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The Prevalence of Hearing Impairment and Associated Risk Factors

The Beaver Dam Offspring Study

Scott D. Nash, MS; Karen J. Cruickshanks, PhD; Ronald Klein, MD, MPH; Barbara E. K. Klein, MD, MPH; F. Javier Nieto, MD, PhD; Guan H. Huang, PhD; James S. Pankow, PhD, MPH; Theodore S. Tweed, MS

Objective: To estimate the prevalence of hearing impairment (HI) and evaluate the cross-sectional associations of environmental and cardiovascular disease risk factors and HI in middle-aged adults.

Design: Data were collected as part of the Beaver Dam Offspring Study, an epidemiological cohort study of aging. Hearing impairment was defined as a pure-tone average (0.5, 1.0, 2.0, and 4.0 kHz) greater than 25 db hearing level in either ear. Word recognition in competing message (WRCM) was measured using the Northwestern University No. 6 word list. Questionnaire information about behaviors, environmental factors, and medical history was also collected.

Participants: The participants (N=3285) were offspring of participants of the population-based Epidemiology of Hearing Loss Study and ranged in age from 21 to 84 years (mean age, 49 years).

Results: The prevalence of HI was 14.1%, and the mean (SD) WRCM score was 64% (15%). In a multivariate model, after age, sex, education, and occupational noise were controlled for, a history of ear surgery (odds ratio [OR], 4.11; 95% confidence interval [CI], 2.37-7.15), a larger central retinal venular equivalent (OR, 1.77; 95% CI, 1.20-2.60 [fourth quartile vs first quartile]), and a higher hematocrit percentage (OR, 0.77; 95% CI, 0.63-0.95 [per 5%]) were independently associated with HI. Factors associated with lower WRCM scores were similar but also included mean intima-media thickness (mean difference, –0.63%; 95% CI, –1.06% to –0.19%; *P*=.005 [per 0.1 mm]) and statin use (mean difference, –2.09%; 95% CI, –3.58% to –0.60%; *P*=.005).

Conclusions: Hearing impairment is a common condition in middle-aged adults. Cardiovascular disease risk factors may be important correlates of age-related auditory dysfunction.

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Author Affiliations: Departments of Population Health Sciences (Mr Nash and Drs Cruickshanks and Nieto) and Ophthalmology and Visual Sciences (Drs Cruickshanks, R. Klein, and B. E. K. Klein), University of Wisconsin School of Medicine and Public Health, and Department of Communicative Disorders, University of Wisconsin (Mr Tweed), Madison; Institute of Statistics, National Chiao Tung University, Hsinchu, Taiwan, Republic of China (Dr Huang); and Division of Epidemiology and Community Health, University of Minnesota School of Public Health, Minneapolis (Dr Pankow).

HEARING IMPAIRMENT (HI) is one of the most common chronic conditions in older adults, affecting at least 29 million Americans.[1] Population-based epidemiological prevalence estimates range from 20.6% in adults aged 48 to 59 years to 90% in adults older than 80 years.[2] The 10-year incidence of HI in these 2 age groups has been estimated to be 22% and 100%, respectively.[3] Furthermore, the severity of this condition has been shown to be associated with a poorer quality of life, communication difficulties, impaired activities of daily living, dementia, and cognitive dysfunction.[4,5]

Besides older age and male sex, environmental factors such as loud noise, socioeconomic status, and ear infections have been associated with HI.[1,3,6-8] Traditional cardiovascular disease (CVD) risk factors may also be important contributors to worse hearing, although the findings from different epidemiological cohorts have been inconsistent. Associations have been found between HI and a history of smoking[6,9]; CVD, including myocardial infarction and stroke[6,10]; higher blood pressure or hypertension[10,11]; and diabetes.[6,12] Most data regarding the association of CVD risk factors and HI have come from the study of cohorts of older persons, after noise and occupational exposures have ceased. Fewer studies have focused on risk factors for HI in younger adults in whom there may be other competing exposures. Recent reports from the National Health and Nutrition Examination Survey (NHANES) (age range, 20-69 years) have shown that persons with increased occupational and firearm noise expo-

sure, increased pack-years of smoking, and diabetes are more likely to have HI.[8,13]

Epidemiological data are needed to characterize the burden of HI across all adult age groups, especially in those younger than 60 years. Also, to investigate associations between CVD risk factors and HI, both pure-tone and speech audiometry (word recognition in competing message [WRCM]) were used in analytic modeling, as there may be both peripheral and central dysfunction in HI. Identifying possible modifiable risk factors may allow early interventions to delay the onset of HI and to diminish the impact on quality of life.

