in which only the nonnoise factors given in the table were considered. Established biologic and nonbiologic risk factors (diabetes mellitus, hypertension, family history of MI, smoking) were strongly associated with the occurrence of MI (odds ratios between 1.7 and 3.1) and were within the range of the usual findings in epidemiologic studies.^{9–11} We found odds ratios of 5.5 in men and 4.5 in women, which are higher than in most other studies, presumably because of incomplete assessment of hyperlipidemia in the controls. However, the inclusion or exclusion of this variable did not considerably affect the estimates that were obtained for any of the noise-related factors in the later analyses.

Table 2 gives the distribution of traffic noise levels in the total sample and in the subsample of subjects who had lived at their current address for at least 10 years. This refers to the highest average sound level measured during the daytime at any outside wall of the subjects' houses. Because noncategorized day and night sound levels were highly correlated ($r = 0.98$, mean difference 7.3 dB(A)), only the results referring to the sound level during the day are given here. Most subjects lived in quiet side streets. Sixteen percent of the subjects' houses were exposed to sound levels of more than 65 dB(A) during the day. Two thirds (69%) of the subjects had lived at their present address for at least 10 years.

For risk analyses, all side streets were classified as "quiet" (sound level criterion: $L_{\text{Day}} \leq 60$ dB(A)). To validate this classification, we examined the sound level data for the complete network of side streets in 2 inner and 2 outer Berlin districts that were available. Most sound levels during the day in these side streets were between 45 and 55 dB(A) (inner districts: 51%; outer districts: 71%) or between 56 and 60 dB(A) (inner districts: 33%; outer districts: 20%). Only a minor number of sound levels (inner districts: 16%; outer districts: 9%) were higher than 60 dB(A). This suggests that exposure misclassification in the reference group was not very likely to have affected (dilution of effect) the results.

Table 2 also gives the adjusted estimates of the relative risk of MI and 95% confidence intervals for men and women in each traffic noise category. In the total sample, we found a slight increase in risk with increasing sound level for men. For men in the highest noise category (>70 dB(A)) compared with the lowest (≤ 60 dB(A)), the odds ratio was 1.3 (95% CI = 0.88–1.8). There was no apparent risk among women (OR = 0.7; CI = 0.32–1.4).

In the subsample of subjects who had lived for at least 10 years at their present address, there was a stronger monotonic increase in risk for men across the noise categories (Table 2). For males in the highest noise category, the odds ratio for MI was 1.8 (1.0–3.2). The result was similar when 15 years of residence was considered (OR = 1.8; CI = 0.86–3.7). When we analyzed the subsample of women, the statistical model did not converge when including all covariates (as a result of the smaller sample size); we therefore created reduced models that included only the classic risk factors. No noise effect was found for women.

TABLE 2. Association Between Traffic Noise Level (dB(A), 6–22 hr) and Myocardial Infarction for Total Sample and for Subsample of Subjects Who Had Lived at Their Current Address for at Least 10 Years

Traffic Noise Level	Men		Women	
	No.	OR (95% CI)*	No.	OR (95% CI)*
<i>Total sample</i>				
$\leq 60^\dagger$	2231	1.0	759	1.0
61–65	355	1.01 (0.77–1.31)	119	1.14 (0.70–1.85)
66–70	300	1.13 (0.86–1.49)	131	0.93 (0.57–1.52)
>70	168	1.27 (0.88–1.84)	52	0.66 (0.32–1.35)
<i>Subsample</i>				
$\leq 60^\dagger$	1547	1.0	529	1.0
61–65	251	1.17 (0.81–1.69)	82	1.04 (0.55–1.97)
66–70	202	1.31 (0.88–1.97)	95	1.11 (0.62–1.98)
>70	111	1.81 (1.02–3.21)	37	0.90 (0.39–2.07)

*Odds ratios for men and for total sample of women are adjusted for the covariates listed in Table 1 and for indicator variables of work noise, aircraft noise, and railway noise. Odds ratios for subsample of women are adjusted only for diabetes mellitus, hypertension, family history of MI, and smoking as a result of small sample size.

