

Long-Term Exposure to Road Traffic Noise and Myocardial Infarction

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Background: An association has been reported between long-term exposure to road traffic noise and the risk of myocardial infarction (MI), but the evidence is limited and inconclusive. No previous study has simultaneously analyzed the role of exposure to noise and air pollution from road traffic in the risk of MI.

Methods: A population-based case-control study on MI was conducted 1992–1994 in Stockholm County. Participants answered a questionnaire and underwent a physical examination. Residential exposure to noise and air pollution from road traffic between 1970 and 1992–1994 was assessed for 3666 participants (1571 cases of MI and 2095 controls), based on residential history combined with information on traffic intensity and distance to nearby roads. Information was also obtained on factors potentially affecting the relationship between noise exposure and MI, such as noise annoyance.

Results: The correlation between long-term individual exposure to noise and air pollution from traffic was high ($r = 0.6$). The adjusted odds ratio for MI associated with long-term road traffic noise exposure of 50 dBA or higher was 1.12 (95% confidence interval = 0.95–1.33). In a subsample, defined by excluding persons with hearing loss or exposure to noise from other sources, the corresponding odds ratio was 1.38 (1.11–1.71), with a positive exposure–response trend. No strong effect modification was apparent by sex or cardiovascular risk factors, including air pollution from road traffic.

Conclusions: The results lend some support to the hypothesis that long-term exposure to road traffic noise increases the risk for MI.

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Road traffic noise is a problem in urban areas worldwide.¹ Acute noise exposure is a stressor that activates the sympathetic and endocrine systems, leading to short-term changes in blood pressure, heart rate, and to elevated levels of stress hormones.^{2–4} Physiologic effects are observed not only at high sound levels in occupational environments, but also at levels of transportation noise typically found in residential settings.^{5,6} It has been hypothesized that long-term residential exposure to transportation noise may cause permanent vascular effects, with hypertension and ischemic heart disease as potential outcomes.⁷

Several studies indicate an increased risk of hypertension related to aircraft noise exposure.^{8–10} A corresponding effect of road traffic noise has also been suggested, although the evidence is weaker.^{11–13} Myocardial infarction (MI) in relation to traffic noise exposure has been less well studied, and the results from cross-sectional studies on aircraft or road traffic noise are inconclusive.⁷ There are 3 longitudinal studies on transportation noise and MI, all focusing on road traffic noise. Studies in Caerphilly and Speedwell found a moderate effect of road traffic noise on MI and an analysis of survivors as well as newly recruited subjects suggested an increased risk among those with high exposure.¹⁴ A recent case-control study in Berlin indicated an increased risk of MI among residents exposed to long-term road traffic noise.^{15,16} These results were consistent with those of a previous case-control study conducted in the same city and using similar methodology on a sample of men only.¹⁷ Traffic-related air pollution may also be a risk factor for cardiovascular disease,^{18,19} but no study has yet incorporated exposure to both noise and air pollution, making it difficult to disentangle effects by the 2 types of exposure.

The aim of the present study was to assess the risk for MI in relation to long-term residential exposure to noise from road traffic, including detailed assessment of traffic-related air pollution and an extensive set of risk factors for cardiovascular disease.

METHODS

Study Subjects

The present study is based on the Stockholm Heart Epidemiology Program conducted in Stockholm County. The

Stockholm study has been described in detail elsewhere.²⁰ Briefly, all men and women living in Stockholm County between 1992 and 1994 in the age range of 45–70 years with no history of MI were included in the study base. The cases included subjects with their first MI during 1992–1994 and were selected at all emergency hospitals in Stockholm County, from coronary records, hospital discharge registers, and from the National Cause of Death Register at Statistics Sweden. The diagnostic criteria for case inclusion were those applied by the Swedish Association of Cardiologists.²¹ In addition, we included patients with myocardial necrosis detected at autopsy that could be related to the relevant time of disease onset. Controls were randomly selected from the study base matched on age, sex, and hospital catchment area. To include at least as many controls as cases, additional controls were selected to compensate for expected lower response rates in this group. In total, 5452 study subjects were included: 2246 cases and 3206 controls. The 603 individuals who died within 28 days of the MI onset were classified as MI fatalities. The questionnaire response rate among cases was 72% for women and 81% for men. The corresponding rates for controls were 70% and 75%.

