ORIGINAL ARTICLE

Road traffic noise and hypertension

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Background: It has been suggested that noise exposure increases the risk of hypertension. Road traffic is the dominant source of community noise exposure.
Objective: To study the association between exposure to residential road traffic noise and hypertension in an urban municipality.
Methods: The study population comprised randomly selected subjects aged 19–80 years. A postal questionnaire provided information on individual characteristics, including diagnosis of hypertension. The response rate was 77%, resulting in a study population of 667 subjects. The outdoor equivalent traffic noise level (Leq 24 h) at the residence of each individual was determined using noise-dispersion models and manual noise assessments. The individual noise exposure was classified in units of 5 dB(A), from <45 dB(A) to >65 dB(A).
Results: The odds ratio (OR) for hypertension adjusted for age, smoking, occupational status and house type was 1.38 (95% confidence interval (CI) 1.06 to 1.80) per 5 dB(A) increase in noise exposure. The association seemed stronger among women (OR 1.71; 95% CI 1.17 to 2.50) and among those who had lived at the address for >10 years (OR 1.93; 95% CI 1.29 to 2.83). Analyses of categorical exposure variables suggested an exposure–response relationship. The strongest association between exposure to traffic noise and hypertension was found among those with the least expected misclassification of true individual exposure, as indicated by not having triple-glazed windows, living in an old house and having the bedroom window facing a street (OR 2.47; 95% CI 1.38 to 4.43).
Conclusion: The results of our study suggest an association between exposure to residential road traffic noise and hypertension.

Noise acts as a ubiquitous stress-mediating factor in the physical environment. General annoyance, disturbances in psychosocial well-being and reduction in sleep quality are commonly reported effects of noise exposure.[1 2] An increased risk of non-auditory physiological effects due to noise, such as hypertension and ischaemic heart disease, have also been suggested.[3–8] Most previous studies have been performed in occupational settings with high noise levels.[5 6 8–10] Community noise is less well studied.

Road traffic is the dominating source of community noise in the urban environment. Few studies have investigated an association between exposure to road traffic noise and hypertension, and the results are conflicting.[3 4 11] Studies in this field have low precision and validity problems, including crude exposure assessments, selection bias and limited control of important confounding factors. Exposure has usually been assessed either from subjective reports or without consideration of important factors that may influence the individual exposure level—for example, window type, bedroom window orientation and type of residence.

The suggested biological mechanism for an association between exposure to community noise and hypertension is that noise induces stress by disturbing sleep and interfering with relaxation and concentration and many other cognitive effects that activate the sympathetic nervous system and the endocrine system.[12] The primary physiological effects of noise exposure are vegetative reactions such as increase in blood pressure, heart rate and finger pulse amplitude, cardiac arrhythmia, and changes in respiration and body movements.[13] Therefore, a hypothesis has emerged that stress due to persistent exposure to environmental noise could result in permanent vascular changes, with increased blood pressure and ischaemic heart disease as potential outcomes.[14–16]

Our objective was to study a possible association between exposure to residential road traffic noise and hypertension among adults in an urban municipality. To better characterise individual noise exposure, we aimed at investigating factors that may influence the true exposure level, such as window type, bedroom window orientation and type of residence.

METHODS

Study population

The study was performed in a municipality with 55 000 inhabitants located 15 km north of Stockholm City. A questionnaire designed for a countywide investigation of health effects related to various environmental factors was distributed in April 1997 to 1000 individuals aged 19–80 years living in the municipality.[17] A stratified random sampling procedure was applied to ensure a sufficient number of subjects exposed to traffic noise, consisting of two strata with 500 residents in each. The noise-exposed group was drawn from those living within 100 m on each side of the highway, main roads or the railway. The other sample was drawn from the remaining parts of the municipality. Statistics Sweden performed the sampling by combining the National Population Register (containing background information for the study population) with the Real Estate Register (containing geographical coordinates for the residence of each individual). The response rate was 77% in both samples. This study focused on exposure to road traffic noise; thus, subjects who were residing close to the railway (n = 91) were not included. One subject who had removed the identification sticker from the returned questionnaire was excluded, as we did not have the address of that subject. In total, the study comprised 667 subjects.

Questionnaire

The survey included 87 questions and was mainly focused on prevalence of allergic diseases and environmental risk factors of regional importance. Information on educational level, employment status, general living conditions, and smoking habits was

Table 1 Number of study subjects in different noise exposure classes according to method of exposure assessment

Noise exposure	Classified by dispersion model: National Road Administration	Sollentuna municipality	Manually classified	Total
⩽45 dB(A)	0	0	125	125
45–50 dB(A)	0	0	120	120
50–55 dB(A)	96	0	209	305
55–60 dB(A)	20	25	47	92
60–65 dB(A)	2	9	12	23
>65 dB(A)	2	0	0	2
Total	120	34	513	667

provided. Data on annoyance from traffic noise and sleep disturbance due to noise were also collected. Hypertension was defined as a positive answer to the question ''Have you been diagnosed with hypertension by a physician during the past five years?''. Individual information on background factors such as age, sex and ethnic background was obtained from the National Population Register.

