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ORIGINAL ARTICLE

Residential traffic noise exposure and vestibular schwannoma – a Danish case–control study

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ABSTRACT

Background: Few risk factors for sporadic vestibular schwannoma (VS) are known. Several studies have proposed an increased risk with occupational noise exposure, whereas no studies have investigated residential traffic noise exposure as a risk factor. The present study investigated if residential traffic noise was associated with vestibular schwannoma in a large, population-based Danish case–control study.

Material and methods: We identified 1454 VS cases, age above 30 years at diagnosis, between 1990 and 2007. For each case, we selected two random population controls, matched on sex and year of birth. Road and railway traffic noise at the residence was calculated for all present and historical addresses between 1987 and index date. Associations between traffic noise and risk for VS were estimated using conditional logistic regression, adjusted for education, disposable personal income, cohabitation status, railway noise exposure, municipal population density, and municipal income.

Results: A two-year time-weighted mean road traffic noise exposure was associated with an adjusted odds ratio of 0.92 (0.82–1.03) for developing VS, per 10 dB increment. There was no clear trend in categorical analyses. Similarly, linear and categorical analyses of residential railway noise did not suggest an association. We found no interaction with demographics, year of diagnosis, individual and municipal socioeconomic variables, and railway noise exposure. The results did not differ by tumor side, spread or size.

Conclusions: The present study does not suggest an association between residential traffic noise and VS.

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Introduction

Vestibular schwannomas (VS) arises from the eighth cranial nerve and accounts for approximately 6% of all intracranial tumors [1]. It is a benign tumor with low lethality, but it has profound negative health effects since surgery of the tumor, even when diagnosed at early stages, entails a number of adverse effects, including hearing loss and facial paralysis [2]. Hence, prevention would be the favored approach from a public health perspective. Unfortunately, little is known about the disease etiology for sporadic VS, but an association has been proposed for a number of environmental risk factors, including ionizing radiation, radio frequency electromagnetic fields in connection with mobile phone use and noise exposure [3,4].

Most [4–7], but not all [8], studies examining the association between self-reported occupational noise exposure and VS have found a direct association. In contrast, two studies estimating occupational noise exposure via a job exposure matrix did not find an association [3,9]. Some of these studies also investigated self-reported leisure-time noise exposure (e.g., shooting, engine noise, concerts, sports events, screaming children); two found no association [4,6], and two found a direct association [3,7]. However, the numbers were small for these analyses and the included types of noise sources and the definition of these varied considerably.

Little is known about the biological mechanisms that could explain an association between noise and VS. Animal studies have found that noise induces mechanical trauma to the eighth cranial nerve, and surrounding structures, including the cochlea [10,11], and it has been proposed that during repair of these damages, DNA errors could be replicated during cell division, resulting in pathological proliferation [3]. The mechanical stress has also been proposed to entail subsequent biological and molecular stress, including oxidative stress, excitotoxity, and inflammation [11]. Rodent studies examining gene expression following acoustic overstimulation suggested that the primary pathways affected were

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immune and inflammatory pathways [11]. A study of noise exposure in zebrafish found genes involved in cell proliferation, apoptosis and immune functions to be significantly regulated in the inner ear after exposure [12]. These processes are known to play a role in human carcinogenesis [13–16]. Furthermore, a study in chinchillas found that noise exposure entailed p53 upregulation in the cochlea and organs of Corti; playing a role in noise-induced cell death. This could also potentially link to human carcinogenesis, since p53 is the most frequently mutated tumor suppressor gene in human tumors [17]. Taken together, several potential pathways exist for an association between noise and VS.

To the best of our knowledge, no previous study has systematically examined the association between traffic noise exposure and VS. However, the biological mechanisms for an association should be plausible for this noise source in the same manner as previously investigated noise sources.

The aim of the present study was to investigate the association between residential traffic noise exposure in relation to VS in a population-based Danish case–control study. Furthermore, we aimed to investigate effect modification by demographics, year of diagnosis, individual and municipal socioeconomic variables, as well as railway noise exposure.