Questionnaire answers were available for 4067 study subjects. For fatal cases, the questionnaire was answered by next-of-kin. We excluded individuals missing more than 5 years of address history since 1970 or who lived outside Stockholm County for more than 5 years ($n = 401$). This left 3666 study subjects; 1571 cases (including 338 fatal), and 2095 controls.

Exposure Information

The questionnaire, distributed in 1992–1994, focused on a large set of potential risk factors for MI, including the physical and psychosocial work environment, social factors, and lifestyle factors such as smoking and physical activity. A supplementary telephone interview was conducted to reduce nonresponse and missing data. Various biologic parameters related to cardiovascular disease were measured in a special health examination performed on cases and controls. The examination was performed at least 3 months after the MI onset for the cases after expected recovery of metabolic stability. The biologic variables for analysis were based primarily on data from the health examination, but some (eg, body mass index) were supplemented with questionnaire data for the 10% of the surviving subjects who answered the questionnaire but did not participate in the clinical testing.

Hypertension was defined as reported use of antihypertensive drugs in the questionnaire, or as a systolic blood pressure of 170 mm Hg or higher, or a diastolic pressure of 95 mm Hg, or higher at the physical examination.

Overweight was defined as a body mass index of 27 kg/m² or more, which corresponded to about the 75th percentile for all controls, calculated from measurements obtained at

the health examination or from values reported in the questionnaire.

Diabetes mellitus was defined on the basis of data from either the questionnaire or the health examination, and included subjects who reported use of insulin, diabetic drug treatment, or diet control at the time of inclusion, or whose fasting blood glucose level exceeded 6.7 mmol/L at the health examination.

Physical inactivity was determined from questions on conditions at work and at home, as well as leisure-time activities. Subjects who reported inactive leisure time (including only occasional walks) during the last 5–10 years were categorized as physically inactive.

Smoking habits were classified into 5 categories: never-smokers, ex-smokers, and current smokers smoking 1–10, 11–20, or more than 20 g of tobacco daily as an average for the period during which the subject had been smoking. Subjects who had never smoked regularly for at least a year were classified as never-smokers. Those who had stopped smoking for more than 2 years before inclusion were classified as ex-smokers. Subjects who smoked at enrollment or had stopped smoking within the last 2 years were classified as smokers.

Air pollution exposure was assessed based on a methodology developed for a previous study on air pollution and lung cancer in Stockholm.²² The method has been further refined and is described in detail elsewhere.^{18,23} Briefly, various air pollutants were assessed from databases including historical information on emissions from road traffic and residential heating in Stockholm County back to 1960. Combining the emission data bases with dispersion models made it possible to estimate the geographic distribution of air pollution levels (annual averages) throughout the county, with a resolution down to 25 × 25 m. We used the time-weighted average traffic-generated NO₂ level at all residential addresses of each study subject during 1970 to 1992–1994 as an indicator of the individual long-term exposure to traffic-related air pollution at home.

Socioeconomic status was determined from questions regarding professional background according to a Swedish socioeconomic index classification of occupations.²⁴ A 3-level variable was constructed, including blue collar workers, low-level white collar workers, and high-level white collar workers,¹⁸ and using the most recent working period before inclusion in the study.