METHODS

The Epidemiology of Hearing Loss Study is an ongoing population-based cohort study that was started in 1993 in Beaver Dam, Wisconsin, to measure hearing outcomes and their risk factors. The original cohort comprised 3753 participants ranging in age from 48 to 92 years who were then followed up every 5 years. In 2005, the offspring of the Epidemiology of Hearing Loss Study participants were enrolled in the Beaver Dam Offspring Study (BOSS), a study of multisensory impairments and aging. Of the 4965 eligible offspring, 3285 (66.2%) participated in BOSS, 1657 (33.4%) did not participate, and 23 (0.5%) died. Data collection occurred from 2005 to 2008. The analysis for this report used data from those 2837 participants with audiometric data (86.4% of participants). The BOSS methods were approved by the internal review board of the University of Wisconsin, Madison, and all participants provided written informed consent.

The hearing examination included otoscopy, tympanometry, and pure-tone air- and bone-conduction audiometry as well as word recognition in quiet and in competing message (WRCM). All examiners were trained and certified in all study protocols. An abnormal otoscopic examination result was defined as drainage, a bulging or retracted eardrum, a visible air-liquid line, or a perforated eardrum. Consistent with guidelines of the American Speech-Language-Hearing Association,[14] audiometric testing was conducted in a sound-treated booth (Industrial Acoustics Co, New York, New York) using a clinical audiometer (GSI-61; Grason-Stadler, Eden Prairie, Minnesota). Headphones (TDH-50; Telephonics, Farmingdale, New York) were used for air-conduction testing, and insert earphones (E-A-Rtone 3A; Cabot Safety Corp, Indianapolis, Indiana) and masking were used when appropriate.

Air-conduction thresholds were determined for each ear at 0.5, 1.0, 2.0, 3.0, 4.0, 6.0, and 8.0 kHz. The clinical audiometer was calibrated every 6 months according to American National Standards Institute standards.[15] Ambient noise levels were routinely measured throughout the study to ensure that testing conditions remained within American National Standards Institute standards.[16] A pure-tone average (PTA) was calculated using the thresholds from the 0.5-, 1.0-, 2.0-, and 4.0-kHz frequencies. Hearing impairment was defined as a PTA (0.5, 1.0, 2.0, and 4.0 kHz) greater than 25 dB hearing level (HL) in either ear (worse ear). In our cohort, 93% of the population was concordant for HI status between ears.

Tests of word recognition in quiet and WRCM were conducted in a sound-treated booth using the Northwestern University Auditory Test No. 6.[17] A 25-word word list was presented to the better ear at 36 dB HL above the individual's threshold at 2 kHz (using a single female voice).[17] A competing message (single male talker) was then added at a level 8 dB HL below the speaker's level in the better ear.[17] The WRCM results were reported as percentage correct.

Trained interviewers administered a hearing-related medical history and a noise exposure questionnaire that included questions about ear-related medical history such as Meniere disease, otosclerosis, ear infections, and ear surgery. A positive history of occupational noise exposure was defined as self-reported occupational noise exposure (holding a full-time job that required speaking in a raised voice or louder to be heard) or having driven a farm tractor without a cab.

Data collection included blood pressure measurements (Dynamap Procare 120; GE Medical Systems, Milwaukee, Wisconsin), with hypertension defined as systolic blood pressure greater than or equal to 140 mm Hg, diastolic blood pressure greater than or equal to 90 mm Hg, or a self-reported physician diagnosis of hypertension and current use of antihypertensive medication. Height and weight were measured to calculate body mass index.[2] Obesity was defined as a body mass index greater than or equal to 30 (calculated as weight in kilograms divided by height in meters squared).

Serum total cholesterol levels were measured using a double enzymatic process that produces hydrogen peroxide (Roche Hitachi 911; Roche Hitachi, Indianapolis, Indiana). After further treatment with peroxidase, 40-aminophenazone, and phenol, a colored product was produced and then measured at 505 nm. Serum high-density lipoprotein cholesterol was measured the same way after the other lipoprotein fractions were precipitated off. The white blood cell count and hematocrit percentage were measured in a complete blood cell count at the time of the blood draw. Hemoglobin A_{1c} was measured from whole blood using a glycohemoglobin analyzer (A1c 2.2 Plus; TOSOH Bioscience, San Francisco, California). Diabetes status was defined as a self-report of physician diagnosis or an elevated hemoglobin A_{1C} level greater than or equal to 6.5% at the time of the examination.

