Low vitamin D status is associated with hearing loss in the elderly: a cross-sectional study

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ABSTRACT

Background: The elderly are at increased risk of both hearing loss (HL) and osteoporosis. Bone mineral density (BMD) has been putatively linked to HL. However, the roles of serum calcium concentrations and vitamin D status have yet to be elucidated.

Objectives: The purpose of this study was to examine the relation between vitamin D status, parathyroid hormone (PTH), total calcium, BMD, and HL in a nationally representative sample of elderly adults.

Methods: Using the NHANES (2005–2010), audiometry and BMD data of 1123 participants aged ≥70 y were analyzed in a cross-sectional manner. HL was defined as pure tone averages >25 dB HL at 500, 1000, and 2000 Hz (low frequency); 500, 1000, 2000, and 4000 Hz (speech frequency); and 3000, 4000, 6000, and 8000 Hz (high frequency) in either ear. Multivariable logistic regression was used to examine the relation between HL and total 25-hydroxyvitamin D [25(OH)D], PTH, total calcium, and BMD, adjusting for covariates.

Results: In multivariable analyses, total 25(OH)D < 20 ng/mL was found to be associated with greater odds of low-frequency HL (OR: 2.02; 95% CI: 1.28, 3.19) and speech-frequency HL (OR: 1.96; 95% CI: 1.12, 3.44). A 1-unit decrease in femoral neck BMD (OR: 4.55; 95% CI: 1.28, 16.67) and a 1-unit decrease in total spine BMD (OR: 6.25; 95% CI: 1.33, 33.33) were found to be associated with greater odds of low-frequency HL. Serum PTH and total calcium were not found to be associated with HL.

Conclusions: In the elderly, low vitamin D status was associated with low-frequency and speech-frequency HL. Low vitamin D status may be a potential risk factor for age-related HL. *Am J Clin Nutr* 2021;113:456–466.

Keywords: vitamin D, hearing loss, osteoporosis, bone mineral density, calcium, NHANES

Introduction

Hearing loss (HL) affects nearly two-thirds of adults aged ≥70 y in the US population (1). Age-related HL is a disabling condition and has been found to be linked to increased risk of falls, depressive symptoms, and the development of dementia in the elderly (2–4). HL in the elderly may be related to normal aging, prior noise exposure, medical comorbidities, and nutritional status, among others, and is likely genetically influenced (5). Osteoporosis, also an age-related condition, has been putatively linked to HL (6). Osteoporosis is a systemic disease characterized by low bone mass, bone fragility, and increased risk of fractures (6). It has been suggested that demineralization of the temporal bone, which includes the otic capsule, can affect the cochlea and therefore may lead to sensorineural hearing loss (SNHL) (7). Middle ear transmission may also be affected because of microfractures in the ossicles (8). Whereas several studies supported a link between low bone mineral density (BMD) and impaired hearing (9–15), other studies have found limited to no association between low BMD and HL (16, 17).

Abnormal calcium homeostasis has been implicated in osteoporosis (18). Although parathyroid hormone (PTH) and ionized calcium also play a role, vitamin D is the principal regulator of calcium homeostasis. The roles of serum calcium concentrations and vitamin D in HL remain to be elucidated. Within the inner ear, calcium ions play an important role in the conduction of electrical impulses between nerve fibers and hair cell transduction (19, 20). Low concentrations of vitamin D, a key regulator of calcium absorption and bone metabolism, were linked to HL in an early case series (21). More recently, a cross-sectional study of 638 diabetic patients found an association between vitamin D deficiency and HL (22). In a smaller study, low vitamin

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Supplemental Table 1 is available from the "Supplementary data" link in the online posting of the article and from the same link in the online table of contents at https://academic.oup.com/ajcn/.

Data Availability: Data described in the article and code book are publicly accessible online via the NHANES website (https://wwwn.cdc.gov/nchs/nhanes/). Analytic code will be made available upon request.

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Abbreviations used: BMD, bone mineral density; CHF, congestive heart failure; LC-MS/MS, liquid chromatography–tandem mass spectrometry; NCHS, National Center for Health Statistics; PTH, parathyroid hormone; SNHL, sensorineural hearing loss; SPL, sound pressure level; TGFβ, transforming growth factor β; 25(OH)D, 25-hydroxyvitamin D.