Job strain was defined as the ratio of the sum scores of the 5 questions on psychologic demands and 6 questions on decision latitude in the Swedish version of the Karasek–Theorell questionnaire.²⁵ Subjects with a ratio above the 75th percentile among all controls were classified as being exposed to job strain.²⁰

Noise Exposure Assessment

The road traffic sound level was assessed for all addresses of each study subject from 1970 until the subject

entered the study in 1992–1994. For incomplete addresses, additional information was gathered from national registers or from parish records. For each address, the 24-hour average A-weighted sound pressure level ($L_{Aeq,24h}$) from road traffic was calculated according to a simplified version of the Nordic prediction method.²⁶ The input parameters were distance and angles to roads, the number of vehicles on each road, and vehicle speed. The angles took into account shielding buildings 2-m above ground. We obtained traffic information from the municipalities, and used maps and area photos to measure distance and angles from the subject's most exposed facade to the 3 nearest roads. Contributions of distant large roads (>20,000 vehicles per 24-hour annual mean level) were assessed separately, using the same method but ignoring effects of shielding buildings. The final exposure estimate combined the levels from distant and nearby roads.

Four research assistants conducted the road traffic noise assessments. They were blind as to whether addresses belonged to cases or controls. To assess interrater reliability, the assistants independently assessed a subsample of addresses. The interrater reliability was found to be high; between-set correlations among the 4 sets of calculated sound levels ranged from 0.84–0.94 based on a subsample of 30 addresses. An acoustic consultant calculated sound levels for the same subsample by using the complete Nordic prediction method. The correlation between the 4 assistants' calculations and those of the consultant ranged from 0.72–0.77.

A total of 38% of the 3666 subjects had only 1 address during the exposure period, 32% had 2 addresses, 17% had 3, and 13% had 4 or more. We used 2 methods to combine levels from several addresses to form a single value of road traffic noise exposure. The first method was a time-weighted average of A-weighted decibel (dBA) values:

$$L_{dB - average} = \frac{\sum L_i Y_i}{\sum Y_i}, \quad (1)$$

where L_i is the sound level ($L_{Aeq,24h}$) of road traffic noise at address i and Y_i is the number of years of residence at address i . The second method was an energy-based time-weighted average:

$$L_{energy - average} = 10 \log \left(\frac{\sum 10^{L_i/10} Y_i}{\sum Y_i} \right). \quad (2)$$

For persons missing less than 5 years in their residential histories or with an address that could not be associated with a noise exposure (eg, addresses to post office boxes), the mean sound level of the control group was imputed. Sound levels were imputed for 210 of the 7700 addresses. Imputed values corresponded to 1313 (1.6%) of the total 84,163 exposure-years.

The available data on railway noise exposure were of varying quality and in different formats. Therefore a crude

assessment had to be used; subjects who had lived for at least 1 year within 200 m of a railway were classified as railway noise exposed; remaining subjects were classified as unexposed.

Aircraft noise exposure was assessed using noise maps for the year 1997 at the Bromma and Arlanda airports, with a method used in previous studies.^{9,10} These maps indicated levels greater than 55 dBA time-weighted 24-hour equivalent sound level, with a 5- and 10-dBA penalty for evening and night-time traffic, respectively.²⁷

We used a job exposure matrix to assess occupational noise exposure. The matrix is described in detail elsewhere.²⁸ Briefly, the exposure matrix was derived from measurements performed by occupational medicine clinics and occupational health services units in Sweden during the period 1970–2004. Estimated exposure for each occupation was coded as less than 75 dBA, 75–84 dBA, or 85 dBA or higher ($L_{Aeq,working-hours}$) based on the consensus of 3 occupational hygienists. The job exposure matrix included 320 occupations and was based on up to 10 different measurements per occupation. We used the job exposure matrix together with questionnaire information on type and duration of occupation to calculate the level and duration of each subject's occupational noise exposure. Subjects were classified as noise-exposed if they had had an occupational exposure of 75 dBA or higher for more than 1 year.

In 2003, a supplementary questionnaire was distributed to enhance the noise exposure assessment. This included questions on hearing impairment, window insulation, bedroom orientation, and noise annoyance. If the case or control had died, the questionnaire was sent to the next-of-kin. The questionnaire response rate among cases was 66% for women and 68% for men, and for controls, 76% for women and 79% for men.