† Reference category.

When the 2 highest noise categories were combined (for comparison with the literature) among men who lived in streets with outdoor traffic noise levels of more than 65 dB(A), we found odds ratios of 1.2 (0.93–1.5) for the total sample and 1.4 (1.0–2.0) for the subsample.

Within the reference group, 2 subgroups were identified (a posteriori). 82% of the subjects of the reference group lived in side streets, which were not in relevant distance to main roads or were completely shielded by sound barriers from these streets, so that noise from these streets could not affect these subjects. The remaining 18% of the reference group were potentially affected by noise from main roads in the near distance, although using exact calculations, the noise criterion of the reference group ($L_{Day} \leq 60$ dB(A)) was not exceeded. A substantially lower MI risk was found in this small subgroup for men (OR = 0.7; CI = 0.5–0.9) and for women (OR = 0.5; CI = 0.3–0.8) when compared with the large subgroup.

Table 3 shows mean annoyance scores for the 2 subgroups of the reference group and the higher noise-exposed groups. Across the exposure groups, a steady increase of noise annoyance resulting from road traffic noise was found with increasing traffic noise level.

Appendix Table 1 (available with the electronic version of this article) shows the distributions of noise annoyances for day and night. During the day, approximately 15% were highly annoyed by road traffic noise (categories 4 and 5 on a 5-point scale), and during the night, approximately 8% were highly annoyed. Annoyance scores were lower for other sources of noise.

Table 4 shows the associations between noise annoyance and MI. To handle all 8 annoyance variables simultaneously, they were treated as continuous variables in the

models. The odds ratios give an estimate of the relative risk per unit of the 5-point scale. All sound level-related variables were excluded from the analyses, as was noise sensitivity, for reasons of collinearity among variables. However, annoyance from noise at work was considered as a covariate. We found risk of MI to be elevated by road traffic noise annoyance at night in men (OR = 1.10; CI = 1.01–1.20) and aircraft noise annoyance at night in women (1.28; 1.01–1.63).

Although cases and controls were matched according to age, additional analyses were carried out, including age as a continuous variable in the models, to assess the impact of residual confounding. The maximum impact on any of the odds ratios was +0.02 when the noise level was considered and ± 0.01 when annoyance was considered.

Occupational noise exposure was considered as a covariate in the analyses. Lower risks were found in men from all of the 3 higher occupational noise categories (56–70, 71–85, >85 dB(A)) in comparison with the reference category (≤ 55 dB(A)) showing odds ratios between 0.6 and 0.7. In women who were occupationally exposed to higher noise levels, odds ratios were between 1.1 and 1.2.

DISCUSSION

Other investigators have studied the nonauditory effects of noise, in particular, cardiovascular effects such as high blood pressure and ischemic heart diseases.^{1,12–17} The biologic plausibility of the association is based on the general stress concept and has been established for a long time on the basis of laboratory and animal experiments.^{1,18–21} It has been estimated that approximately 2% to 3% of ischemic heart diseases in the general population could be attributed to the traffic noise, if the noise hypothesis is true.^{3,22}

TABLE 3. Association Between Traffic Noise Level (dB(A), 6–22 hr) and Mean Annoyance Score Resulting from Traffic Noise