Participants were considered to have a history of CVD if they self-reported having had a physician-diagnosed stroke, myocardial infarction, or angina. Carotid intima-media thickness (IMT) was measured at 6 sites in each carotid artery using B-mode ultrasonography (Biosound AU4; Biosound Esaote, Indianapolis, Indiana).[18,19] Retinal vessel caliber measures—the central retinal arteriolar equivalent (CRAE) and the central retinal venular equivalent (CRVE)—were obtained using Ivan software (Fundus Photograph Reading Center, Department of Ophthalmology and Visual Sciences, University of Wisconsin) from digital eye fundus images centered on the optic disc (Canon Dgi-45NM Fundus Camera; Canon Inc, Paramus, New Jersey).[20] These retinal vessel measures have been associated with cardiovascular and cerebrovascular disease in numerous epidemiological studies.[20] It is thought that CRAE and CRVE represent different pathophysiologic phenomena, with a decreased CRAE linked to increased blood pressure and an increased CRVE associated with inflammation and endothelial dysfunction.[21]

The questionnaire captured data on the highest education level obtained, the longest held job, and water source at age 13 years (municipal vs well water). Smoking was defined as ever having smoked more than 100 cigarettes. Pack-years were calculated for smokers (number of cigarettes smoked per day divided by 20 and then multiplied by the number of years smoked). A history of heavy drinking was defined as ever having consumed 4 or more alcoholic beverages daily. Participants were considered physically active if they currently engaged in a regular activity long enough to work up a sweat at least once a week. Participants self-reported their use of statins and nonsteroidal anti-inflammatory drugs.

All analyses were performed using SAS version 9.2 (SAS Institute, Cary North Carolina) and Stata version 11.1 (StataCorp LP, College Station, Texas). Participants with audiometric data were compared with nonparticipants and with those without audiometric data using χ^2 tests for categorical vari-

Table 1. Select Characteristics in the Beaver Dam Offspring Study, 2005-2008[a]

Variable	All (N=3285)	Women (n=1795)	Men (n=1490)
Age, mean (SD), y	49.2 (9.9)	49.0 (9.9)	49.5 (9.9)
Education level, y			
≤12	981 (30.1)	509 (28.7)	472 (31.9)
13-15	1092 (33.5)	628 (35.4)	464 (31.3)
>16	1184 (36.4)	639 (36.0)	545 (36.8)
Longest held job[b]	757 (24.3)	188 (11.2)	569 (39.5)
Municipal water as a child	2464 (75.7)	1344 (75.8)	1120 (75.6)
Noisy job	1439 (43.9)	544 (30.3)	895 (60.2)
History of CVD[c]	109 (3.4)	38 (2.1)	71 (4.8)
Diabetes mellitus[d]	206 (6.3)	95 (5.3)	111 (7.5)
Hypertension[e]	1130 (38.4)	517 (32.3)	613 (45.8)
Obese[f]	1258 (44.7)	620 (40.6)	638 (49.6)
History of heavy drinking	596 (18.3)	180 (10.1)	416 (28.2)
Current smoking	578 (17.7)	286 (16.1)	292 (19.7)
Smoking, pack-years			
0	1784 (55.6)	1063 (60.6)	721 (49.5)
≤11	701 (21.8)	403 (23.0)	298 (20.5)
>11	725 (22.6)	288 (16.4)	437 (30.0)
Weekly exercise	2020 (62.1)	1102 (62.2)	918 (62.0)
Total serum cholesterol, mean (SD), mg/dL	203.3 (39.3)	204.6 (38.6)	201.9 (40.1)
HDL serum cholesterol, mean (SD), mg/dL	50.0 (14.8)	55.2 (15.1)	43.9 (11.8)
White blood cell count, mean (SD), /μL	7300 (2400)	7400 (2000)	7300 (2800)
Hematocrit, %	43.6 (3.6)	41.8 (3.0)	45.7 (3.1)
Mean IMT, mean (SD), mm	0.7 (0.1)	0.6 (0.1)	0.7 (0.2)
CRVE, right eye, mean (SD), μm	224.4 (20.1)	223.5 (20.1)	225.4 (20.0)
CRAE, right eye, mean (SD), μm	150.1 (14.6)	151.4 (14.7)	148.6 (14.3)
Statin use	431 (15.2)	171 (11.0)	260 (20.1)
NSAID use	1694 (59.5)	922 (59.5)	772 (59.6)
History of ear infection	1804 (57.9)	1053 (61.8)	751 (53.2)
History of ear surgery	143 (4.4)	70 (3.9)	73 (5.0)
Meniere disease	17 (0.5)	11 (0.6)	6 (0.4)
Otosclerosis	18 (0.6)	15 (0.8)	3 (0.2)
Abnormal otoscopic examination result	20 (0.8)	11 (0.8)	9 (0.8)
Word recognition in quiet, %	89.6 (9.2)	91.1 (8.6)	88.0 (9.7)
Word recognition in competing message %	63.5 (14.7)	66.2 (12.5)	60.5 (16.3)

Abbreviations: CVD, cardiovascular disease; CRAE, central retinal arteriolar equivalent; CRVE, central retinal venular equivalent; HDL, high-density lipoprotein; IMT, intima-media thickness; NSAID, nonsteroidal anti-inflammatory drug.