Exposure assessments

The exposure to outdoor residential road traffic noise during 1997 was assessed for all subjects in A-weighted average sound pressure levels (dB(A)) and expressed as the annual mean 24-h equivalent noise level (Leq 24 h). The individuals were classified into exposure categories of 5 dBA, from ⩽45 dB(A) to >65 dB(A), according to the noise level at their residence (table 1). For the six-lane highway that intersects the municipality, the Swedish National Road Administration calculated the noise propagation using a validated Nordic prediction model for road traffic noise.[18] This dispersion model covered the addresses of 120 subjects. The Sollentuna Environment and Health Protection Administration applied a similar prediction model to classify exposure around other major roads in the area in 55–60 and 60–65 dB(A) exposure categories. This model covered another 34 subjects. For residences not covered by any of these models (n = 513), the noise exposure was manually classified into groups of 5 dB(A) by an environmental health officer with extensive knowledge of the local traffic noise propagation and blinded to the outcome status. Important parameters for all assessment methods were traffic flow, geographical location and existing noise measurements.

Table 2 presents the characteristics of the study subjects, including living conditions and selected lifestyle factors.

Statistical analysis

The association between exposure to road traffic noise and hypertension was investigated using logistic regression, and is presented as odds ratios (OR) with 95% confidence intervals (95% CI). In most analyses, exposure to road traffic noise was used as a continuous variable and the results are presented per 5 dB(A) increase in noise exposure. As the exposure measure was determined in 5 dB(A) wide classes, the continuous variable used the class middle for everyone in that class. The top and bottom classes were open, and the subjects in these classes were given a value of 2.5 dB(A) from the nearest class boundary. In the analysis using a categorical exposure variable, subjects exposed to noise levels of 60–65 dB(A) and >65 dB(A) were merged with those exposed to 55–60 dB(A) owing to small numbers in the top categories of exposure. The final multiple logistic regression model included age as a linear term,

Table 2 Background characteristics of study subjects in Sollentuna, Sweden 1997

	n (%)	Mean (SD)
Age (years)		**48 (16)**
Sex		
Male	310 (46)	
Female	357 (54)	
Smoking		
Never smokers	341 (52)	
Former smokers	187 (28)	
Number of cigarettes		14 (8)
Current smokers	131 (20)	
Number of cigarettes		13 (8)
Occupation		
Within the working force*	465 (73)	
Retired	100 (15)	
Otherwise outside the working force†	74 (12)	
Type of residence		
Apartment	278 (42)	
Single family house	384 (58)	
Duration of residence (years)		
<1	75 (11)	
1–10	293 (44)	
>10	297 (45)	
Window type		
Triple glazing	418 (65)	
Double glazing	228 (35)	
Building year		
Up to 1975	423 (66)	
After 1975	215 (34)	
Bedroom window		
Facing the street	331 (51)	
Facing other	318 (49)	

*Employed, self-employed, on parental leave, student.
†Homemakers, unemployed, on disability or sick leave.

an indicator variable for house type, occupation in three levels, smoking status and amount smoked for former and current smokers. Model selection was based on the evaluation of the influence of each covariate on the effect estimate of exposure to road traffic noise on hypertension. Only those with complete data on all covariates were included in the multiple logistic regression analyses. To explore the potential modification of the effect of noise exposure, an interaction term between the covariate and the noise variable was included in the model, and p values for the interaction term are presented. All statistical analyses were performed with Stata V.8.2.

RESULTS

Altogether, 80 (13%) subjects in the whole study population were diagnosed with hypertension. The OR for hypertension adjusted for age, smoking, occupational status and house type was 1.38 (95% CI 1.06 to 1.80) per 5 dB(A) increase in exposure to road traffic noise. Analyses using categorical exposure variables suggested an exposure–response relationship between road traffic noise and hypertension (table 3). Analyses of potential modification of the effect of road traffic noise on hypertension by other factors showed a stronger association among those who had lived at their residence for >10 years (OR 1.93; 95% CI 1.29 to 2.83), those who lived in a house built before 1976 (OR 1.83; 95% CI 1.29 to 2.61) and those who had their bedroom windows facing the street (OR 1.82; 95% CI 1.22 to 2.70; fig 1). A stronger effect was also suggested for those living in single-family houses (OR 1.74; 95% CI 1.20 to 2.51) and those who did not have triple-glazed windows (OR 1.66; 95% CI 1.17 to 2.34). There was some indication that the effect was stronger among female subjects (OR 1.71; 95% CI 1.17 to 2.50), although the sex difference was not significant.