Traffic Noise Level		Men		Women	
		No.	Mean Score (95% CI)	No.	Mean Score (95% CI)
<i>Annoyance during the day</i>					
≤ 60	side streets	1808	1.8 (1.7–1.8)	629	1.9 (1.8–2.0)
≤ 60	side streets and main roads	423	2.0 (1.9–2.1)	130	2.2 (2.0–2.5)
61–65	main roads	355	2.3 (2.2–2.4)	119	2.4 (2.2–2.6)
66–70	main roads	300	2.8 (2.6–2.9)	131	2.8 (2.6–3.1)
>70	main roads	168	2.6 (2.4–2.8)	52	3.1 (2.7–3.5)
<i>Annoyance during the night</i>					
≤ 60	side streets	1808	1.4 (1.3–1.4)	629	1.4 (1.3–1.4)
≤ 60	side streets and main roads	423	1.5 (1.4–1.6)	130	1.5 (1.3–1.6)
61–65	main roads	355	1.6 (1.5–1.7)	119	1.8 (1.6–2.0)
66–70	main roads	300	2.0 (1.8–2.1)	131	2.0 (1.8–2.3)
>70	main roads	168	1.6 (1.8–2.6)	52	2.2 (1.8–2.6)

TABLE 4. Association Between Noise Annoyance and Myocardial Infarction

Source of Noise	Men		Women	
	Day OR (95% CI)*	Night OR (95% CI)*	Day OR (95% CI)*	Night OR (95% CI)*
Road traffic	1.04 (0.97–1.12)	1.10 (1.01–1.20)	1.03 (0.90–1.18)	0.98 (0.84–1.14)
Aircraft	1.01 (0.93–1.10)	1.05 (0.93–1.19)	1.13 (0.97–1.32)	1.28 (1.01–1.63)
Rail	0.92 (0.82–1.04)	0.99 (0.85–1.15)	0.96 (0.78–1.18)	0.94 (0.71–1.24)
Industrial	1.06 (0.93–1.21)	0.91 (0.77–1.08)	1.11 (0.89–1.39)	1.02 (0.76–1.36)
Construction	1.08 (1.00–1.17)	1.10 (0.87–1.39)	1.05 (0.93–1.20)	1.17 (0.87–1.57)
Other outdoor	0.96 (0.88–1.05)	0.96 (0.86–1.07)	0.99 (0.85–1.15)	1.00 (0.82–1.22)
Impact noise	1.04 (0.95–1.14)	1.02 (0.90–1.16)	0.94 (0.79–1.11)	0.95 (0.75–1.20)
Other indoor	0.92 (0.84–1.02)	0.99 (0.87–1.12)	1.03 (0.88–1.21)	1.09 (0.89–1.33)

*Odds ratio per unit on a 5-point scale; separate models for males and females, day and night. Odds ratios are adjusted for the covariates listed in Table 1, work noise annoyance, and all other annoyance variables given in this table.

In the present study, findings from an earlier study using largely the same methods were confirmed. Male subjects who lived in streets with average A-weighted sound levels during the day of more than 70 dB(A) showed an increase in risk of MI compared with those who lived in streets with levels of less than or equal 60 dB(A). In the total sample, we found a modest odds ratio of 1.3. In the subsample of subjects who had been living at their present for at least 10 years, the odds ratio was 1.8. There was a clear dose-response relationship of higher risk with increasing traffic noise. Noise levels of 65 dB(A) to 70 dB(A) outdoors have been considered as a relevant threshold of adverse health effects of noise.^{3,23–25} When the 2 highest noise categories are combined ($L_{Day} > 65$ dB(A)), the relative risk for men was 1.2 in the total sample and 1.4 in the subsample. The finding that the estimated effect is larger with longer residence is plausible and in accordance with the test hypothesis. The disease outcome under study has a long induction time. One would expect many years of chronic noise stress exposure before pathologic changes become manifest.^{26,27} Residence time has also been found in other studies to be an important effect (exposure) modifier of the relationship between traffic noise and cardiovascular diseases.^{5,28–30}

