ORIGINAL ARTICLE

Exposure to loud noise, bilateral high-frequency hearing loss and coronary heart disease

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ABSTRACT

Objectives Bilateral high-frequency hearing loss is an indicator for chronic exposure to loud noise. This study aimed to examine the association between bilateral high-frequency hearing loss and the presence of coronary heart disease (CHD).

Methods This study included 5223 participants aged 20–69 years who participated in the audiometry examination of the National Health and Nutrition Examination Survey 1999–2004. Bilateral high-frequency hearing loss was defined as the average high-frequency (3, 4 and 6 kHz) hearing threshold ≥25 dB in both ears. CHD was defined as self-reported diagnoses by doctors or other health professionals.

Results Compared with those with normal high-frequency hearing, participants with bilateral high-frequency hearing loss were more likely to have CHD (OR 1.91; 95% CI 1.28 to 2.85) after adjustment for various covariates. This association was particularly strong for currently employed workers who were exposed to loud occupational noise (OR 4.23; 95% CI 1.32 to 13.55). For this subgroup, there was no significant association of CHD with unilateral high-frequency hearing loss, and unilateral or bilateral low-frequency hearing loss. Furthermore, there was no significant association of CHD with any types of hearing loss for participants who were not exposed to loud noise. Stratified analyses for participants exposed to loud noise showed that the observed association was particularly strong for those who were less than 50 years of age, less educated and current smokers.

Conclusions On the basis of an objective indicator for personal chronic exposure to loud noise, this study confirmed that exposure to loud occupational noise is associated with the presence of CHD.

What this paper adds

- The lack of information on personal exposure to loud noise is a major limitation of noise exposure assessment in epidemiological studies about noise exposure and health outcomes. Bilateral high-frequency hearing loss, an objective indicator for chronic exposure to loud noise, may be used to assess noise-related adverse health effects.
- Participants with bilateral high-frequency hearing loss were more likely to have coronary heart disease (CHD) compared with those with normal high-frequency hearing; the association was particularly strong for workers who were currently or previously exposed to loud noise in the workplace.
- Stratified analyses for participants exposed to loud noise showed that the association between bilateral high-frequency hearing loss and the presence of CHD was particularly strong for those who were less than 50 years of age, less educated and current smokers.
- On the basis of an objective indicator for personal chronic exposure to loud noise, this study confirmed the previous finding that chronic exposure to occupational noise is associated with the presence of CHD.

INTRODUCTION

Excessive noise is a potent environmental stressor that may cause annoyance, sleep disturbance and psychological stress.[1 2] Importantly, some evidence has suggested that long-term exposure to loud noise is associated with cardiovascular disease (CVD) outcomes in community[3 4] and occupational settings.[5 6] We previously reported that workers who were chronically exposed to loud noise in the workplace had a twofold to threefold increase in the presence of coronary heart disease (CHD) compared with those not exposed to loud occupational noise.[7] However, previous epidemiological findings are not consistent, as some studies did not find adverse cardiovascular effects associated with noise exposure.[8–10]

In large epidemiological studies, noise exposure is typically assessed using noise prediction models for community noise and self-reports or historical records for occupational noise.[7 11–13] These methods are able to estimate community noise exposure in residential areas and occupational noise exposure in the workplace; however, these exposure assessments are not able to accurately reflect actual personal noise exposure in the home and workplace. For example, the use of hearing protective devices such as earplugs and muffs may substantially reduce actual personal exposure to loud noise in the workplace. The lack of personal noise exposure information is the major limitation of previous studies, which could potentially result in underestimations of the true adverse health effects associated with noise exposure. This limitation may at least partly explain the inconsistent findings of previous epidemiological studies.

Bilateral high-frequency hearing loss is an indicator for long-term exposure to loud noise, especially in an occupational setting.[14 15] The severity of

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bilateral high-frequency hearing loss is associated with the intensity and duration of personal noise exposure.[16 17] Therefore, bilateral high-frequency hearing loss can serve as an objective indicator for personal chronic exposure to loud noise in examining noise-related adverse health effects. The current study aimed to investigate the association between bilateral high-frequency hearing loss and the presence of CHD using a large nationally representative sample from the US National Health and Nutrition Examination Survey (NHANES) 1999–2004. In addition, to explore potential biological mechanisms underlying the observed association, this study also examined the associations of bilateral high-frequency hearing loss with cardiovascular biomarkers including blood lipids and systemic inflammatory mediators.

METHODS

NHANES is a series of cross-sectional surveys to assess the health status of adults and children in the USA. During each survey, a nationally representative sample of the civilian non-institutionalised population is selected using a complex, stratified, multistage probability sampling procedure. The survey includes a household interview and a medical examination at a mobile examination centre. In the household interview, various health-related questionnaires, including those about noise exposure in the workplace and leisure time, behavioural risk factors of CVD, and medical conditions, are administered by a trained interviewer. In the medical examination, various anthropometric and physiological measurements are performed by trained medical personnel. Detailed information on the survey and sampling methodology is provided elsewhere.[18 19]

There are three consecutive NHANES surveys (1999–2000, 2001–2002 and 2003–2004) that encompass data on both occupational noise exposure and audiometry examination. In accordance with the National Center for Health Statistics (NCHS) Analytic Guidelines,[19] we aggregated these three surveys to create a combined NHANES 1999–2004 data set. For participants 20–69 years of age, the overall response rate was 77% for the household interview and 73% for the medical examination.[20] The NHANES 1999–2004 was reviewed and approved by the NCHS Institutional Review Board; informed consent was obtained from all participants.