Annoyance by noise was dichotomized as "annoyed" or "not annoyed," where annoyed included participants reporting mild, moderate, or strong annoyance. Participants with multiple addresses were classified according to their latest address (mean residence time 16 years). Noise annoyance from various sources such as transportation, ventilation, and neighbors was also defined from the supplementary questionnaire. Subjects who had been disturbed daily or weekly were classified as "noise-annoyed" from that source. Participants reporting sleep disturbance 2 or more times per month were categorized as sleep disturbed. Use of earplugs to reduce sleep disturbance due to noise was dichotomized as "each day or couple of times a week/month" and "never." Subjects who reported use of hearing aids were classified as hearing impaired.

Statistical Methods

Odds ratios and 95% confidence intervals were calculated using unconditional logistic regression analyses. Analysis of cases as nonfatal and fatal was performed by multinomial logistic regression using controls as reference.

Regression coefficients were adjusted for the matching variables age (5 categories), sex and catchment area (10 categories), and for a number of covariates. Each of a set of potential cofounders was evaluated against a preliminary multivariate logistic regression model, predicting MI from road traffic noise exposure and matching variables. A covariate was selected if the inclusion of this variable in the preliminary model changed the regression coefficient for noise exposure by more than 10%. The covariates included in the fully adjusted regression model were diabetes (dichotomous), physical activity (dichotomous), and smoking (5 categories). In addition, we also adjusted for air pollution (continuous) and occupational noise exposure (dichotomous), although these changed the regression coefficient for road traffic noise exposure by only 7% and 3%, respectively. All statistical analyses were performed with STATA 8.0 (Stata Corp., College Station, TX).

Ethics

Ethical approval for the study was obtained from the Ethical Committee at Karolinska Institutet in accordance with the Declaration of Helsinki of the World Medical Association.

RESULTS

Table 1 shows the distribution of cases and controls for various MI risk factors and covariates. Current smoking, physical inactivity, diabetes mellitus, and overweight were more common among cases than controls, and a similar tendency was seen for job strain, occupational noise exposure, and hypertension. To provide information on the relation between noise exposure and MI risk factors, the table also shows the distribution of road traffic noise exposure among controls according to the same variables using 50 dBA as the cut-off value. The road traffic noise-exposed controls

TABLE 1. Selected Characteristics of Myocardial Infarction Cases and Population Controls in Stockholm 1992–1994, and of Population Controls by Road Traffic Noise Exposure

Variables	Case Status		Road Traffic Noise Exposure ^a (Controls Only)	
	Case n = 1571 (%) ^b	Control n = 2095 (%) ^b	<50 dB n = 1397 (%) ^b	≥50 dB n = 698 (%) ^b
Sex (men)	1067 (68)	1374 (66)	944 (68)	430 (62)
Age (y)				
45–50	150 (10)	202 (10)	149 (11)	53 (8)
51–55	211 (13)	273 (13)	195 (14)	78 (11)
56–60	298 (19)	393 (19)	284 (20)	109 (16)
61–65	355 (23)	475 (23)	319 (23)	156 (22)
66–70	577 (35)	752 (36)	450 (32)	302 (43)
Smoking				
Never	390 (25)	853 (41)	578 (41)	275 (39)
Former	392 (25)	630 (30)	433 (31)	197 (28)
Current, 1–10 g/d	116 (7)	125 (6)	78 (6)	47 (7)
Current, 11–20 g/d	456 (29)	369 (18)	236 (17)	133 (19)
Current, >20 g/d	217 (14)	118 (6)	72 (5)	46 (7)
Physical inactivity	887 (52)	776 (37)	483 (35)	293 (42)
Diabetes	317 (20)	153 (7)	92 (7)	61 (9)
Socioeconomic status				
Blue collar	618 (40)	669 (32)	445 (32)	224 (32)
Low-level white collar	297 (19)	431 (21)	282 (20)	149 (21)
High-level white collar	618 (40)	987 (47)	666 (48)	321 (46)
BMI ≥27	637 (42)	638 (30)	419 (30)	209 (30)
Job strain	490 (33)	515 (25)	345 (25)	170 (25)
Occupational noise exposure (≥75 dB for at least 1 y)	666 (44)	768 (37)	449 (34)	238 (31)
Railway noise exposure (≤200 m from a railway track)				
High traffic-related air pollution ^c	822 (52)	1048 (50)	526 (38)	522 (75)
Hypertension	550 (36)	571 (27)	377 (27)	194 (28)

^aTime-weighted average of road traffic sound levels ($L_{Aeq,24h}$) for each of the subject's addresses during the exposure period (from 1970 to 1992–1994).