We found no higher MI risk among women with respect to traffic noise level, even after controlling for a large number of potentially confounding factors. Our finding of a slight increase in MI risk with increasing traffic noise level only in men is consistent with our finding of a positive relationship between noise annoyance resulting from road traffic noise and MI incidence only in men. The negative results among women were not controlled for possible differential effects of the intake of sex hormones, which may protect or promote adverse (noise-) stress effects.^{31,32} In noise experiments, physiological reactions controlled by the autonomic nervous system were less pronounced in women

than in men.^{33,34} In large cross-sectional studies, a higher prevalence of high blood pressure was found among men exposed to traffic noise but not among exposed women.^{35,36} Other authors have discussed the negative findings of a traffic noise and blood pressure study carried out on women with respect to the use of contraceptives.³⁷ Different time activity patterns may also contribute to differences in noise effects between the sexes. However, the sample of women was much smaller than the sample of men in our study, which could also contribute to the inconsistent findings.

No explanation can be given for the lower risk found in a subgroup of the reference group (a posteriori testing). Those subjects lived in side streets that were potentially affected by nearby main roads, although the noise exposure (L_{Day}) was below 60 dB(A) as for most of the reference group subjects who lived on side streets that were unaffected by main roads. It is possible that the subgroup with the lower risk was exposed to even higher traffic noise. The traffic volume dilutes with larger distances from main roads. Furthermore, those subjects were more annoyed by traffic noise than the rest of the reference group. Because monotonic trends between sound level and annoyance are repeatedly found in social surveys,^{38,39} a higher traffic noise exposure, on average, can be inferred for those subjects. The difference in MI risk between the 2 subgroups is presumably the result of unknown factors unrelated to traffic noise. On acoustical grounds, no distinction can be made between the 2 subgroups (a priori testing).

The primary focus of the NaRoMI study was traffic noise. The noise exposure of each individual's home was precisely assessed within a range of the sound levels during the day between ≤ 60 and 80 dB(A). This means that subjects who lived only 20 or 30 yards from one another could differ in their outdoor noise exposure by 20 dB(A), which is a 100-fold range in terms of sound energy. It was impossible to

be similarly precise with respect to occupational noise exposure. It was not possible to take noise measurements at each individual's workplace as a result of practical and legislative limitations (permission for measurement). Occupational noise exposure was therefore assessed by interviews of the subjects. Estimates of the noise exposure were made according to expert ratings regarding the noise exposure of characteristic occupational environments. The noise indicator used in the present analyses was negatively associated with MI incidence. The "healthy worker effect" may help to explain this finding.⁴⁰

The study results support the hypothesis that chronic exposure to road traffic noise increases the risk for MI in men, thus confirming the results of the previous study using a similar case-control design. Although the previous study suggested a threshold effect, the data here suggest a monotonic increase in risk with increasing sound level. The effect estimates are larger than those of the previous study, particularly when the years of residence of the study subjects are considered in the analyses. This stronger association is probably the result of improvement in the assessment of noise exposure since the previous study, with the availability of noise maps embedded in a detailed graphic information system.