Material and methods

Study population

Cases of VS were identified through two sources: first, in Denmark, VS cases are treated mainly in one clinical center at Copenhagen University Hospital, Rigshospitalet, and this clinical database contains detailed information on each person referred for a diagnosis of VS, including diagnostic work-up to establish a confirmed diagnosis, treatment information, and pathology report [18]. We included information on the size of the tumor given as the extrameatal diameter in millimeters, measured at diagnosis, and the spread of the VS discriminated into intrameatal spread only or intra- and extrameatal spread, as well as information on whether the tumor was left- or right sided. Furthermore, cases were identified by linking to the nationwide population-based cancer registry to identify patients not registered in the clinical registry and vice versa. The Cancer Registry has almost complete coverage of the population since 1942 and contains detailed information on topography and morphology according to the International Classification of Diseases for Oncology (ICD-O), since 1978. We included cases with the ICD-10 code C72.4.

Eligible cases were born in Denmark, had no previous diagnosis of cancer (except nonmelanoma skin cancer), lived in a geocodable address in Denmark at time of diagnosis, were diagnosed between 1990 and 2007 and were above 30 years of age at time of diagnosis. Through the two above sources, we identified 1511 cases.

For each case, two random controls, matched on sex and year of birth, were selected from the civil registration system. Controls that were dead, emigrated or diagnosed with cancer (except nonmelanoma skin cancer) before diagnosis date of their matched case where excluded. Substitute controls, matched on sex and year of birth, and under risk at age at diagnosis of the matched case, where sampled from surplus controls from the present study. All controls in the final sample where required to be alive, free of cancer (except nonmelanoma skin cancer), born in Denmark and living at a geocodable address in Denmark at the time of diagnosis of matched case. Based on these criteria, we identified 3022 controls.

The present study was a strictly register-based study with no contact to the study population, and, therefore, the study required no permission from an ethical committee (according to Danish legislation). It was approved by the Danish Data Protection Agency (2012-41-0983), and it complies with the current laws of Denmark, in which the study was performed. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Exposure assessment

Residential address history for all cases and controls between 1 July 1987 and index date was collected using the Civil Registration System. Road traffic noise exposure was calculated at the most exposed facade for all present and historical addresses using SoundPLAN, a software implementing the joint Nordic prediction method for road traffic noise. Input variables were geographical coordinate, road links with information on annual average daily traffic, vehicle distribution, travel speed and road type; and building polygons for all Danish buildings. We obtained traffic data for all Danish roads with more than 1000 vehicles per day from a national database [19]. We assumed a flat terrain and that urban areas, roads and areas with water were hard surfaces and all other areas acoustically porous. No information was available on noise barriers. Road traffic noise was expressed as L_{den} (day, evening and night). Exposure to railway noise was calculated for all addresses using SoundPLAN, with implementation of NORD2000. Input variables were geographical coordinate, railway links with information on annual average daily train lengths, train types, travel speed; building polygons and noise barriers along the railway. Railway traffic noise was expressed as L_{den} at the most exposed facade.

Covariates

The following information on individual socioeconomic position was obtained by linkage to Statistics Denmark: Highest attained education, cohabiting status, and information on individually disposable income determined as household income after taxation and interest per person, adjusted for number of household persons and divided into quintiles based on Danish background population (age standardized). All information was assessed one year before index date. The following information on municipal level was furthermore included from linkage to Statistics Denmark: median equated

municipal disposable income in year 2000, and municipal population density in year 2000.

Statistical methods

We investigated the association using conditional logistic regression. For each person, we calculated the one- and two-year time-weighted mean exposure to road traffic and railway noise preceding index date, taking the complete residential history in that period into account.

Linearity of all continuous variables was investigated visually and by linear spline models with boundaries at the three quartiles among cases. No deviation from linearity was observed.

We calculated odds ratios (OR) and 95% confidence intervals (CI) for the risk of VS associated with exposure to road and railway traffic noise in two models, with increasing adjustment: Model 1 was adjusted for year of birth and sex (by design). Model 2 was additionally adjusted for education (basic school; vocational education; higher education), disposable personal income (Household income 1 year before index date after taxation and interest, adjusted for number of persons in the household and categorized by gender-specific distribution of disposable household income per person, in quintiles), cohabitation status (married/cohabiting, single, widow/widower or divorced), municipal population density (<150 persons/km^2, 150–499 persons/km^2, 500–1000 persons/km^2, >1000 persons/km^2), municipal income (median equated municipal disposable income in year 2000 according Statistics Denmark, in quartiles), and for opposite traffic noise source (road or railway). All analyses were run with strata for the matched case–control pairs.