^bTotal number of subjects within each variable may vary slightly due to missing data.

^cEstimated time-weighted exposure to air pollution from traffic at residential addresses during the exposure period using NO₂ as indicator. The median level among controls (12.9 $\mu\text{g}/\text{m}^3$) was used for classification into high or low exposure.

were older and less physically active than those in the low-exposure group. Occupational noise exposure was slightly less common among road traffic noise-exposed controls compared with nonexposed controls, whereas railway noise exposure was more common. As expected, air pollution was strongly related to road traffic noise exposure. Hypertension was not strongly related to road traffic noise exposure among the controls (OR = 1.11, 95% CI = 0.87–1.41, after adjustment for sex, age, catchment area, diabetes, physical activity, smoking, air pollution, and occupational noise).

Figure 1 shows the association between long-term individual exposure to air pollution (NO₂) and noise from road traffic (dB L_{Aeq,24h}). The correlation was high (0.6) between the 2 variables. We used a cut-off value of 42 dBA in the

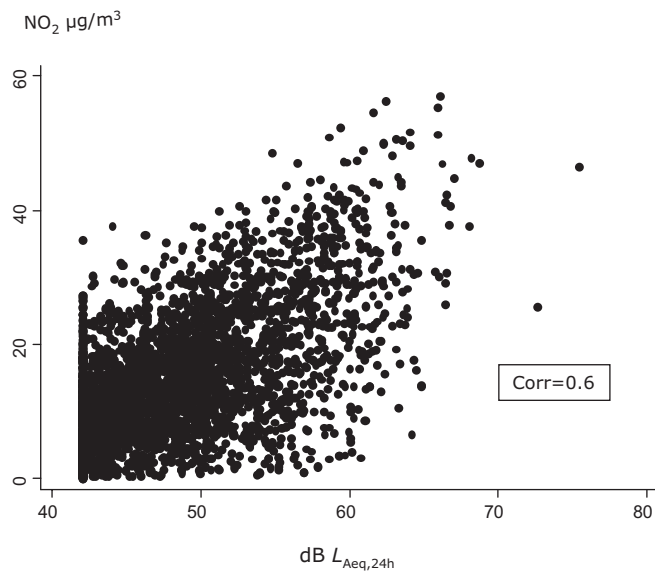


FIGURE 1. Correlation between individual long-term exposure to air pollution (NO₂ µg/m³) and noise (dB L_{Aeq,24 hours}) from road traffic in a case-control study on MI from Stockholm.

noise exposure assessment, and no detailed estimation was performed below this level.

We calculated the average road traffic noise exposure over the entire exposure period in 2 ways, as the average dBA value (Eq. 1) or as the energy average expressed in dBA (Eq. 2). These 2 measures were highly correlated (*r* = 0.95), and the median difference between the 2 measures was only 0.1 dBA. In the following analyses, we have calculated exposure as the average dBA value (Eq. 1).

Table 2 shows odds ratios (ORs) and 95% confidence intervals (CIs) for MI in relation to the reference exposure category (<50 dBA). Road traffic noise exposure of 50 dBA or higher was associated with an OR for MI of 1.12 (95% CI = 0.95–1.33). There was a suggestive exposure-response trend (OR for linear increase over 5 dBA categories of 1.06, 0.95–1.16). Similar results were found for fatal and nonfatal cases.