Study sample

There were 12 056 participants 20–69 years of age in the NHANES 1999–2004, half of which were selected for audiometry examination. Persons who had severe ear pain and those who were unable to remove their hearing aids were excluded, leaving 5742 participants eligible for audiometric testing. After excluding 324 persons who did not perform audiometric testing, 111 persons with missing audiometric data and 3 persons with unacceptable results (the hearing threshold difference between the 1 kHz test and retest was greater than 10 dB), there were 5304 participants with complete audiometric testing data. We further excluded 81 persons with missing data on education attainment, cigarette smoking status or body mass index (BMI), leaving 5223 participants for the current analysis.

Audiometry examination and hearing loss

Audiometric testing was performed in a sound-isolating room at the mobile examination centre by health technicians using an Interacoustics Model AD226 audiometer with standard TDH-39 headphones and Etymotic EarTone 3A insert earphones.[21] The health technicians were professionally trained and their performance was regularly monitored by certified audiologists from the National Institute for Occupational Safety and Health. Air conduction hearing thresholds were measured for each ear using a pure tone at seven frequencies (0.5, 1, 2, 3, 4, 6 and 8 kHz) over an intensity range of −10 to 120 dB. The 1 kHz frequency was tested twice in each ear to assess the reliability of the measurements. If the threshold difference between the two tests was greater than 10 dB, the measurement was considered unreliable and was excluded from the analyses (n=3). For those with qualified measurements, the first 1 kHz test was used in the analyses. If the threshold difference between two ears was ≥25 dB at both 0.5 and 1 kHz or ≥40 dB at any higher frequencies, a cross-over retesting protocol was performed to determine the true threshold of the test ear. Retesting was performed using insert earphones, which were smaller and had less direct contact with the head. If a person did not respond to any levels of a signal tone at a particular frequency, a non-response code was entered for the hearing threshold at that frequency. The non-response code was treated as missing data in this study (n=5). Detailed information on audiometric testing procedures is available elsewhere.[21]

In a noisy environment, both ears are exposed to loud noise simultaneously, and noise-induced hearing loss most commonly develops at high frequencies.[14–17] Therefore, we used bilateral high-frequency hearing loss as an indicator for personal chronic exposure to loud noise in assessing the association with the presence of CHD. Meanwhile, since other types of hearing loss, including unilateral high-frequency hearing loss as well as unilateral and bilateral low-frequency hearing loss, are typically not related to chronic noise exposure, for the purpose of comparison, we also examined the associations of CHD with each of these types of hearing loss.

In this study, we specified that high frequencies comprise 3, 4 and 6 kHz.[14] For each ear, the average high-frequency hearing threshold was calculated using the arithmetic mean of hearing thresholds at 3, 4 and 6 kHz.

Bilateral high-frequency hearing loss: average high-frequency hearing threshold ≥25 dB in both the right and left ears.

Unilateral high-frequency hearing loss: average high-frequency hearing threshold ≥25 dB in only one ear, left or right ear.

Similarly, low frequencies comprise 0.5, 1 and 2 kHz. For each ear, the average low-frequency hearing threshold was calculated using the arithmetic mean of the hearing thresholds at 0.5, 1 and 2 kHz. Bilateral and unilateral low-frequency hearing loss was defined using the same method for high-frequency hearing loss.

Noise exposure assessment

Noise exposure in the workplace: On the basis of current and previous noise exposure status in the workplace for currently employed workers at the time of the interview, participants were divided into unexposed or exposed groups by answering 'no' or 'yes' to the question: 'Thinking of all the jobs you have ever had, have you ever been exposed to loud noise at work for at least three months? By loud noise I mean noise was so loud that you had to speak in a raised voice to be heard'.

Noise exposure in leisure time: Leisure-time noise comprised firearm noise and other recreational loud noise. Firearm noise exposure was defined as exposure outside of work to firearm noise for 'an average of at least once a month for a year'. Other recreational noise exposure was defined as exposure outside of work to loud noise such as loud music or power tools for 'an average of at least once a month for 1 year'. In NHANES 1999–2004, loud noise means that the 'noise was so loud that you had to speak in a raised voice to be heard'. Participants

who reported exposure to any type of loud noise in their leisure time were regarded as exposed, but otherwise regarded as unexposed, to loud noise in leisure time.

Coronary heart disease

CHD was defined as a self-report of diagnosis by a doctor or other health professional. For example, self-reported CHD was defined by answering 'yes' to the question 'Has a doctor or other health professional ever told you that you had coronary heart disease?'. CHD in this study comprised self-reported diagnosis of CHD, angina pectoris and myocardial infarction.

Biochemistry measurement

During the medical examination at the mobile examination centre, a blood specimen was drawn from the participant's antecubital vein by a trained phlebotomist. Biochemistry measurements comprised homocysteine; systemic inflammatory mediators including leucocytes, platelets, fibrinogen and C reactive protein; and blood lipids including total cholesterol, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein cholesterol and triglyceride. A detailed description of the laboratory measurement procedures is available elsewhere.[22]

Potential confounding factors

In addition to age, sex and BMI, we took into account the following cardiovascular risk factors. On the basis of the questionnaire design and the frequency distribution of each variable, these potential confounding factors were categorised as follows:

Race/ethnicity: non-Hispanic white (Caucasian), non-Hispanic black (African-American), Mexican American and other.