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D status was associated with a higher risk of sudden SNHL (23). Interestingly, high vitamin D concentrations have also been associated with worse hearing at mid and high frequencies (24), whereas another study found no association between vitamin D status and HL among postmenopausal osteoporotic women (12).

The purpose of this study is to examine the relation between vitamin D status, PTH status, total calcium, BMD, and HL in a nationally representative sample. The study is important because it may identify possible risk factors for age-related SNHL.

Methods

Data source and study design

The NHANES is a continuous cross-sectional series of surveys administered by the National Center for Health Statistics (NCHS). It is reviewed and approved by the NCHS Research Ethics Review Board. A complex, multistage probability sampling design is used to select a nationally representative sample of the resident civilian noninstitutionalized US population. The survey consists of an interview component with demographic, socioeconomic, dietary, and health-related questions, and an examination component comprising medical, dental, and physiological measurements and laboratory tests. Data files are publicly accessible online via the NHANES website (https://wwwn.cdc.gov/nchs/nhanes/). The current study utilizes data from the 2005/2006 and 2009/2010 cycles, the most recent cycles where both audiometry and BMD data were available for subjects of the same age group. This study includes participants aged ≥70 y for whom data on audiometry, vitamin D, PTH, total calcium, BMD, and relevant covariates were available.

Audiometric measurements and definition of HL

Audiometry data were available for 1670 participants aged ≥70 y (**Figure 1**). A Welch Allyn otoscope (model 25020) was used for otoscopic examination of the ears. The Earscan Acoustic Impedance tympanometer (Micro Audiometrics) was used to evaluate the functional health of the middle ear system. Tympanometry was performed by measuring the sound pressure level (SPL) of a 226-Hz probe tone introduced into the ear canal at 85 dB SPL while varying the air pressure in the ear canal from 200 daPa to −312 daPa. The quality of the tympanogram was noted along with the peak response. Audiometric tests were administered by a trained examiner in a dedicated sound-isolating room (model Delta 143; Acoustic Systems) in the mobile examination center (25). Hearing threshold testing was conducted on both ears at 7 frequencies (500, 1000, 2000, 3000, 4000, 6000, and 8000 Hz) using a model AD226 audiometer (Interacoustics). Hearing sensitivity was measured through pure tone air conduction audiometry by presenting pure tone signals to the ear through earphones and varying the signal intensity until the hearing threshold was determined for each frequency. Automated audiometric testing was used unless the examinee had difficulties operating the response switch, in which case manual testing was used. Signal intensity ranged from −10 to 100 decibels (dB) at 500–6000 Hz and from −10 to 90 dB at 8000 Hz for automated audiometric testing. For the manual audiometric mode, signal intensity through 120 dB and 110 dB were used at 500–6000 Hz and 8000 Hz, respectively. Subjects who were unable to remove hearing aids for testing and subjects with ear pain who could not tolerate headphones were excluded.

SNHL was inferred when findings from the otoscopic examination were normal and there were adequate- or good-quality results from the tympanogram with a peak of >0.3 mL. Individuals with abnormal otoscopic results, poor-quality tympanogram results, or a peak response of ≤0.3 mL were excluded from further analyses because these individuals may have had conductive or mixed HL (26, 27). Among the 1670 subjects with available audiometric data, 547 were excluded based on these criteria, leaving 1123 subjects available for analysis (Figure 1).

In this study, the primary outcome variables are low-frequency, speech-frequency, and high-frequency HL. Low-frequency HL was defined as pure tone averages at 500, 1000, and 2000 Hz >25 dB HL in either ear; speech-frequency HL was defined as pure tone averages at 500, 1000, 2000, and 4000 Hz >25 dB HL in either ear; and high-frequency HL was defined as pure tone averages at 3000, 4000, 6000, and 8000 Hz >25 dB HL in either ear.

Vitamin D status

For the 2005/2006 cycle, the DiaSorin RIA 25-hydroxyvitamin D [25(OH)D] assay (CV = 9.1%–10.6%) was used. The data were converted using a regression equation developed by the liquid chromatography–tandem mass spectrometry (LC-MS/MS) reanalysis of sera from 1448 participants (28):

$$\text{LC} - \text{MS/MS}_{\text{equivalent}} = 8.36753 + 0.97012 * \text{RIA}_{\text{original}} \quad (1)$$

The 2005/2006 25(OH)D data used in this study were the LC-MS/MS equivalent 25(OH)D measurements that could be combined with the 2009–2010 25(OH)D data for analysis.