Figure 2 shows associations (ORs) between MI and road traffic noise exposure (≥50 dBA) for subsamples of subjects, defined by excluding persons where exposure misclassification regarding noise is more likely. Subsamples of subjects were identified using 5 exclusion variables: railway noise exposure (743 subjects), aircraft noise exposure (17), ventilation noise annoyance (147), neighbor-noise annoyance (289), and hearing impairment (297). The exclusion of subjects exposed to railway noise resulted in an OR for MI of 1.23 (1.01–1.50). Other exclusions appeared to have a more modest impact on the risk estimates. However, in a subsample excluding all 1207 persons exposed to other noise sources or with hearing impairment, the MI risk associated with road traffic noise was clearly elevated (1.38; 1.11–1.71). For this model, risk estimates indicated an increasing trend with exposure and the OR for linear trend over road traffic noise categories was 1.18 (1.03–1.36). As an alternative to the subsample analyses, we calculated the OR for the full sample of subjects, adjusted for the 5 exclusion variables and for matching factors, air pollution, and occupational noise

TABLE 2. Association (OR) Between Long-Term Road Traffic Noise Exposure (L_{Aeq,24h}) and Myocardial Infarction in a Population-Based Case-Control Study in Stockholm

Road Traffic Noise (dB) ^a	Controls No.	All Cases		Fatal Cases		Nonfatal Cases	
		No.	OR ^b (95% CI) ^b	No.	OR ^b (95% CI) ^b	No.	OR ^b (95% CI) ^b
Dichotomous							
<50	1369	923		155		768	
≥50	683	543	1.12 (0.95–1.33)	113	1.18 (0.87–1.61)	430	1.12 (0.93–1.33)
Categorical							
<50	1369	923		155		768	
50–54	384	312	1.15 (0.95–1.39)	62	1.20 (0.85–1.69)	250	1.14 (0.93–1.39)
55–59	214	161	1.05 (0.81–1.36)	36	1.12 (0.72–1.76)	125	1.04 (0.79–1.36)
≥60	85	70	1.21 (0.83–1.77)	15	1.24 (0.64–2.42)	55	1.21 (0.81–1.82)

^aTime-weighted average of road traffic sound levels (L_{Aeq,24h}) for each of the subject's addresses during the exposure period (from 1970 to 1992–1994).

^bOdds ratios and 95% confidence intervals adjusted for matching criteria (sex, catchment area, and age) and for smoking, physical inactivity, diabetes, air pollution, and occupational noise exposure.

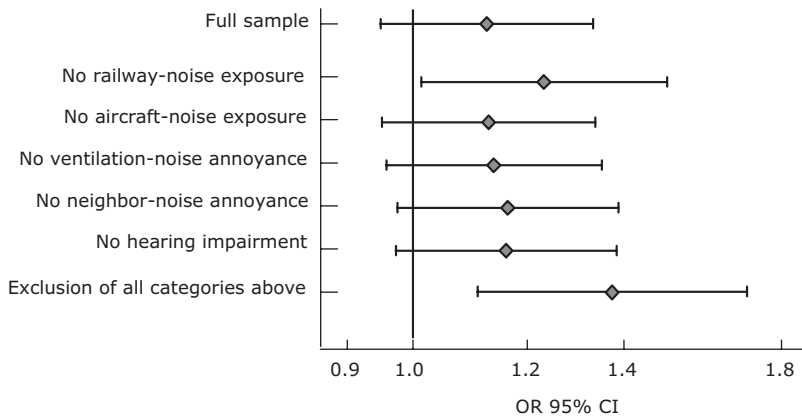


FIGURE 2. Associations (OR, 95% CI) between road traffic noise exposure (cut-off ≥ 50 dB $L_{Aeq,24h}$) and MI for subgroups defined by excluding persons with noise exposure from other sources or reported hearing loss in a case-control study from Stockholm. All models were adjusted for matching variables (age, sex, and catchment area) and for diabetes, physical inactivity, smoking, air pollution, and occupational noise exposure.