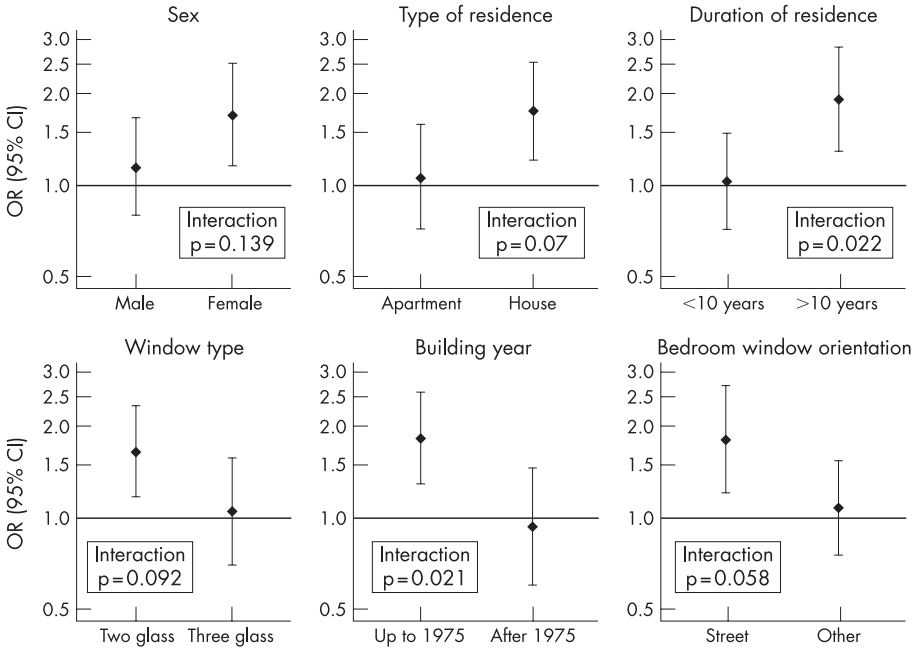

Figure 1 Odds ratios (OR) and 95% confidence intervals (CI) for hypertension associated with a 5 dB(A) increase in exposure to road traffic noise by sex, type of residence, duration of residence, window type, building year and bedroom window orientation. Odds ratios were adjusted for age, type of residence, occupation, smoking status and number of cigarettes.

To better characterise individual exposure, we used information on those residential factors that are likely to influence the true individual exposure level. The indicators of misclassification of true exposure were houses built after 1975 (when new Swedish regulations for building constructions including higher standards for thermal isolation were settled), houses equipped with triple-glazing (that reduce indoor noise levels) and bedroom windows not directly facing the street (that result in reduced exposure to night-time noise). The results indicate stronger associations with decreasing misclassification of the true individual noise exposure, up to an OR of 2.47 (95% CI 1.38 to 4.43) in the group where we expect least exposure misclassification (table 4).

Several variables exerted confounding on the association between exposure to traffic noise and hypertension, and were thus adjusted for in the analyses (age, residence type, occupational level and smoking). Many other potential confounders were also evaluated—for example, education and hearing loss, but these factors did not influence the effect estimate (fig 2). Excluding those who had lived at their residence for <1 year did not affect the results. In addition, there was virtually no difference in effect estimates using either of the different methods for noise exposure assessment separately, suggesting that these methods assessed exposure equally well.

DISCUSSION

We found an association between exposure to road traffic noise and hypertension. Other studies have reported an association between hypertension and occupational noise exposure[5 6 8]or exposure to aircraft noise.[19–21] There is a lack of previous epidemiological data linking exposure to road traffic noise and hypertension, although a few studies have suggested some association.[3 4] In addition, a recent study reported an increased risk of myocardial infarction among men associated with long-term exposure to road traffic noise.[7]

The results of our study point to a linear exposure–response relationship between road traffic noise and hypertension at lower noise levels compared with previous reports. In a study of noise exposure and annoyance or sleep disturbances in the same study population, we found a distinct exposure–response relationship.[22] These findings indicate that our method of determining individual exposure to road traffic noise is reasonably valid in terms of perception of noise exposure. In addition, we combined two ways of exposure assessment, using calculated dispersion models and manual classification, and both produced virtually the same results.

We evaluated confounding from many factors including smoking and occupational status, but residual confounding may still be present, especially from noise at other locations, for example, at work. In addition, some factors that may act as risk

Table 3 Association between exposure to road traffic noise and hypertension (n = 608)

	n	Number with hypertension (%)	OR (95% CI)*
Continuous (per 5 dB(A) increase)			1.38 (1.06 to 1.80)
Category (db(A))			
⩽45	115	6 (5)	1.00 (reference)
45–50	105	13 (12)	1.74 (0.60 to 5.01)
50–55	281	39 (14)	2.07 (0.82 to 5.24)
>55	107	22 (21)	3.47 (1.27 to 9.43)

Adjusted for age, type of residence, occupational status, smoking status and number of cigarettes.