SI conversion factors: To convert cholesterol values to millimoles per liter, multiply by 0.0259; white blood cell count to $\times 10^9$/L, multiply by 0.001.

[a] Values are expressed as number (percentage) unless otherwise indicated. The numbers may not add up to 3285 because of missing values.

[b] Production/labor/manufacturing.

[c] Physician-diagnosed stroke, myocardial infarction, or angina.

[d] Physician diagnosis and treated diabetes or elevated hemoglobin A_{1c} level greater than 6.5% at the time of the examination.

[e] Systolic blood pressure greater than or equal to 140 mm Hg, diastolic blood pressure greater than or equal to 90 mm Hg, or physician diagnosis of hypertension and currently taking hypertensive medication.

[f] Body mass index greater than or equal to 30 (calculated as weight in kilograms divided by height in meters squared).

ables and *t* tests for continuous variables. Participants with HI that was asymmetrical (PTA difference between ears >20 dB) were excluded from models evaluating associations with HI (n=70). Logistic regression was used to estimate odds ratios (ORs) and to examine risk factor associations. Selected risk factors included those associated with HI in published articles as well as well-known markers of or risk factors for CVD. Age- and sex-adjusted models were first run for each potential individual risk factor. Those that were associated with HI ($P<.20$) were then entered into a multivariable model. At that stage, those with insignificant *P* values ($P>.05$) that did not substantially change the other variables coefficients (±20%) when removed were left out of the final model. The associations between exposure variables and HI were examined for possible interactions with age and sex. Multivariable least squares regression was also used to estimate mean WRCM scores and to test for associations between exposure variables and WRCM using the same modeling approach.

Analyses were then performed by running the final multivariable model with least squares regression procedures using the worse ear PTA as a continuous outcome. To minimize heterogeneity from multiple causes of HI, the final model was rerun excluding participants with hearing loss that developed before the age of 30 years, a history of ear surgery, or conductive hearing loss as well as the original exclusion criterion based on asymmetrical hearing loss (n=110). Because there is most likely a heritable component to HI, and participants in BOSS were recruited from families, models were rerun using the generalized estimating equation (GEE) method to account for reported familial relationships and to determine whether these relationships affected the results of the study.

RESULTS

Participation in BOSS tended to be higher among eligible persons living closest to Beaver Dam, Wisconsin

Table 2. Prevalence of Hearing Impairment[a] in the Beaver Dam Offspring Study, 2005-2008

	All		Women		Men	
Age, y	No. at Risk	% (95% CI)	No. at Risk	% (95% CI)	No. at Risk	% (95% CI)
21-34	170	2.9 (1.0-6.7)	100	0	70	7.1 (2.4-15.9)
35-44	827	6.4 (4.7-8.1)	458	3.7 (2.0-5.4)	369	9.8 (6.7-17.8)
45-54	1057	10.9 (9.0-12.8)	564	5.9 (3.9-7.8)	493	16.6 (13.3-19.9)
55-64	605	25.1 (21.7-28.6)	321	15.6 (11.6-19.5)	284	35.9 (30.3-41.5)
65-84	178	42.7 (35.4-50.0)	100	33.0 (23.8-42.2)	78	55.1 (44.1-66.2)
Total	**2837**	14.1 (12.9-15.4)	**1543**	8.6 (7.2-10.0)	**1294**	20.7 (18.5-22.9)

Abbreviation: CI, confidence interval.
[a] Pure-tone average (0.5, 1.0, 2.0, and 4.0 kHz) greater than 25 dB hearing level in either ear.

($P<.001$). Participants also tended to be slightly older (48 years vs 46 years, respectively; $P<.001$) and were more likely to be women (54.6% vs 44.4%; $P<.001$) than nonparticipants. After adjustment for age, sex, and location of residence, there was a statistically significant difference in parental history of HI (OR, 1.21; 95% confidence interval [CI], 1.05-1.39) between those participants with hearing examination data and those without (including both participants and nonparticipants).

Participants in BOSS ranged in age from 21 to 84 years (mean age, 49 years); 45.6% of the cohort were men; and 69.9% had more than 12 years of education (**Table 1**). There was a low prevalence of Meniere disease, otosclerosis, ear surgery, and abnormal otoscopic examination results (0.5%, 0.6%, 4.4%, and 0.8%, respectively). The mean (SD) word recognition score in quiet was 89.6% (9.2%), and the mean (SD) WRCM score was 63.5% (14.7%).