Effect modification of the association between road traffic noise and VS was investigated by age (<median/≥median), sex, education (basic/vocational/higher), income (tertiles), year of diagnosis (<2000/≥2000), municipal income (quartiles), municipal population density (<150 persons/km^2, 150–499 persons/km^2, 500–1000 persons/km^2, >1000 persons/km^2) and railway noise exposure (yes/no), in order to explore potential effect modification. p values for heterogeneity across strata were calculated using χ^2 tests.

Furthermore, in sensitivity analyses, we investigated the association between 5- and 10-year time-weighted mean road traffic noise and VS among cases and their matched controls with 5 years or more of exposure history (1330 cases; 2314 controls) and 10 years or more of exposure history (1004/1712), respectively.

Correlations between variables were assessed using Spearman rank correlation coefficients (R_s).

We used SAS version 9.3 (SAS Institute, NC, USA).

Results

From a study population of 4533 individuals, we excluded 196 with missing address or exposure information at some point in time during the two years before index date, and 271 with missing information on covariates. After these exclusions, 30 cases had no matching controls and 32 controls had no matching case and were excluded. The final study population comprised of 4004 individuals; 1454 cases and 2550 controls.

Table 1. Characteristics of vestibular schwannoma cases and controls[a].

	Cases ($n = 1454$)	Controls ($n = 2550$)
Men	48.5	46.7
Age (years)	56.9 (35.9–73.3)	56.6 (35.7–73.1)
Education		
Basic	34.1	39.8
Vocational	40.9	40.0
Higher	25.0	20.2
Personal income[b]		
1st tertile (lowest)	15.8	18.0
2nd tertile	17.0	19.0
3rd tertile	20.8	20.8
4th tertile	21.9	20.9
5th tertile (highest)	24.5	21.3
Cohabitation status		
Married or cohabiting	72.4	69.0
Unmarried	10.6	11.6
Widowed	7.3	7.8
Divorced	9.4	11.6
Municipal income[c]		
1st quartile	19.5	21.3
2nd quartile	27.0	27.7
3rd quartile	26.1	25.0
4th quartile	27.4	26.0
Municipal population density		
>1000 persons/km^2	14.6	16.5
500–1000 persons/km^2	15.2	14.9
150–499 persons/km^2	17.1	15.3
<150 persons/km^2	53.2	53.3
Railway noise		
0 dB	85.1	84.6
>0–55 dB	10.2	10.4
>55 dB	4.8	4.9
Road traffic noise, dB	55.8 (48.8–68.6)	56.3 (49.0–68.7)
Tumor side, %		
Right	38.4	–
Left	41.2	–
Missing	20.4	–
Tumor spread, %		
Intra + Extrameatal	69.3	–
Intrameatal	24.5	–
Missing	6.3	–
Tumor size, mm	11.0 (4.0–37.0)	–
Missing	5.1	–

[a]Values are given in percentages, except for age, road traffic noise and tumor size, for which we present medians (5–95%).
[b]Household income 1 year before index date after taxation and interest, adjusted for number of persons in the household and categorized by gender-specific distribution of disposable household income per person.
[c]Median equated municipal disposable income in year 2000 according Statistics Denmark.

The distribution of exposure and covariate variables between cases and controls are presented in Table 1. Cases were more often male, had a long education, a higher income and were more often married or cohabiting. They were somewhat more likely to live in more affluent municipalities and less likely to live in the most densely populated municipalities. They were less likely to be exposed to railway noise and had a lower median road traffic noise exposure compared to controls. Finally, information on tumor size and spread was available for the majority of cases, whereas information on tumor side was less often obtainable. Median tumor size at diagnosis was 11 mm, and most cases had both intra and extrameatal growth. Left-sided tumors were somewhat more common than right sided.

Table 2. Associations between exposure to residential road traffic noise and risk for vestibular schwannoma.