Table 4 The association between exposure to road traffic noise per 5 dB(A) and hypertension according to the number of indicators of potential exposure misclassification (n = 559)

Indicators of exposure misclassification*	n	OR (95% CI)†
3 of 3	67	0.83 (0.43 to 1.60)
2 of 3	120	0.98 (0.56 to 1.73)
1 of 3	221	1.47 (0.93 to 2.33)
0 of 3	151	2.47 (1.38 to 4.43)

*Triple-glazed windows, building built after 1975; bedroom window not directly facing the street.
†Adjusted for age, type of residence, occupational status, smoking status and number of cigarettes.

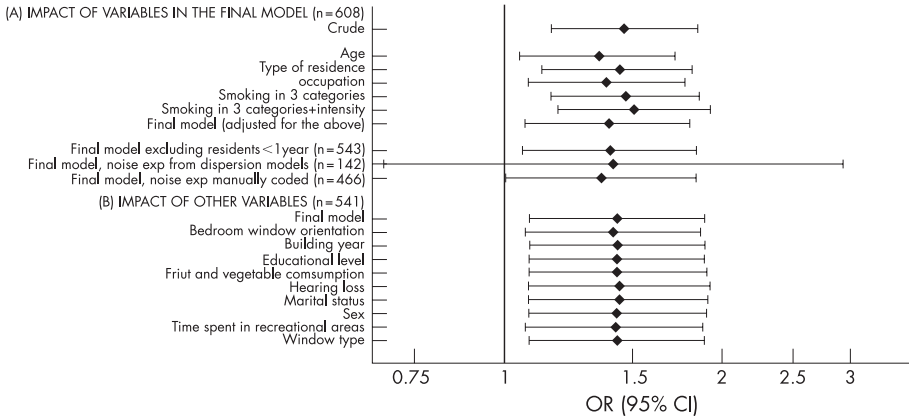

Figure 2 Odds ratios (OR) and 95% confidence intervals (CI) for hypertension associated with a 5 dB(A) increase in exposure to road traffic noise. (A) The individual and total confounding effect on the point estimate from the variables included in the final model; (B) the additional confounding effect from variables not included in the final model.

factors for hypertension were not recorded—for example, alcohol consumption and physical inactivity—although a strong association with exposure to residential road traffic noise seem unlikely, making bias due to confounding from such factors less likely.

Taking hearing loss into account did not seem to change the results. A positive relationship between hearing loss and hypertension has previously been found.[23] However, the scientific support for an association between hearing loss and hypertension is weak and somewhat speculative, and the interrelationship has to be interpreted with caution.

Disease outcome in this study was based on self-reported diagnosis of hypertension, which might be a source of bias. However, it has been reported that self-administered questionnaires may have good accuracy to confirm hypertension.[24] As exposure was assessed objectively by geographical dispersion models or by an operator blinded to disease status, the data on exposure and outcome were collected independently, making differential misclassification of exposure or disease less likely. In addition, the high response rates reduce the possibility that the results were strongly influenced by selection bias.

Although our study was cross-sectional, we had access to crude data on duration of residence in the categories <1 year, 1–10 year and >10 years. Stratification on that variable indicated an association primarily among those who had lived at the address for at least 10 years, suggesting that least misclassification of true individual exposure in that group or that 10 years of exposure might be needed to exert an effect. As non-differential misclassification of exposure is important to consider, we especially focused on several other factors that are likely to affect the individual exposure to road traffic noise. These include triple-glazed windows that have a noise-isolating effect, modern buildings that are better isolated and bedroom windows that do not directly face the street. All these factors are supposed to result in lower night-time exposure levels. Indeed, when none of these factors were present—that is, where we assume that the modelled exposure level better reflects the true individual exposure level—the association was particularly strong (OR 2.47; 95% CI 1.38 to 4.43).

The association seemed stronger among women than among men. Although there may be biological reasons—for example, the use of hormonal contraceptives that could explain such differences—it may also be due to chance or different patterns in misclassification of exposure. The relationship was also stronger among those living in single-family houses than among those living in apartments. These findings could partly be due to differences in building construction. Three glass windows were present in 50% of the apartments compared with 27% of the single-family houses.

In conclusion, our results suggest an association between residential exposure to road traffic noise and hypertension. This implies that road traffic noise may be a risk factor for cardiovascular disease.

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An ethics committee was consulted, but a formal approval was judged unnecessary for this study.

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