The overall prevalence of HI was 14.1% (95% CI, 12.9-15.4) and ranged from 2.9% in persons aged 21 to 34 years to 42.7% in those aged 65 to 84 years (**Table 2**). When analyses were run using the better ear instead of either ear (bilateral HI), the prevalence of HI was 6.8% (192 of 2837 participants) (95% CI, 5.8-7.7). After sex was controlled for, older age was associated with greater odds of HI (OR, 1.58; 95% CI, 1.48-1.67 [per 5-year increase]). Men were more likely than women to have HI after age was controlled for (OR, 3.0; 95% CI, 2.37-3.79).

Age-sex-adjusted models for HI are shown in **Table 3**. Several traditional risk factors for CVD were significantly associated with HI, including pack-years of smoking (OR, 1.61; 95% CI, 1.16-2.23 [>11 vs 0 pack-years]). Hypertension, diabetes, and obesity were not associated with HI in this middle-aged cohort. Several environmental variables, such as having a noisy job, were associated with HI in age- and sex-adjusted models (OR, 1.67; 95% CI, 1.29-2.16).

In the final multivariable model, less education, a noisy job, a history of ear surgery, and a larger CRVE were associated with an increased odds of HI. A higher hematocrit percentage was associated with a decreased odds of HI (**Table 4**). Point estimates and CIs were essentially unchanged when the noisy job variable was limited to noise exposure at the participants' primary job (excluding tractor noise) and in GEE models accounting for familial relationships (results not shown).

Some individuals with HI reported a young age at onset (≤30 years) (n=116) or a history of ear surgery (n=44) or had a measured conductive hearing loss (n=36). When the final model was rerun excluding this group (n=110 with at least 1 condition), the results were similar to those in the whole cohort (**Table 5**). The main difference was that the hematocrit percentage was no longer statistically significantly associated with HI.

After this model was evaluated using a continuous outcome (mean worse-ear PTA), the results were also similar. Age (mean difference, +2.38; 95% CI, +2.17 to +2.58 [per 5 years]), male sex (mean difference, +5.86; 95% CI, +4.93 to +6.79), lower levels of education (mean difference, +2.63; 95% CI, +1.65 to +3.60 [≤12 years vs ≥16 years]), a noisy job (mean difference, +1.46; 95% CI, +0.64 to +2.28), and a history of ear surgery (mean difference, +8.88; 95% CI, 6.87-10.89) were associated with a higher PTA, while the hematocrit percentage (mean difference, –0.82; 95% CI, –1.44 to –0.19 [per 5%]) was associated with a lower PTA.

In a multivariable linear regression model with WRCM as a continuous outcome (**Table 6**), age; sex; education; PTA; reporting a longest held job in labor, production, or farming; statin use; a greater CRVE; and a greater mean IMT were statistically associated with lower mean WRCM scores. Participants who had municipal water as a child had higher scores than those who reported well water sources. The results were similar in GEE models after familial relationships were accounted for (results not shown).

COMMENT

The prevalence of HI was 14.1% in this population of middle-aged adults. Although the prevalence of HI in BOSS was less than 10% among those younger than 45 years, it was substantially greater in older adults. Hearing impairment was more likely in men, in participants with lower education levels, and in those working in noisy occupations or with a history of ear surgery. Other factors associated with HI that could be considered risk factors for CVD were a larger CRVE and a larger hematocrit percentage. Central retinal venular equivalent (a microvascular measure), IMT (a macrovascular measure), and statin use (a possible indicator of a clinician's concern about high CVD risk) were associated with hearing measured by WRCM. These results suggest that there may be cardiovascular antecedents of HI, as measured by pure-tone or speech audiometry, which are detectable even in middle age.

Table 3. Age- and Sex-Adjusted Odds Ratios (ORs) and 95% Confidence Intervals (CIs) for Hearing Impairment[a] in the Beaver Dam Offspring Study, 2005-2008