	Cases/Controls	Model 1[a] OR (95% CI)	Model 2[b] OR (95% CI)
Average L_{den} over the 1-year period before diagnosis			
Linear estimate, per 10 dB	1454/2550	0.89 (0.80–0.99)	0.92 (0.83–1.03)
Categorical estimate			
<55 dB	667/1089	1.00 (ref.)	1.00 (ref.)
55 ≤ 60 dB	365/614	0.96 (0.81–1.13)	0.98 (0.83–1.16)
60 ≤ 65 dB	238/513	0.77 (0.64–0.92)	0.80 (0.66–0.96)
≥65 dB	184/334	0.89 (0.72–1.09)	0.95 (0.76–1.17)
Average L_{den} over the 2-year period before diagnosis			
Linear estimate, per 10 dB	1454/2550	0.89 (0.80–0.98)	0.92 (0.82–1.03)
Categorical estimate			
<55 dB	666/1080	1.00 (ref.)	1.00 (ref.)
55 ≤ 60 dB	368/612	0.97 (0.82–1.14)	1.00 (0.84–1.18)
60 ≤ 65 dB	241/524	0.75 (0.63–0.90)	0.78 (0.65–0.94)
≥65 dB	179/334	0.85 (0.69–1.05)	0.91 (0.74–1.13)

[a]Crude.
[b]Adjusted for personal income, marital status, education, railway noise exposure, municipal population density, and municipal income.

Table 3. Associations between exposure to residential railway noise at diagnosis and risk of vestibular schwannoma.

	Cases/Controls	Model 1[a] OR (95% CI)	Model 2[b] OR (95% CI)
Linear estimate, per 10 dB	1454/2550	0.99 (0.96–1.03)	1.00 (0.96–1.03)
Categorical estimate			
Nonexposed	1237/2158	1.00 (ref.)	1.00 (ref.)
≤55 dB	148/266	0.94 (0.69–1.27)	0.97 (0.71–1.32)
>55 dB	69/126	0.96 (0.77–1.19)	0.96 (0.77–1.20)

[a]Crude.
[b]Adjusted for personal income, marital status, education, road traffic noise exposure, municipal population density and municipal income.

The correlation ($R_{Spearman}$) between road and railway traffic noise 1 year before diagnosis was 0.07 and the correlation between exposure to road traffic noise 1 and 2 years before diagnosis was 0.99.

We did not find an association between road traffic noise and VS: A two-year time-weighted mean exposure was associated with an adjusted OR of 0.92 (0.82–1.03) for developing VS, per 10 dB increment. There was no clear trend in categorical analyses. Results were similar for analyses of one-year time-weighted mean exposure (Table 2). Similarly, linear and categorical analyses of residential railway noise did not suggest an association (Table 3).

We found no interaction of the association between road traffic noise and VS with any of the investigated factors: Demographics, year of diagnosis, individual and municipal socioeconomic variables, as well as railway noise exposure (Table 4).

As supplementary analyses, we investigated the association between 5- and 10-year time-weighted mean exposure and VS among cases and their matched controls with 5 and 10 years or more of exposure history available, respectively. The results were similar to the associations found for one- and two-year exposure in the entire study population (Table s1). We did also not find an association between residential road traffic noise and VS when investigating sub-types of tumors by side, spread and size (Table s2).

Table 4. Modification of the association between residential road traffic noise (linear, per 10 dB increase) over the 2-year period before diagnosis and risk of vestibular schwannoma by age, sex, socioeconomic factors, diagnosis year, municipal income and population density and railway noise.

Covariates	Cases/controls	Model 2 OR (95% CI)	*p* interaction
Age[a]			
<Median (56.7 years)	718/1285	0.92 (0.79–1.07)	.97
≥Median (56.7 years)	736/1265	0.92 (0.79–1.07)	
Sex[a]			
Men	705/1191	0.91 (0.78–1.06)	.81
Women	749/1359	0.93 (0.80–1.08)	
Education[b]			
Basic	496/1016	0.95 (0.80–1.13)	.37
Vocational	595/1019	0.97 (0.82–1.15)	
Higher	363/515	0.80 (0.64–1.00)	
Personal income[c]			
1st tertile	383/781	0.90 (0.74–1.10)	.93
2nd tertile	475/850	0.95 (0.78–1.15)	
3rd tertile	596/919	0.91 (0.77–1.09)	
Year of diagnosis[a]			
<2000	652/1147	0.89 (0.75–1.05)	.59
≥2000	802/1403	0.94 (0.82–1.09)	
Municipal income[d]			
1st quartile	284/544	0.90 (0.72–1.14)	.61
2nd quartile	393/707	1.04 (0.84–1.28)	
3rd quartile	379/637	0.87 (0.69–1.08)	
4th quartile	398/662	0.87 (0.71–1.07)	
Municipal population density[e]			
1000+/km^2	212/421	0.79 (0.60–1.04)	.34
500–1000/km^2	221/380	0.81 (0.61–1.08)	
150–<500/km^2	248/390	1.08 (0.82–1.41)	
<150/km^2	773/1359	0.95 (0.82–1.11)	
Railway noise exposure[f]			
Yes	217/392	0.95 (0.71–1.27)	.82
No	1237/2158	0.92 (0.81–1.03)	