TABLE 3. Exposure to Noise and Air Pollution From Road Traffic in Relation to the Risk of Myocardial Infarction in a Case-Control Study in Stockholm

Air Pollution ($\mu\text{g}/\text{m}^3$ NO_2) ^a	Noise (dBA) ^a								
	<50			50–57			>57		
	Control No.	Cases No.	OR (95% CI) ^b	Control No.	Cases No.	OR (95% CI) ^b	Control No.	Cases No.	OR (95% CI) ^b
<17	1092	732	—	226	163	0.96 (0.76–1.22)	31	30	1.30 (0.76–2.24)
17–28	240	162	0.93 (0.73–1.18)	186	161	1.24 (0.97–1.60)	62	55	1.16 (0.77–1.73)
>28	21	20	1.28 (0.65–2.50)	71	58	0.97 (0.66–1.43)	105	72	0.95 (0.67–1.33)

^aAir pollution and noise exposure using the 65th (air: $17 \mu\text{g}/\text{m}^3$ NO_2 ; noise: 50 dB ($L_{Aeq,24h}$)) and 90th (air: $28 \mu\text{g}/\text{m}^3$ NO_2 ; noise: 57 dB ($L_{Aeq,24h}$)) percentile for categorization.
^bOdds ratios and 95% confidence intervals adjusted for matching criteria (sex, catchment area, and age) and for smoking, physical inactivity, diabetes, and occupational noise exposure.

exposure. The adjusted OR for MI associated with road traffic noise exposure (≥ 50 dBA) was 1.13 (0.95–1.33).

We performed extensive analyses on possible effect modification of the relation between road traffic noise exposure and MI by various risk factors, assessed in the questionnaire and health examination (eAppendix I, <http://links.lww.com/A727>). No differences were found between men and women, between physically active and inactive subjects, or between subjects with or without diabetes mellitus, and no clear trend in ORs was found for groups of subjects defined according to age group, smoking habits, or socioeconomic status. There was the suggestion of higher risk among former smokers and low-level white collar workers. The OR related to noise exposure tended to be higher for subjects with job strain than for subjects with no job strain. A higher OR was also suggested for hypertensive compared with nonhypertensive subjects, with ORs of 1.27 (0.94–1.72) and 1.06 (0.86–1.31), respectively.

Furthermore, we analyzed the association between road traffic noise exposure (≥ 50 dBA) for the whole exposure period and MI by various noise-related variables assessed in the supplementary questionnaire (eAppendix I, <http://links.lww.com/A727>). We found no substantial differences in risk estimates among groups of subjects defined according to

these variables, with the exception that the risk of MI associated with road traffic noise exposure was particularly elevated in those annoyed by noise in the bedroom (OR 1.61; 1.02–2.54), in comparison with other rooms (OR 0.83; 0.52–1.32). No elevated risk was apparent in participants reporting sleep disturbance due to noise.

Table 3 shows the risk of MI in subcategories defined by residential exposure to air pollution (NO_2 $\mu\text{g}/\text{m}^3$) and noise ($\text{dBL}_{Aeq,24h}$) from road traffic. The reference category includes individuals with low exposure to both air pollution and noise from road traffic. No strong interaction effect in relation to MI risk is seen between the 2 variables. If anything, an elevated risk was suggested in the categories with high exposure to either air pollution or noise from road traffic.

DISCUSSION

Our findings lend some support to the hypothesis that long-term exposure to residential road traffic noise increases the risk for MI. Subjects exposed to road traffic noise of 50 dBA or higher ($L_{Aeq,24h}$) since 1970 tended to have an increased risk for MI compared with subjects having an exposure of less than 50 dBA, with a suggested exposure-response relationship. The odds ratio was higher after exclud-

ing persons with noise exposure from other sources or reported hearing loss. Overall, our results are in agreement with previous longitudinal studies on road traffic noise and MI.^{14–17} We found no clear differences in risk related to sex, as had been found in some earlier studies.^{15,16}