Variable	OR (95% CI)
Age, per 5 y	1.69 (1.57-1.80)
Male sex	3.48 (2.67-4.54)
Education level, y	
≤12	1.94 (1.42-2.66)
13-15	1.33 (0.95-1.86)
≥16	1 [Reference]
Longest held job[b]	1.68 (1.27-2.22)
Municipal water as a child	0.71 (0.54-0.93)
Noisy job	1.67 (1.29-2.16)
History of CVD[c]	1.63 (0.97-2.73)
Diabetes mellitus[d]	1.21 (0.79-1.83)
Hypertension[e]	1.16 (0.89-1.51)
Obesity[f]	1.18 (0.92-1.52)
History of heavy drinking	1.28 (0.95-1.73)
Current smoking	1.40 (0.99-1.98)
Smoking, pack-years	
0	1 [Reference]
≤11	1.03 (0.77-1.38)
>11	1.61 (1.16-2.23)
Weekly exercise	0.77 (0.60-0.99)
Serum total cholesterol, per 10 mg/dL	1.02 (0.99-1.05)
Serum HDL cholesterol, per 5 mg/dL	0.93 (0.89-0.98)
White blood cell count, 5 × per 1000/μL	1.33 (0.98-1.80)
Hematocrit, per 5%	0.81 (0.67-1.00)
Mean IMT, per 0.1 mm	1.01 (0.93-1.09)
CRVE, quartile	
1st	1 [Reference]
2nd	1.29 (0.88-1.88)
3rd	1.73 (1.20-2.48)
4th	1.67 (1.16-2.41)
CRAE, quartile	
1st	1 [Reference]
2nd	0.94 (0.66-1.34)
3rd	1.12 (0.79-1.58)
4th	1.19 (0.82-1.73)
Statin use	0.78 (0.57-1.07)
NSAID use	0.93 (0.72-1.20)
History of ear infection	1.26 (0.97-1.63)
History of ear surgery	4.44 (2.63-7.49)

Abbreviations: CVD, cardiovascular disease; CRAE, central retinal arteriolar equivalent; CRVE, central retinal venular equivalent; HDL, high-density lipoprotein; IMT, intima-media thickness; NSAID, nonsteroidal anti-inflammatory drug.

SI conversion factors: To convert cholesterol values to millimoles per liter, multiply by 0.0259; white blood cell count to $\times 10^9$/L, multiply by 0.001.

[a] Pure-tone average (0.5, 1.0, 2.0, and 4.0 kHz) greater than 25 dB hearing level in either ear.

[b] Production/labor/manufacturing vs management/technical/service.

[c] Physician-diagnosed stroke, myocardial infarction, or angina.

[d] Physician diagnosis and treated diabetes or an elevated hemoglobin A_{1C} level greater than 6.5% at the time of the examination.

[e] Systolic blood pressure greater than or equal to 140 mm Hg, diastolic blood pressure greater than or equal to 90 mm Hg, or physician diagnosis of hypertension and currently taking hypertensive medication.

[f] Body mass index greater than or equal to 30 (calculated as weight in kilograms divided by height in meters squared).

Table 4. Multivariate Odds Ratios (ORs) and 95% Confidence Intervals (CIs) for Hearing Impairment[a] in 2612 Participants in the Beaver Dam Offspring Study, 2005-2008

Variable	OR (95%CI)
Age, per 5 y	1.70 (1.58-1.83)
Male sex	3.80 (2.76-5.25)
Education level, y	
≤12	1.79 (1.27-2.52)
13-15	1.19 (0.83-1.70)
≥16	1 [Reference]
Noisy job	1.57 (1.19-2.08)
History of ear surgery	4.11 (2.37-7.15)
CRVE, quartile	
1st	1 [Reference]
2nd	1.37 (0.93-2.03)
3rd	1.66 (1.14-2.42)
4th	1.77 (1.20-2.60)
Hematocrit, per 5%	0.77 (0.63-0.95)

Abbreviation: CRVE, central retinal venular equivalent.

[a] Pure-tone average (0.5, 1.0, 2.0, and 4.0 kHz) greater than 25 dB hearing level in either ear.

Table 5. Multivariate Odds Ratios (ORs) and 95% Confidence Intervals (CIs) for Sensorineural Hearing Impairment[a] in 2517 Participants[b] in the Beaver Dam Offspring Study, 2005-2008

Variable	OR (95% CI)
Age, per 5 y	1.99 (1.81-2.19)
Male sex	3.74 (2.55-5.50)
Education level, y	
≤12	1.77 (1.17-2.69)
13-15	1.18 (0.76-1.84)
≥16	1 [Reference]
Noisy job	1.45 (1.03-2.04)
CRVE, quartile	
1st	1 [Reference]
2nd	1.40 (0.88-2.22)
3rd	1.59 (1.01-2.52)
4th	1.69 (1.06-2.69)
Hematocrit, per 5%	0.81 (0.63-1.03)

Abbreviation: CRVE, central retinal venular equivalent.

[a] Pure-tone average (0.5, 1.0, 2.0, and 4.0 kHz) greater than 25 dB hearing level in either ear.

[b] Excludes participants with a hearing loss that developed before the age of 30 years, a history of ear surgery, conductive hearing loss, and hearing asymmetries greater than 20 dB.