[a]Adjusted for personal income, marital status, education, railway noise exposure, municipal population density and municipal income.
[b]Adjusted for personal income, marital status, railway noise exposure, municipal population density and municipal income.
[c]Adjusted for marital status, education, railway noise exposure, municipal population density and municipal income.
[d]Adjusted for personal income, marital status, education, railway noise exposure and municipal population density.
[e]Adjusted for personal income, marital status, education, railway noise exposure and municipal income.
[f]Adjusted for personal income, marital status, education, municipal population density and municipal income.

Discussion

In this nationwide case–control study, we found that residential exposure to road traffic and railway noise was not associated with subsequent risk of VS, regardless of exposure window and subgroup of disease. We did not find any effect modification of road traffic noise by demographics, year of diagnosis, individual and municipal socioeconomic variables or railway noise exposure.

To the best of our knowledge, this is the first study investigating traffic noise and VS. However, the association between other noise sources and VS has previously been assessed in case–control studies. The majority of these found a direct association between self-reported noise sources (occupational or nonoccupational) and VS [4–7], whereas studies on calculated occupational noise exposure (using a job-exposure matrix) found no association [3,9]. Thus, the results of the present study are along the lines of the previous studies on calculated noise exposure and vestibular schwannoma; not supporting a harmful effect of noise in relation to this. The discrepancy between studies on self-reported and modeled noise exposure could be explained by the fact that recall bias, a common problem in case–control studies, may affect the studies relying on self-reported exposure history. One of the studies on self-reported noise exposure investigated the validity of the exposure information by comparing self-reported data to data derived from a job-exposure matrix for 94 VS cases and 191 controls; finding a moderate agreement, which was somewhat higher for cases than for controls [20]. On the other hand, using a job-exposure matrix leads to classification of each job in only one noise category, neglecting all individual variation, which could result in nondifferential error. Hence, the inconsistency between self-reported occupational exposure and occupational exposure modeled via a job-exposure matrix could also be due to the dilution of any modest effect by the job-exposure matrix.

Another explanation for discrepancy between the null result of the present study, and other studies finding an association; primarily for occupational exposure, could be that the traffic noise levels of the present study – although perhaps persistent – are comparably low, with 55 dB, 60 dB and 65 dB used in the present study for the categorical road traffic exposure categories. In occupational studies, for example in Germany [20], participants classified their work place as 'noisy' only at average exposure levels of 80 dB. Given how noise is measured between 60 dB and 80 dB, this is a factor of 100 in sound intensity (loudness) and a factor of 4 in the perceived loudness (sound intensity). Unfortunately, however, the exposure distribution in the present study, with a median of 56 dB and a 95% of 69 dB both 1 and 2 years before diagnosis, did not allow us to investigate exposure categories above 65 dB. If noise affects VS only above a higher threshold, we are thus not able to identify it in the present study.

It is generally reported that the incidence rates of VS have increased over the last decades, potentially as a result of improved accessibility to diagnostic tools [21]. The findings of a direct association between noise and VS in some studies have been suggested to be attributable to diagnostic bias: Persons exposed to loud noise may develop hearing problems more often than the general population, and therefore, more frequently undergo medical examinations of the ear, leading to a higher discovery rate of the tumor [4], which is generally relatively benign and slow growing and may therefore go unnoticed for many years [2,22]. The prevalence of undiagnosed VS in studies of magnetic resonance imaging series suggests a higher prevalence than that reported in epidemiological studies [22].