Educational attainment: less than high school, high school (including general equivalency diploma) and more than high school.

Leisure time physical activity: On the basis of self-reported frequency and duration of participation in various moderate and vigorous physical activities during the past 30 days, the weekly total number of minutes spent in moderate (weekly frequency multiplied by the average duration of activity) and vigorous (weekly frequency multiplied by the average duration of activity multiplied by 2) physical activity was calculated for each participant. Ideal, intermediate and poor levels of physical activity were defined as ≥150, 1–149 and 0 min of physical activity each week, respectively.[23 24]

Cigarette smoking status: current smokers (had smoked ≥100 cigarettes in their lifetime and still smoked at the time of the interview), former smokers (had smoked ≥100 cigarettes in the lifetime but did not smoke at the time of the interview) and never-smokers (had not smoked ≥100 cigarettes in their lifetime).

Diabetes: the presence of diabetes was defined as a self-reported diagnosis by a doctor or other health professional.

Statistical analysis

NHANES uses a complex sampling design to select participants to represent the US general population. This study was based on the participants who participated in the audiometry examination of NHANES 1999–2004. The sample weights for the combined 6-year data were constructed: for the first two survey cycles (1999–2000 and 2001–2002), the sample weights were constructed by multiplying the 4-year audiometry subsample weights by (2/3); for the third survey cycle (2003–2004), the sample weights were constructed by multiplying the 2-year audiometry subsample weights by (1/3).[19] The sample weights (or appropriate weights for some subgroup analyses of biochemistry profiles) were incorporated into all analyses to account for the complex sampling design, differential probabilities of selection, non-coverage and non-response of the survey.[19] The SAS Survey Procedure (SAS 9.3, SAS Institute Inc, Cary, North Carolina, USA) was used to perform the statistical analysis.

The characteristics of the participants were compared between each hearing loss group and normal-hearing group using the Rao-Scott χ^2 test (design-adjusted version of the normal χ^2 test) for categorical variables and the two sample t test for continuous variables. For those continuous variables that are not normally distributed (eg, C reactive protein and triglyceride), log-transformed data were used in the relevant analyses. To assess the reliability of each estimated proportion or mean, we calculated the relative SE (RSE) by dividing the SE of the estimate by the estimate itself, multiplying that result by 100. An estimate with RSE above 30% was considered statistically unreliable.[19]

The association between bilateral high-frequency hearing loss (predictor variable) and the presence of CHD (dependent variable) was examined using multiple logistic regression after adjustment for various cardiovascular risk factors, including age (continuous), sex, BMI (quartiles), race/ethnicity (Caucasian, African-American, Mexican American or other), education attainment (less than high school, high school or equivalent, or more than high school), cigarette smoking (current smoker, former smoker or never-smoker), leisure time physical activity (poor, intermediate, ideal) and the presence of diabetes (yes, no). Moreover, we performed stratified analyses by self-reported noise exposure status in the workplace or leisure time to examine how noise exposure affects the associations between bilateral high-frequency hearing loss and the presence of CHD. We further repeated the above analyses using unilateral high-frequency hearing loss, unilateral low-frequency hearing loss and bilateral low-frequency hearing loss as predictor variable, respectively, to examine the relationships of different types of hearing loss with the presence of CHD.

RESULTS

A total of 5223 participants were included in this study, of whom 34% (unweighted prevalence 36%) had either unilateral or bilateral high-frequency hearing loss. Compared with those with normal high-frequency hearing, participants with high-frequency hearing loss were older, less educated and physically inactive; they were more likely to be male, Caucasian, current or former smokers, and have diabetes, angina pectoris, myocardial infarction or CHD. These features are particularly evident for participants with bilateral high-frequency hearing loss (table 1).

There were 255 CHD cases in the study sample, and the overall prevalence of CHD was 4% (unweighted prevalence 5%); the prevalence was 2% (unweighted prevalence 2%) for those with normal high-frequency hearing, 5% (unweighted prevalence 6%) for those with unilateral high-frequency hearing loss and 12% (unweighted prevalence 12%) for those with bilateral high-frequency hearing loss. Compared with those with normal high-frequency hearing, participants with bilateral high-frequency hearing loss were 1.91 times (95% CI 1.28 to 2.85) more likely to have CHD, after adjustment for age, sex, race/ethnicity, BMI, education attainment, cigarette smoking, leisure time physical activity and the presence of diabetes (table 2). This observed association was existent among participants who were exposed to loud noise in the workplace or leisure time, but was particularly strong for currently employed workers who were currently or previously exposed to loud noise in the workplace (n=1137; OR 4.23; 95% CI 1.32 to 13.55; table 2). However, there were no significant associations for participants

Table 1 Characteristics of participants by status of high-frequency hearing loss*