For 2007–2010, ultra-high-performance LC-MS/MS (CV = 3.3%–5.2%) was used for the quantitative detection of 25-hydroxyvitamin D_3 [25(OH)D_3], epi-25-hydroxyvitamin D_3 [epi-25(OH)D_3], and 25-hydroxyvitamin D_2 [25(OH)D_2]. Total 25(OH)D was calculated as the sum of 25(OH)D_2 and 25(OH)D_3. Low vitamin D status was defined as total 25(OH)D concentrations <20 ng/mL (50 nmol/L) in this study. This cutoff was chosen based on provocative testing in healthy adults demonstrating a PTH decrease in those with 25(OH)D < 20 ng/mL who received vitamin D supplementation, but not in those with 25(OH)D >20 ng/mL (29). Vitamin D status was also examined as a categorical variable with 3 categories (<20 ng/mL, 20 ng/mL to <30 ng/mL, ≥30 ng/mL). The following equation was used to convert 25(OH)D concentrations in nanomoles per liter to nanograms per milliliter (28):

$$1\ \text{ng/mL} = 2.4959\ \text{nmol/L} \quad (2)$$

PTH

PTH concentrations were collected for the 2005/2006 cycle using the Elecsys 1010 analyzer (CV = 2.2%–5.3%; Roche Diagnostics). For PTH status in this study, the normal range was defined as 10–65 pg/mL. Because there was only 1 subject with