The overall prevalence of HI in our study was similar to, although somewhat lower than, that in NHANES (16.1%), a nationwide estimate among adults younger than 65 years.[1] The small difference between this estimate of prevalence and ours could possibly be explained by differences in the distributions of age, sex, race/ethnicity, other characteristics related to HI, or sampling variability. The Epidemiology of Hearing Loss Study, Framingham Heart Study, and Blue Mountains Hearing Study cohorts had similar prevalence estimates (46% in adults aged 48-92 years, 47% in adults 57-89 years, and 44.6% in adults aged >49 years, respectively).[2,7,22]

The most consistent cardiovascular factors associated with worse hearing measured by PTA or WRCM in BOSS were microvasculature and macrovascular measurements. A larger CRVE was associated with both HI and WRCM, and a larger carotid artery IMT was associated with WRCM. A few studies have examined the relationships between microvasculature and macrovascular factors and HI. In the Blue Mountains Hearing Study, retinopathy was associated with HI in women, but wider retinal venular diameter and narrower retinal arteriolar

diameter were not associated with HI in either men or women.[23] It is not known what accounts for the differences between studies. The CRVE is associated with retinal tissue hypoxia, systemic inflammation, and high lipid and glucose levels. Some of these measures have been hypothesized to be involved in the pathogenesis of HI.[12,13] Intima-media thickness is a subclinical measure of atherosclerosis, which predicts adverse cardiovascular outcomes.[24] Therefore, these results may add support to a possible cardiovascular link to HI.

Participants taking statin drugs had lower mean WRCM scores then those not taking statins. Statin drugs are mostly prescribed to persons with high cholesterol levels. Although serum cholesterol levels themselves were not associated with WRCM scores in BOSS, individuals who were taking statins may represent the ones with the worst cholesterol level profiles. Lipids have been implicated in the atherosclerotic process and may indirectly influence blood flow to both the brain and the microvasculature of the ear. The WRCM task may be capturing age-related changes in the central auditory cortex.

Participants with a higher hematocrit percentage were less likely to have HI, contrary to the expectation that the hematocrit is strongly correlated with, and can act as a marker of, blood viscosity.[25] Increased viscosity has been shown to be associated with increased blood pressure and ischemic heart disease as well as with diminished oxygen levels in the cochlea in animal models.[25-27] Gates et al[10] found no association between hematocrit and hearing thresholds in the Framingham cohort. Other studies have shown that individuals with worse hearing or hearing loss had increased blood viscosity or hematocrit levels.[28] The contradictory results of this study could be attributable to the possibility that (1) the hematocrit percentage is an imperfect surrogate for blood viscosity; (2) that blood vessels may be less atherosclerotic in middle-aged adults and therefore more resilient to increases in viscosity; or (3) that hematocrit levels in persons in this age range may be more a reflection of diet or vitamin use. Alternatively, low hematocrit levels could also damage hearing because of associated impairment in oxygen transport. Low hematocrit levels and anemia have been linked to CVD and cardiomyopathy.[29,30]

Socioeconomic status was associated with worse hearing when measured either by PTA (education) or by WRCM (longest held job, municipal water as a child). Municipal water as child (as opposed to well water) could be a marker of either increased socioeconomic status or decreased exposure to substances such as pesticides in drinking water. Prevalence and incidence studies from the Epidemiology of Hearing Loss Study[2,3] as well as national prevalence estimates from NHANES have shown associations with HI and level of education and occupation.[1] Although persons with lower levels of education may tend to work in occupations that have higher levels of noise exposure (such as production, labor, or manufacturing), the education-HI association remained after occupational noise was controlled for. Low socioeconomic status may be associated with less healthy behaviors and less access to health care and has been consistently shown to be associated with CVD and therefore may be associated with HI through a CVD pathway.[31]

Table 6. Multivariate Mean Differences and 95% Confidence Intervals (CIs) for Word Recognition in Competing Message in 2597 Participants in the Beaver Dam Offspring Study, 2005-2008[a]

	Mean Difference (95% CI)	*P* Value
Age, per 5 y	−1.52 (−1.85 to −1.19)	<.001
Male sex	−3.11 (−4.25 to −1.98)	<.001
Education level, y		
≤12	−2.07 (−3.45 to −0.69)	.003
13-15	−1.84 (−3.10 to −0.59)	.004
≥16	1 [Reference]	
Longest held job[b]	−2.21 (−3.56 to −0.86)	.001
Statin use	−2.09 (−3.58 to −0.60)	.005
CRVE, quartile		
1st	1 [Reference]	
2nd	−2.67 (−4.11 to −1.24)	<.001
3rd	−1.52 (−2.93 to −0.11)	.04
4th	−2.37 (−3.79 to −0.95)	.001
Mean IMT, per 0.1 mm	−0.63 (−1.06 to −0.19)	.005
PTA, per 5 dB	−1.01 (−1.21 to −0.81)	<.001
Municipal water as a child	+1.29 (+0.13 to +2.44)	.03

Abbreviations: CRVE, central retinal venular equivalent; IMT, intima-media thickness; PTA, pure-tone average (0.5, 1.0, 2.0, and 4.0 kHz).
[a] Model $R^2 = 0.20$.
[b] Production/labor/manufacturing vs management/technical/service.