		Types of high-frequency hearing loss		
	Normal (n=3366)	Unilateral† (n=660)	Bilateral† (n=1197)	Either† (n=1857)
Age, mean (SE), year	37.4 (0.3)	46.1 (0.4)	53.6 (0.4)	50.7 (0.3)
Sex, N (%)				
Men	1259 (39.2)	368 (60.6)	837 (71.5)	1205 (67.3)
Women	2107 (60.8)	292 (39.4)	360 (28.5)	652 (32.7)
Ethnicity, N (%)				
Caucasian	1510 (66.2)	316 (72.4)	654 (79.7)	970 (76.9)
African-American	762 (13.4)	122 (8.8)	162 (5.9)	284 (7.0)
Mexican-American	778 (8.8)	149 (6.9)	294 (5.6)	443 (6.1)
Other	316 (11.6)	73 (11.9)	87 (8.9)	160 (10.0)
Education, N (%)				
<High school	828 (15.8)	213 (19.0)‡	468 (25.2)	681 (22.9)
High school	761 (23.7)	144 (24.8)‡	292 (29.3)	436 (27.5)
>High school	1777 (60.5)	303 (56.2)‡	437 (45.5)	740 (49.6)
Physical activity, N (%)				
Poor	1280 (31.8)	306 (40.0)	587 (42.1)	893 (41.3)
Intermediate	778 (24.2)	138 (22.8)	237 (22.3)	375 (22.5)
Ideal	1308 (44.0)	216 (37.2)	373 (35.6)	589 (36.2)
Cigarette smoking, N (%)				
Current smoker	814 (26.3)	166 (27.3)	325 (28.1)	491 (27.8)
Former smoker	625 (19.3)	183 (28.7)	420 (34.6)	603 (32.3)
Never–smoker	1927 (54.4)	311 (44.0)	452 (37.3)	763 (39.9)
BMI, mean (SE), kg/m^2	27.7 (0.2)	29.0 (0.3)	28.9 (0.2)	28.9 (0.2)
Having diabetes, N (%)				
Yes	141 (3.3)	61 (6.1)	198 (14.2)	259 (11.1)
No	3225 (96.7)	599 (93.9)	999 (85.8)	1598 (88.9)
Having angina pectoris, N (%)				
Yes	35 (0.8)	17 (2.4)	83 (7.0)	100 (5.3)
No	3320 (99.2)	639 (97.6)	1106 (93.0)	1745 (94.7)
Having MI, N (%)				
Yes	33 (1.0)	21 (2.6)	81 (6.9)	102 (5.3)
No	3332 (99.0)	637 (97.4)	1111 (93.1)	1748 (94.7)
Having CHD, N (%)				
Yes	68 (1.7)	42 (5.2)	145 (11.9)	187 (9.4)
No	3298 (98.3)	618 (94.8)	1052 (88.1)	1670 (90.6)

*Unweighted sample sizes and weighted percentages. All relative SEs for estimated proportions or means <30%. p<0.01 for all comparisons between each hearing loss group and the normal high-frequency hearing group, unless otherwise specified.
†Average high-frequency hearing threshold ≥25 dB for a pure tone at 3, 4 and 6 kHz in one ear (unilateral), both ears (bilateral), and either one ear or both ears (either).
‡p Value >0.05 compared with the normal high-frequency hearing group.
BMI, body mass index; CHD, coronary heart disease; MI, myocardial infarction.

who were not exposed to loud noise in the workplace or leisure time (table 2). Furthermore, for currently employed workers who were currently or previously exposed to loud noise in the workplace, there were no significant associations of CHD with unilateral high-frequency hearing loss (figure 1 and table 2) as well as unilateral and bilateral low-frequency hearing loss (figure 1 and eTable 1).

CHD includes myocardial infarction and angina pectoris. We further examined the associations of bilateral high-frequency hearing loss with myocardial infarction and angina pectoris using the same statistical methods. These results are consistent with the findings of CHD analysis. For those who were exposed to loud noise in the workplace or leisure time, the ORs were larger compared with the results of CHD analysis; however, the corresponding 95% CIs are wider because of the small numbers of cases (table 2).

To examine exposure–response relations between the average high-frequency hearing threshold and the presence of CHD, we divided participants into quartiles according to the average high-frequency hearing thresholds in the left ear, right ear and mean of the thresholds in the left and right ears, respectively. eTable 2 shows a strong exposure–response trend between the average high-frequency hearing threshold and the prevalence of CHD. The trend was particularly evident for those who were exposed to loud noise in the workplace or leisure time; for example, when the average hearing threshold was lower (quartile 1 or 2), there were no or very few CHD cases, but when the average hearing threshold increased (quartile 3 or 4), the prevalence of CHD was substantially increased, especially for participants with the highest hearing threshold (quartile 4). Since there were no CHD cases in quartile 1 for most subgroups in eTable 2, ORs could not be calculated for these subgroups; therefore, only the number of CHD cases and the prevalence were presented in the table.

Owing to the sample size requirements, the stratified analyses by cardiovascular risk factors were conducted including participants who were exposed to loud noise in the workplace or leisure time (figure 2). The association between bilateral

Table 2 Adjusted ORs (95% CIs) of CHD for participants with high-frequency hearing loss compared with those with normal high-frequency hearing*