The suggestion of an inverse, albeit nonsignificant, association between road traffic noise and VS in the present study may be explained by a number of factors: First, since the study is registry based, we only had a minimal number of registry-based covariates available, and hence, residual confounding may affect our results. Little is known about the etiology of VS, but it has generally been found that a long education is associated with a higher disease incidence [23]. Despite finding no significant interaction with educational level, this was the strongest covariate in the adjusted models of road traffic noise and VS in the present study. Another potential explanation could be that undiagnosed cases were specifically disturbed by traffic noise and moved to more quiet areas before being diagnosed. However, when examining moving history among cases and controls in the 5-year period before index date, we do not find a difference in mean number of addresses ($p = .11$), making this an unlikely explanation. Finally, smoking has been found to confer a protective effect against VS [24]. Being registry based, the present study includes no data on smoking status or history, but as smoking is known to be more widespread among those with a lower education in Denmark [25], this could be a potential confounder, which we do not capture the effect of by adjusting for education only. If smokers are more exposed to road traffic noise, which could be a result of their generally lower socioeconomic position, entailing that they live in cheaper housing with more traffic noise exposure, then this could explain the finding of a nonsignificant protective effect of road traffic noise in the present study. In general, however, the study does not indicate an association between traffic noise and VS, and thus, the potential residual confounding is not of a magnitude that affects our conclusion of the study, rendering it a minor cause of concern.

We did not have information on occupational noise exposure in the present study. If noise does in fact affect development of VS, we would expect the total noise exposure across sources to be the relevant exposure, rather than traffic noise specifically. One could even speculate that there could be synergistic effects of being constantly exposed to noise through both occupational and residential traffic exposure. In order for noise from occupational sources to be a strong confounder of the association investigated in the present study, this would require both a strong association with traffic noise as well as with VS risk. We have no information about the correlation between traffic noise exposure and exposure to noise from nontraffic sources in this study. It could be speculated that occupational noise exposure is more common in more rural areas of Denmark, where most larger factories are located. In that case, this should be inversely correlated with traffic noise exposure, which is more widespread in urban areas. However, if this was a strong risk factor, it should potentially entail a geographic distribution with more VS cases in nonurban areas. This has not been found in a previous Danish study on the geographic distribution of VS [26]. Finally, the literature on noise as a risk factor for VS does not provide convincing evidence

for an association, which could mean that confounding by nontraffic sources should not strongly affect the findings of the present study.

The strengths of the present study include its size and the registry-based approach, taking the entire Danish population as a starting point, and using national registries of high quality with nearly complete coverage of all residents. This approach minimized the risk of selection bias. Furthermore, the exposure was based on registry information on address history, road lines and traffic, from national registries and municipalities. This is a primary study strength, as the modeled exposure for both cases and controls allowed us to circumvent recall bias; a common cause of concern otherwise associated with interview-based case–-control studies.

The present study did also harbor some limitations. First, we had information on exposure only two years preceding VS diagnosis for the entire study population. VS is a slow-growing tumor, which can have persisted unnoticed for years before causing symptoms, which result in the discovery of the tumor [2,22]. Hence, the relatively short time-window may not be appropriate to detect a potential effect of traffic noise on VS, if it operates through pathways of relevance for initial development of the disease. However, when examining the association with 5- and 10-year mean exposure among cases and their matched controls with 5 and 10 years of exposure history or more, produced a similar result as for one- and two-year exposure. Furthermore, the population was relatively settled, with 63% of participants with 10-year exposure history living in the same address or a maximum of two addresses over the 10-year period; meaning that 10-year exposure may be a good proxy for even longer exposure history for a large part of the participants. Second, despite the use of a well-validated noise exposure model relying on high-quality input data, exposure misclassification may persist in the present study due to lack of information on time spent at home, window opening habits and hearing impairment, which may all affect the true, individual noise exposure. As such misclassification should be unrelated to case/control status, we expect it to be nondifferential, which could potentially be the cause of the null-results of the present study. Third, as mentioned earlier, we lack information on other sources of noise exposure, for example, occupational and leisure-time noise, and this should be taken into account when interpreting the study findings.

In conclusion, the results of the present study do not suggest an association between residential traffic noise exposure and VS in a large, population-based case–control study, based on modeled noise exposure. The lack of an association is consistent across time periods investigated, subtypes of disease and participant subgroups defined by demographic factors, year of diagnosis, individual and municipal socioeconomic indicators, and railway noise exposure.

Disclosure statement

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