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REFERENCES

- Babisch W. Traffic noise and cardiovascular disease: epidemiological review and synthesis. *Noise Health*. 2000;2:9–32.
- Kempen EEMV, Kruijze H, Boshuizen HC, et al. The association between noise exposure and blood pressure and ischaemic heart disease: a meta-analysis. *Environ Health Perspect*. 2002;110:307–317.
- Babisch W. The noise/stress concept, risk assessment and research needs. *Noise Health*. 2002;4:1–11.
- Passchier-Vermeer W, Passchier WF. Noise exposure and public health. *Environ Health Perspect*. 2000;108(suppl 1):123–131.
- Babisch W, Ising H, Kruppa B, et al. The incidence of myocardial infarction and its relation to road traffic noise—the Berlin case-control studies. *Environ Int*. 1994;20:469–474.
- Weinstein ND. Individual differences in relations to noise: a longitudinal study in a college dormitory. *J Appl Psychol*. 1978;63:458–466.
- Senatsverwaltung für Stadtentwicklung. Umwelatlas Berlin. Available at: http://www.stadtentwicklung.berlin.de/umwelt/umweltatlas/dinh_07.htm. Accessed October 28, 2003.
- Schust M, Stark H, Keil T, et al. The rank of noise at the workplace within the epidemiology of heart disease—results of the NaRoMI-study. In: Brambilla G, Ianiello C, Maffei L, eds. *Proceedings of the 5th European Conference on Noise Control in Naples 2003*. Roma: Istituto di Acustica; 2003.
- Yusuf S, Reddy S, Ounpuu S, et al. Global burden of cardiovascular diseases: part I: general considerations, the epidemiologic transition, risk factors, and impact of urbanization. *Circulation*. 2001;104:2746–2753.
- Navas-Nacher EL, Colangelo L, Beam C, et al. Risk factors for coronary heart disease in men 18 to 39 years of age. *Ann Intern Med*. 2001;134:433–439.
- Myers RH, Kiely DK, Cupples LA, et al. Parental history is an independent risk factor for coronary artery disease: the Framingham Study. *Am Heart J*. 1990;120:963–969.
- Rehm S. Research on extraaural effects of noise since 1978. In: Rossi G, ed. *Proceedings of the 4th International Congress on Noise as a Public Health Problem in Turin 1983*, vol 1. Milano: Edizioni Tecniche a cura del Centro Ricerche e Studi Amplifon; 1983:527–547.
- Thompson SJ. Effects of noise on the cardiovascular system: appraisal of epidemiologic evidence. In: Rossi G, ed. *Proceedings of the 4th International Congress on Noise as a Public Health Problem in Turin 1983*, vol 1. Milano: Edizioni Tecniche a cura del Centro Ricerche e Studi Amplifon; 1983:711–714.
- Schwarze S, Ettema JH. Summary of team 3: non auditory physiological effects. In: Berglund B, Lindvall T, eds. *New advances in noise research, part II. Proceedings of the 5th International Congress on Noise as a Public Health Problem in Stockholm 1988*, vol 5. Stockholm: Swedish Council for Building Research; 1990:301–302.
- Schwarze S, Thompson SJ. Research on non-auditory physiological effects of noise since 1988: review and perspectives. In: Vallet M, ed. *Noise and man '93. Proceedings of the 6th International Congress on Noise as a Public Health Problem in Nice 1993*, vol 3. Arcueil Cedex: Institut National de Recherche sur les Transports et leur Sécurité; 1993:252–259.
- Lercher P, Stansfeld SA, Thompson SJ. Non-auditory health effects of noise: review of the 1993–1998 period. In: Carter N, Job RFS, eds. *Noise effects '98. Proceedings of the 7th International Congress on Noise as a Public Health Problem in Sydney 1998*, vol 1. Sydney: Noise Effects '98 PTY Ltd; 1998:213–220.
- Stansfeld SA, Lercher P. Non-auditory physiological effects of noise: five year review and future directions. In: Jong RD, Houtgast T, Franssen EAM, et al., eds. *ICBEN 2003. Proceedings of the 8th International Congress on Noise as a Public Health Problem in Rotterdam 2003*. Schiedam: Foundation ICBEN; 2003:84–90.
- Henry JP. Biological basis of the stress response. *NIPS*. 1993;8:69–73.
- Borg E. Physiological and pathogenic effects of sound. *Acta Otolaryngol*. 1981;Suppl 381:3–67.
- Kjellberg A. Subjective, behavioral and psychophysiological effects of noise. *Scand J Work Environ Health*. 1990;16(suppl 1):29–38.
- Ising H. Extraaural effects of chronic noise exposure in animals—a review. In: Ising H, Kruppa B, eds. *Lärm und Krankheit—noise and disease. Proceedings of the International Symposium 'Noise and Disease' in Berlin 1991*, vol 88. Schriftenreihe des Vereins für Wasser-, Boden- und Lufthygiene. Stuttgart: Gustav Fischer Verlag; 1993:57–64.
- Neus H, Boikat U. Evaluation of traffic noise-related cardiovascular risk. *Noise Health*. 2000;2:65–77.
- EEA. *Traffic Noise: Exposure and Annoyance*. Copenhagen: European Environmental Agency. Available at: http://themes.eea.eu.int/Sectors_and_activities/transport/indicators/consequences/noise_exposure/Noise_TERM_2001.doc.pdf. Accessed June 9, 2004.
- Guidelines for Community Noise*. Geneva: World Health Organization. Available at: http://www.euro.who.int/Noise/Publications/20030528_2. Accessed March 16, 2004.
- Health Council of The Netherlands. Effects of noise on health. *Noise/News International*. 1996:137–150.
- Lercher P, Koffler WW. Behavioral and health responses associated with road traffic noise exposure along alpine through-traffic routes. *Sci Total Environ*. 1996;189:85–89.
- Thompson SJ. Cardiovascular and fetal effects of noise. In: IEH, ed. *Workshop on Non-auditory Health Effects of Noise*. Leicester: Institute for Environment and Health; 1997.
- Babisch W, Ising H, Gallacher JEJ, et al. Traffic noise and cardiovascular risk: the Caerphilly and Speedwell studies, third phase—10 years follow-up. *Arch Environ Health*. 1999;54:210–216.
- Bluhm G, Nordling E, Berglund N. Increased prevalence of hypertension in a population exposed to road traffic noise. In: Boone R, ed. *Internoise 2001. Proceedings of the 2001 International Congress and Exhibition on Noise Control Engineering in The Hague*, vol 3. Maastricht: Nederlands Akoestisch Genootschap; 2001:1563–1566.
- Neus H, Eiff A-Wv, Rüdell H, et al. Traffic noise and hypertension. The Bonn traffic noise study. In: Rossi G, ed. *Proceedings of the 4th*