Noise exposure	Number of participants (number of cases)	Types of high-frequency hearing loss		
		Unilateral†	Bilateral†	Either†
CHD				
All participants				
Yes/no	5223 (255)	1.47 (0.86 to 2.49)	1.91 (1.28 to 2.85)	1.73 (1.18 to 2.54)
Loud noise in the workplace or leisure time				
Yes	1968 (98)	2.03 (0.91 to 4.52)	2.95 (1.44 to 6.07)	2.57 (1.33 to 4.96)
No	1856 (39)	1.48 (0.56 to 3.95)	2.00 (0.77 to 5.21)	1.74 (0.81 to 3.73)
Loud noise at leisure time				
Yes	1337 (79)	2.09 (0.81 to 5.38)	2.35 (1.03 to 5.39)	2.25 (1.06 to 4.77)
No	3879 (175)	1.07 (0.61 to 1.88)	1.52 (0.99 to 2.30)	1.33 (0.90 to 1.97)
Loud noise in the workplace‡				
Yes	1137 (44)	1.78 (0.37 to 8.52)	4.23 (1.32 to 13.55)	3.25 (1.04 to 10.19)
No	2292 (46)	2.31 (0.99 to 5.36)	1.29 (0.54 to 3.10)	1.75 (0.87 to 3.50)
Myocardial infarction				
All participants				
Yes/no	5215 (135)	1.15 (0.53 to 2.46)	1.60 (0.94 to 2.73)	1.42 (0.83 to 2.42)
Loud noise in the workplace or leisure time				
Yes	1966 (51)	3.59 (1.02 to 12.66)	4.55 (1.44 to 14.36)	4.12 (1.37 to 12.37)
No	1856 (22)	0.58 (0.12 to 2.73)	1.18 (0.34 to 4.14)	0.92 (0.32 to 2.62)
Loud noise at leisure time				
Yes	1335 (36)	4.71 (1.06 to 20.88)	4.00 (0.85 to 18.71)	4.31 (1.01 to 18.40)
No	3873 (99)	0.66 (0.28 to 1.57)	1.16 (0.61 to 2.21)	0.98 (0.54 to 1.76)
Loud noise in the workplace‡				
Yes	1136 (26)	3.11 (0.58 to 16.77)	4.40 (1.07 to 18.20)	3.89 (1.00 to 15.19)
No	2292 (25)	0.95 (0.24 to 3.79)	0.99 (0.34 to 2.90)	0.98 (0.38 to 2.52)
Angina pectoris				
All participants				
Yes/no	5200 (135)	1.38 (0.67 to 2.87)	2.40 (1.24 to 4.66)	1.96 (1.09 to 3.53)
Loud noise in the workplace or leisure time				
Yes	1962 (55)	2.04 (0.60 to 6.99)	4.62 (1.50 to 14.26)	3.40 (1.19 to 9.66)
No	1851 (18)	1.50 (0.38 to 5.88)	0.88 (0.16 to 4.94)	1.17 (0.31 to 4.37)
Loud noise at leisure time				
Yes	1331 (45)	2.13 (0.54 to 8.31)	3.09 (0.96 to 9.99)	2.66 (0.86 to 8.21)
No	3863 (90)	0.84 (0.34 to 2.08)	1.83 (0.87 to 3.85)	1.42 (0.73 to 2.76)
Loud noise in the workplace‡				
Yes	1135 (20)	2.42 (0.22 to 26.35)	10.62 (1.76 to 64.12)	6.46 (1.13 to 37.11)
No	2286 (21)	1.80 (0.56 to 5.80)	0.73 (0.17 to 3.17)	1.21 (0.39 to 3.78)

*Unweighted sample sizes and weighted ORs (95% CIs); adjusted for age, sex, race/ethnicity, BMI, education attainment, cigarette smoking, leisure time physical activity and the presence of diabetes.
†Average high-frequency hearing threshold ≥25 dB for a pure tone at 3, 4 and 6 kHz in one ear (unilateral), both ears (bilateral), and either one ear or both ears (either).
‡For currently employed workers who were currently or previously exposed to loud noise in the workplace.
BMI, body mass index; CHD, coronary heart disease.

high-frequency hearing loss and the presence of CHD was particularly strong for participants who were less than 50 years of age, less educated, and current smokers.

Overall, compared with those with normal high-frequency hearing, participants with bilateral high-frequency hearing loss had worse biochemistry profiles, including an increased number of leucocytes, increased concentrations of C reactive protein and decreased concentrations of HDL cholesterol after adjustment for age, sex, race/ethnicity, BMI, education attainment, cigarette smoking, leisure time physical activity and the presence of diabetes (table 3).

DISCUSSION

On the basis of a large nationally representative sample with detailed information on cardiovascular risk factors, we found that the prevalence of CHD was significantly higher among participants with bilateral high-frequency hearing loss compared with those with normal high-frequency hearing; this association was particularly strong for participants who were exposed to loud noise in the workplace (OR 4.23; 95% CI 1.32 to 13.55). For this subgroup, there was no significant association of CHD with unilateral high-frequency hearing loss, and unilateral or bilateral low-frequency hearing loss. Furthermore, there was no significant association of CHD with any type of hearing loss for participants who were not exposed to loud noise in the workplace or leisure time. This study confirmed the previous finding that chronic exposure to loud occupational noise, indicated by bilateral high-frequency hearing loss, is associated with increased prevalence of CHD.[7]

In our study, age was potentially an important confounding factor because age is a risk factor for both CHD (atherosclerosis) and bilateral high-frequency hearing loss (age-related