Although several CVD risk factors were associated with HI in this study, smoking, diabetes, and hypertension were not. The lack of associations may be attributable to the younger age of this cohort or to the fairly low prevalence of prevalent CVD (3.4%), diabetes (6.3%), and smoking exposure (17.7% current smokers; 22.6% >11 pack-years). Agrawal et al[8] used a much higher cut point for pack-years of smoking and had a higher prevalence of diabetes, which may have allowed them increased power for detecting an association in NHANES. Although 38.4% of the current cohort was considered hypertensive, data were not available to assess the duration of hypertension, and because of the young mean age in BOSS, these participants may not have had hypertension long enough to have affected hearing.

Participants with a history of a noisy job and a history of ear surgery had increased odds of having HI. Relationships between occupational noise and worsening hearing have been shown in many contexts, including large population-based cohort studies.[2,6,7] A report using NHANES data showed that individuals with occupational loud noise exposure had a 60% increased odds of HI.[8] Our assessment of occupational noise exposure was based on self-report, which may result in an underestimation of the effects of noise exposure. It is possible that there remained some residual confounding owing to noise in our multivariate models. Participants with ear surgery reported procedures such as tympanoplasty and mastoidectomy, which can have clear direct effects on hearing.

In this study, HI was defined by the PTA in the worse ear to avoid underestimation of the prevalence of HI. Although this definition may increase the heterogeneity of types of HI, it is a useful measure of the number of persons with HI in the cohort and has been used in other

epidemiological studies of HI.[1-3,8,9,12] In this cohort, 93% of the participants were concordant between ears.

Other studies have used other combinations of frequencies or other audiogram patterns to reduce the heterogeneity inherent in cross-sectional studies of HI. However, we have previously shown in population-based cohorts that most cases of HI have a typical sensorineural pattern, with high frequencies affected more than, and before, lower frequencies.[32] There were too few individuals with a flat hearing loss in the low range to support subset analyses (n=16). To assess the impact of heterogeneity of HI, we conducted subset analyses that excluded cases in which the HI might be attributable to trauma or middle ear problems. In the subset of participants (n=2517) in which those with an onset of HI before the age of 30 years, a history of ear surgery, or conductive or asymmetrical hearing loss were excluded, the risk factor model results were similar to those in the whole population.

There are few current epidemiological studies that have measured hearing in middle-aged adults.[33] The BOSS cohort had a large sample size, data were collected using standardized protocols and accepted methodologies, and there were multiple hearing end points, allowing a clearer understanding of factors associated with HI. Using both pure-tone and speech audiometry measures, we found consistent evidence of associations between cardiovascular risk factors and HI. Responses to tone and speech audiometric tests reflect the function of the auditory system, including central processing, but the additional complexity of speech understanding may make these tasks better indicators of central processing. Our findings with tones and WRCM suggest that age-related changes in the cochlear and central auditory association areas may contribute to HI in adults.

This study was cross-sectional and could not demonstrate causal relationships. As described in the "Results" section, participants in BOSS had significantly higher odds of a parental history of HI than nonparticipants, and it has been shown that HI is highly heritable, with estimates ranging from 47% to 68%, depending on the statistical adjustments for confounders.[34] Therefore, our study may have an overestimated prevalence of HI in white adults. Overestimation was probably unlikely, however, since the prevalence of HI in BOSS (14.1%) was close to, and even lower than, a national estimate from the NHANES for non-Hispanic whites (18.0%).[1] Furthermore, when GEE models were used to allow clustering of participants based on family structure, associations and their standard errors between risk factors and HI differed only slightly. Despite biological plausibility and predefined statistical procedures, some of the associations found in multivariable modeling may have been attributable to type 1 errors.

In conclusion, this study described the prevalence of HI in a cohort of adults ranging in age from 21 to 84 years and identified possible modifiable correlates (CRVE, hematocrit percentage, statin use, and mean IMT) of auditory dysfunction measured by HI and WRCM, suggesting that HI, if detected early, may be a preventable chronic disease.

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Correpondence: Scott D. Nash, MS, Department of Population Health Sciences, University of Wisconsin School of Medicine and Public Health, 610 N Walnut St, Rm 1036 WARF, Madison, WI 53726 (snash@wisc.edu).
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