Risk estimates related to road traffic noise were more clearly elevated after exclusion of subjects with reported annoyance due to noise from other sources, primarily railways. This suggests that the association in the full sample may have been diluted due to misclassification of total noise exposure. Although studies on railway noise and cardiovascular endpoints are lacking, adverse effects of railway noise, such as annoyance and sleep disturbance, are well documented.^{29,30} A recent study on aircraft noise exposure and incidence of hypertension showed stronger effects in those not annoyed by noise from other sources,¹⁰ supporting the interpretation that exposure misclassification may dilute associations for those exposed to noise from several sources. Subjects with self-reported hearing impairment were excluded because earlier evidence suggests that hearing loss may protect against nonauditory health effects of occupational noise³¹ and residential aircraft noise.⁹ However, analysis with exclusion variables entered as covariates in the regression model provided similar results as in the analysis based on the full sample, suggesting that selection bias may have affected the results based on the subsamples.

Risk estimates for MI appeared particularly elevated for participants reporting noise annoyance mostly in their bedroom. This finding is in line with previous studies showing that noise induces cardiovascular effects during sleep³² and that disrupted sleep may be a risk factor for MI.^{33–35} Taken together, the evidence suggests that night-time noise exposure may be of particular health importance. It is also possible that this elevation is a consequence of more precise exposure estimates. The participants were generally at home during the night making the exposure assessment based on the residential address most relevant for this time period.

Earlier studies have suggested an association between traffic-related air pollution and MI.^{18,19} Given the high correlation between air pollution and noise, it is plausible that the association between noise and MI may be confounded by air pollution. However, our data suggest independent effects of noise and air pollution on the risk of MI, with traffic-related air pollution affecting primarily mortality.¹⁸ There was no strong evidence of an interaction between exposure to noise and air pollution on the risk of MI. It should be noted that this analysis lacked power because of the strong correlation between the 2 types of exposures.

Previous studies have suggested an association between road traffic noise and hypertension.^{11–13} In our study, there was no clear association between road traffic noise and hypertension among controls. However, we found that the association between road traffic noise and MI appeared ele-

vated in those with hypertension. This agrees with the notion that effects of road traffic noise on MI may partly be mediated by hypertension, which is a well-known risk factor for cardiovascular disease.³⁶

The major strengths of the present study were the longitudinal and objective assessment of road traffic noise exposure, precise assessment of disease outcome, and extensive information on a number of covariates including air pollution and occupational noise exposure. The detailed road traffic noise exposure assessment for all addresses since 1970 made it possible to estimate the exposure for a longer period (22–24 years) than in previous longitudinal studies (up to 15 years¹⁷). A further strength was that our exposure assessment was based on information on traffic intensity including small municipal roads. This made it possible to obtain exposure assessment from 50 dBA and above. In comparison, the Berlin studies evaluated risk in comparison to a 60-dBA or lower ($L_{Aeq,6-22h}$) exposure category.^{15,17} Our results suggest that an effect of noise on MI may exist already at levels above 50 dBA ($L_{Aeq,24h}$). This is consistent with research indicating an increased risk for hypertension at exposures of greater than 50 dBA (FBN) of aircraft noise^{9,10} or greater than 50 dBA ($L_{Aeq,24h}$) of road traffic noise.¹²

Misclassification of disease is probably negligible in the present study, given the strict diagnostic criteria, the high reliability of case identification, and the low probability of selection bias.^{21,37,38} However, misclassification of exposure should be considered. Some of our data on road traffic volume did not cover the relevant time period, and exposure variation related to the floor of apartment buildings was not considered. We used a simplified assessment model that, although found to be of high validity, would be less precise than more detailed methods.²⁶ The resulting biases would probably be unrelated to disease outcome and lead to attenuation of the effect estimates. Moreover, only residential exposure was considered, even though road traffic noise is common in other settings such as work places and recreational areas. This imprecision in assessment of total road traffic noise exposure may further have attenuated the estimated effect. It should be noted that it was not possible to differentiate between effects of short- and long-term exposure because of the low mobility of the population during the study period.¹⁸ Nonetheless, our study lends some support to the hypothesis that long-term exposure to road traffic noise is associated with an increased risk for MI.

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