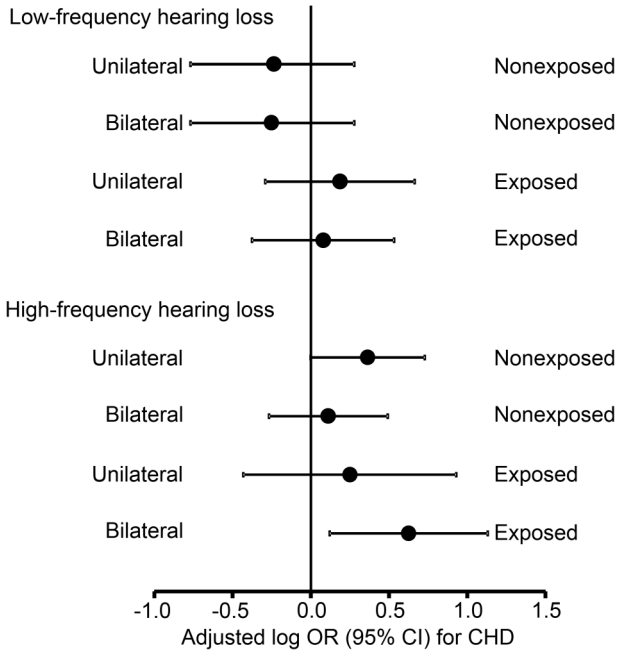

Figure 1 Adjusted log ORs of coronary heart disease (CHD) for participants with different types of hearing loss, stratified by exposure status to occupational noise. The analysis was restricted to currently employed workers (n=3429), and was adjusted for age, sex, race/ethnicity, body mass index, education attainment, cigarette smoking, leisure time physical activity and the presence of diabetes. The normal high-frequency hearing group and normal low-frequency hearing group were used as the reference group, respectively, for the high-frequency hearing loss group and low-frequency hearing loss group.

hearing loss). In our data analysis, we adjusted for age as a continuous covariate, and the influence of age on the observed association was thus diminished. Furthermore, the stratified analyses showed that the observed association was only present among participants aged less than 50 years, but not present for those aged ≥50 years. This observation is in contrast to age-related hearing loss, which mainly affects people aged over 50 years.[25] Therefore, age was not likely to confound the observed association between bilateral high-frequency hearing loss and the presence of CHD in this study.

In addition to bilateral high-frequency hearing loss, which typically originated from chronic exposure to occupational noise,[14–17] we also examined the associations of CHD with other types of hearing loss, including unilateral high-frequency hearing loss, as well as unilateral and bilateral low-frequency hearing loss. Since these types of hearing loss were typically not related to chronic exposure to occupational noise,[14 15 26] the null associations of CHD with these types of hearing loss for workers exposed to loud occupational noise provided additional support for the association between exposure to occupational noise and CHD outcomes. Furthermore, it was notable that there were no significant associations of CHD with any types of hearing loss for participants who were not exposed to loud noise in the workplace or leisure time, suggesting that it was chronic exposure to occupational noise that was responsible for the observed association between bilateral high-frequency hearing loss and CHD outcomes.

Previous studies conducted in occupational settings have found significant associations of bilateral high-frequency hearing loss with blood pressure and the presence of hypertension. In a previous study of 790 production-line workers from an aircraft-manufacturing company,[27] Chang *et al*[27] found that noise levels

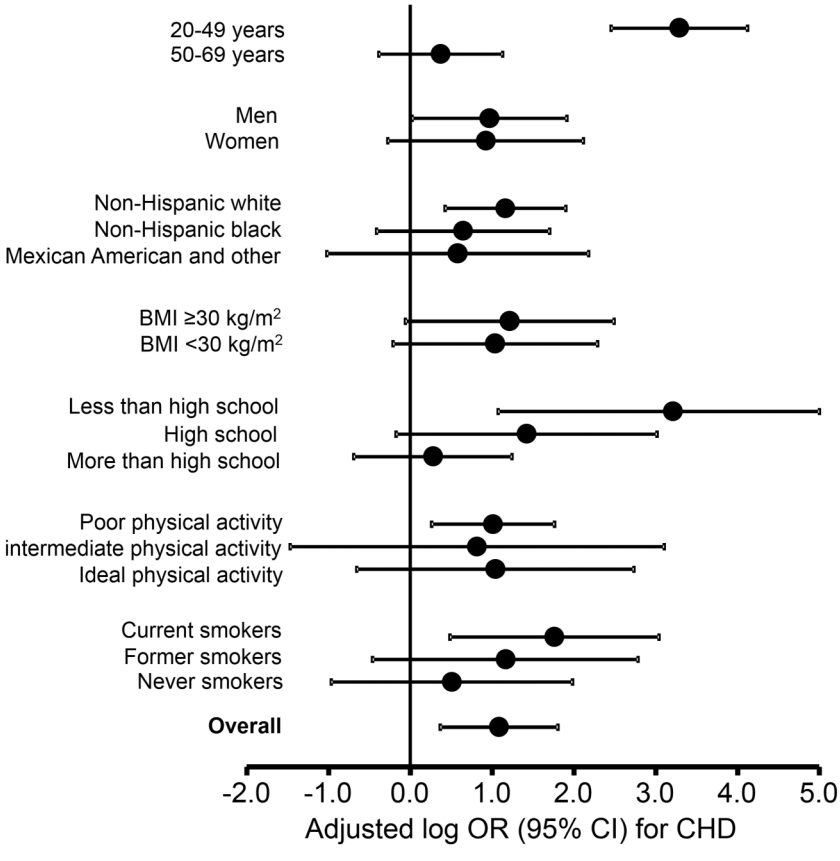

Figure 2 Adjusted log ORs of CHD for participants with bilateral high-frequency hearing loss compared with those with normal high-frequency hearing, stratified by selected cardiovascular risk factors. The analysis was restricted to participants who were exposed to loud noise in the workplace or their leisure time (n=1968), and was adjusted for all covariates when appropriate. BMI, body mass index; CHD, coronary heart disease.