- International Congress on Noise as a Public Health Problem in Turin 1983*, vol 1. Milano: Edizioni Tecniche a cura del Centro Ricerche e Studi Amplifon; 1983:693–698.
31. Farley TMM, Meirik O, Chang CL, et al. Combined oral contraceptives, smoking, and cardiovascular risk. *J Epidemiol Community Health*. 1998;52:775–785.
 32. Cairns V, Keil U, Doering A, et al. Oral contraceptive use and blood pressure in a German metropolitan population. *Int J Epidemiol*. 1985;14:389–395.
 33. Neus H, Schirmer G, Rüdell H, et al. On the reaction of finger pulse amplitude to noise. *Int Arch Occup Environ Health*. 1980;47:9–19.
 34. Ising H, Braun C. Acute and chronic endocrine effects of noise: review of the research conducted at the Institute for Water, Soil and Air Hygiene. *Noise Health*. 2000;2:7–24.
 35. Herbold M, Hense H-W, Keil U. Effects of road traffic noise on prevalence of hypertension in men: results of the Lübeck blood pressure study. *Soz Präventivmed*. 1989;34:19–23.
 36. Belojevic G, Saric-Tanaskovic M. Prevalence of arterial hypertension and myocardial infarction in relation to subjective ratings of traffic noise exposure. *Noise Health*. 2002;4:33–37.
 37. Eiff AWv, Neus H, Otten H. Health effects of environmental noise on man. Results of a prospective study. In: Zwicker E, ed. *Inter-noise 85. Proceedings of the 1985 International Congress and Exhibition on Noise Control Engineering in München*, vol II. Bremerhaven: Wirtschaftsverlag NW; 1985:961–964.
 38. Miedema HME, Vos H. Exposure-response relationships for transportation noise. *J Acoust Soc Am*. 1998;104:3432–3445.
 39. Gierke HEv, Eldred KM. Effects of noise on people. *Noise/News International*. 1993;6:67.
 40. Babisch W. Epidemiological studies of the cardiovascular effects of occupational noise—a critical appraisal. *Noise Health*. 1998;1:24–39.