Table 3 Biochemistry profiles for participants stratified by status of high-frequency hearing loss*

		Types of high-frequency hearing loss		
	Normal (n=3229)	Unilateral† (n=646)	Bilateral† (n=1165)	Either† (n=1809)
Leucocytes, mean (SE), $\times 10^9$/L	7.26 (0.05)	7.17 (0.09)	7.38 (0.08)‡	7.30 (0.07)‡
Platelet, mean (SE), $\times 10^9$/L	272.8 (1.6)	271.2 (2.7)	260.6 (2.8)	264.7 (2.2)
Fibrinogen, mean (SE), g/L§	3.56 (0.04)	3.52 (0.06)	3.65 (0.05)	3.61 (0.05)
CRP, median (IQR), mg/L	0.18 (0.06–0.42)	0.22‡ (0.09–0.47)	0.23‡ (0.10–0.48)	0.22‡ (0.09–0.47)
Homocysteine, mean (SE), μmol/L	7.83 (0.08)	8.65 (0.13)	9.47 (0.16)	9.16 (0.12)
Total cholesterol, mean (SE), mmol/L	5.11 (0.02)	5.41 (0.06)	5.48 (0.05)	5.45 (0.04)
HDL-cholesterol, mean (SE), mmol/L	1.38 (0.01)	1.30 (0.02)	1.28 (0.02)‡	1.29 (0.01)‡
LDL-cholesterol, mean (SE), mmol/L¶	3.06 (0.03)	3.24 (0.05)	3.30 (0.05)	3.27 (0.05)
Triglyceride, median (IQR), mmol/L¶	1.17 (0.84–1.77)	1.43 (1.00–2.07)	1.58 (1.07–2.36)	1.54 (1.05–2.20)

*Unweighted sample sizes; the sample size for each variable is slightly different because of missing data, unless otherwise specified. All relative SEs for estimated means <30%.
†Average high-frequency hearing threshold ≥25 dB for a pure tone at 3, 4 and 6 kHz in one ear (unilateral), both ears (bilateral), and either one ear or both ears (either).
‡p Value <0.05 compared with the normal high-frequency hearing group after adjustment for age, sex, race/ethnicity, BMI, education attainment, cigarette smoking, leisure time physical activity and the presence of diabetes.
§Only available during the NHANES 1999–2002 for participants aged 40 years and older, 4-year audiometry weights were incorporated into the analysis. The number of participants in the normal, unilateral, bilateral and either group was 838, 321, 694 and 1015, respectively.
¶This is a morning fasting sample. The number of participants in each group (normal/unilateral/bilateral/either): LDL-cholesterol, 1444/293/515/808; triglyceride, 1551/314/575/889.
BMI, body mass index; CRP, C reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; NHANES, National Health and Nutrition Examination Survey.

in the workplace were strongly correlated with individual high-frequency (4 and 6 kHz) hearing thresholds, and workers with bilateral high-frequency hearing loss were more likely to have hypertension. In a similar study with 269 workers from an automobile assembly plant,[28] Tarter and Robin found that high-frequency (4 kHz) hearing threshold and length of working (≥5 years) in a noisy workplace (>85 dBA) were both significantly associated with blood pressure levels and the presence of hypertension among African-American workers, but not among Caucasian workers. On the basis of a study of 245 retired metal assembly workers,[29] Talbott *et al* found that severe high-frequency (3, 4 and 6 kHz) hearing loss (≥65 dBA) was associated with the presence of hypertension in older workers (ages 64–68), but not in younger workers (ages 56–63); this observation is consistent with the findings of another study by Talbott *et al*.[30] Although the previous findings are not entirely consistent, these studies suggest that bilateral high-frequency hearing loss, in conjunction with noise exposure history, may be a useful marker to reflect actual personal exposure to occupational noise in assessing noise-related adverse health effects.

Our study has some limitations to be considered. First, since this is a cross-sectional study, the temporal relationships between noise exposure, bilateral high-frequency hearing loss and CHD outcomes are not clear. Also, owing to the cross-sectional nature of NHANES, persons who died from noise-related CHD could not be included in the survey, leading to potential underestimations of true adverse CHD effects associated with noise exposure. Future prospective cohort studies of healthy participants without hearing loss and heart disease are needed to confirm our findings. Furthermore, CHD was defined as self-reported diagnoses by doctors or other health professionals. Owing to incorrect recall or lack of knowledge on disease classification, disease misclassification might be present. However, given the severity of CHD, disease misclassification was less likely to be common and would not substantially affect the study outcomes. Finally, as discussed previously,[7] our stratified analysis was based on self-reported exposure to loud noise in the workplace or leisure time. Owing to differences in personal perceptions, misclassification of noise exposure might be present in the stratified analysis by noise exposure status.

CONCLUSIONS

This study found that bilateral high-frequency hearing loss, an objective marker of chronic exposure to loud noise, was associated with the presence of CHD; the association was particularly strong for workers who were currently or previously exposed to loud noise in the workplace; and there was no significant association of CHD with any types of hearing loss for participants who were not exposed to loud noise. These observations confirmed the previous finding that chronic exposure to occupational noise is associated with the presence of CHD. Given the cross-sectional nature of this study, future prospective cohort studies of healthy participants without hearing loss and heart disease are needed to confirm our findings.

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Exposure to Loud Noise, Bilateral High-Frequency Hearing Loss, and Coronary Heart Disease

Occupational and Environmental Medicine

Online Supplementary Materials

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eTable 1. Adjusted ORs of CHD for Participants with Low-Frequency Hearing Loss Compared with Those with Normal Low-Frequency Hearing[a]

	Noise Exposure Status	No. of Participants (No. of Cases)	Types of Low-Frequency Hearing Loss		
			Unilateral[b]	Bilateral[b]	Either[b]
All participants	Yes/No	5223 (255)	1.23 (0.74-2.06)	1.14 (0.75-1.73)	1.19 (0.80-1.77)
Loud noise in the workplace or leisure time[c]	Yes	1968 (98)	1.97 (0.97-4.04)	0.90 (0.41-1.96)	1.45 (0.79-2.65)
	No	1856 (39)	0.15 (0.03-0.83)	0.72 (0.18-2.90)	0.38 (0.12-1.24)
Loud noise in leisure time[c]	Yes	1337 (79)	2.90 (1.19-7.03)	0.81 (0.36-1.86)	1.75 (0.84-3.67)
	No	3879 (175)	0.67 (0.36-1.24)	1.26 (0.77-2.05)	0.95 (0.61-1.47)
Loud noise in the workplace[cd]	Yes	1137 (44)	1.54 (0.51-4.62)	1.20 (0.42-3.42)	1.41 (0.57-3.51)
	No	2292 (46)	0.58 (0.17-1.90)	0.56 (0.17-1.90)	0.57 (0.24-1.34)

Abbreviations: CHD, coronary heart disease; OR, odds ratio.

[a]Unweighted sample sizes and weighted odds ratios (95% CIs); adjusted for age, sex, race/ethnicity, body mass index, education attainment, cigarette smoking, leisure time physical activity, and the presence of diabetes.

[b]Average low-frequency hearing threshold ≥ 25 dB for a pure-tone at 0.5, 1, and 2 kHz in one ear (unilateral), both ears (bilateral), and either one ear or both ears (either).

[c]Loud noise means that noise was so loud that a person had to raise voice to be heard.

[d]For currently employed workers who were currently or previously exposed to loud noise in the workplace.

eTable 2. Number of Cases (Prevalence %) for CHD, Stratified by Quartiles of Average High-Frequency Hearing Threshold[a]

	Ear	Quartiles of Average High-Frequency Hearing Threshold				Overall (%)
		1 (best)	2	3	4 (worst)	
All participants[b] (n = 5223)	Left	10 (0.6)[e]	28 (1.9)	68 (4.5)	149 (11.3)	255 (4.3)
	Right	8 (0.7)[e]	30 (1.8)	70 (4.5)	147 (10.5)	255 (4.3)
	Mean	11 (0.8)[e]	23 (1.3)	74 (4.9)	147 (11.3)	255 (4.3)
Exposed to loud noise in the workplace or leisure time (n = 1968)[c]	Left	0 (NA)[f]	6 (0.7)[e]	24 (4.5)	68 (12.4)	98 (4.7)
	Right	0 (NA)[f]	6 (1.2)[e]	30 (4.9)	62 (11.4)	98 (4.7)
	Mean	0 (NA)[f]	4 (0.5)[e]	26 (4.4)	68 (12.7)	98 (4.7)
Exposed to loud noise at leisure time (n = 1337)[c]	Left	0 (NA)[f]	4 (0.6)[e]	20 (6.4)	55 (14.2)	79 (5.5)
	Right	0 (NA)[f]	5 (1.7)[e]	24 (5.8)	50 (13.8)	79 (5.5)
	Mean	0 (NA)[f]	3 (0.6)[e]	20 (5.6)	56 (15.0)	79 (5.5)
Exposed to loud noise in the workplace for current workers (n = 1137)[cd]	Left	0 (NA)[f]	3 (0.6)[e]	10 (2.7)[e]	31 (10.4)	44 (3.9)
	Right	0 (NA)[f]	3 (1.0)[e]	12 (3.5)[e]	29 (9.2)	44 (3.9)
	Mean	0 (NA)[f]	3 (0.7)[e]	11 (2.8)[e]	30 (10.3)	44 (3.9)

Abbreviations: CHD, coronary heart disease; NA, not available.

[a]Unweighted sample sizes and weighted prevalence in parentheses.

[b]Adjusted ORs (95% CIs) for quartiles 1-4: (1) left ear: 1.00, 1.95 (0.75-5.05), 2.86 (1.08-7.55), and 3.88 (1.59-9.50); (2) right ear: 1.00, 1.39 (0.49-3.98), 2.01 (0.75-5.36), and 2.29 (0.86-6.12); (3) mean: 1.00, 0.82 (0.29-2.27), 1.81 (0.78-4.17), and 2.27 (0.96-5.36).

[c]Loud noise means that noise was so loud that a person had to raise voice to be heard.

[d]For currently employed workers who were currently or previously exposed to loud noise in the workplace.

[e]Relative standard error > 30%, indicating the estimated prevalence is not reliable.

[f]Weighted prevalence could not be calculated because there were no CHD cases.