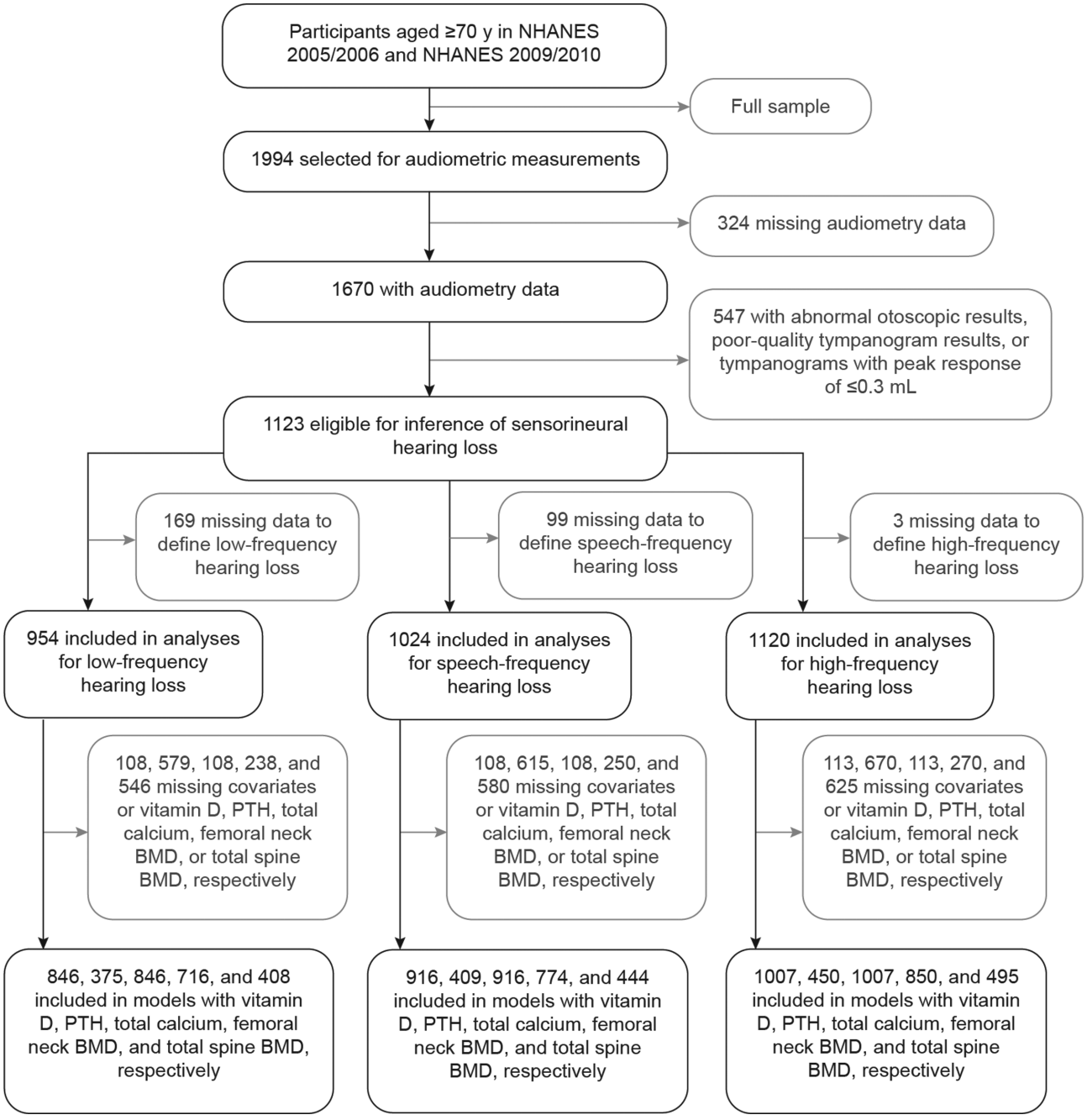

FIGURE 1 Flow diagram of analytic sample of adults aged ≥70 y from NHANES 2005/2006 and NHANES 2009/2010. BMD, bone mineral density; PTH, parathyroid hormone.

low PTH, the low and normal range categories were collapsed to avoid empty cells.

Total calcium

Total calcium was determined as part of the routine biochemistry profile with a Synchron LX20 (CV = 1.0%–1.2%; Beckman Coulter, Inc.) until 2007 and a UniCel DxC 800 Synchron (CV = 0.9%–2.8%; Beckman Coulter, Inc.) from 2008 onwards. For subjects with hypoalbuminemia (albumin concentrations < 4.0 mg/dL), their total calcium was corrected using the following equation:

$$\text{correctedcalcium} = \text{totalcalcium} + 0.8(4 - \text{serumalbumin}) \quad (3)$$

For calcium status in this study, hypocalcemia was defined as total calcium < 8.4 mg/dL and hypercalcemia was defined as concentrations >10.2 mg/dL. Because there was only 1 subject with hypocalcemia, the low and normal range categories were collapsed to avoid empty cells.

BMD

Femur and spine scans were performed with a Hologic QDR-4500A fan-beam densitometer (Hologic, Inc.) by trained and certified radiology technologists. The radiation exposure from DXA for the femur or spine scans is <20 uSv for both. Participants who weighed >300 lbs (136 kg), had a self-reported history of radiographic contrast material use in the past 7 d, or had nuclear medicine studies in the past 3 d were excluded from DXA scans. For femur scans, the left hip was routinely scanned unless the participant reported a fractured left hip, a left hip replacement, or a pin in the left hip, in which case the right hip was scanned. Scans were analyzed by the Department of Radiology at the University of California, San Francisco, using Hologic Discovery software version 12.4 (Hologic, Inc.).

Frailty

To adjust for the potential confounding effects of frailty, a modified 4-item frailty phenotype score (30, 31) was calculated using the following items:

- Exhaustion, defined by answering "some difficulty," "much difficulty," or "unable to do" when asked, "How much difficulty do you have walking from one room to another on the same level?"
- Low physical activity, defined as answering "no" when asked, "Over the past 30 days, did you do moderate activities for at least 10 minutes that cause only light sweating or a slight to moderate increase in breathing or heart rate?" For the 2009–2010 cycle, this question was split up into work moderate activities and recreational moderate activities. Low physical activity in these cases is defined as answering "no" to both questions.
- Weakness, defined by answering "some difficulty," "much difficulty," or "unable to do" when asked, "How much difficulty do you have lifting or carrying something as heavy as 10 pounds [like a sack of potatoes or rice]?"
- Low body weight, defined by BMI $\leq$ 18.5 kg/m^2.

Frail individuals were defined as those with 3 or 4 of the items; prefrail individuals were defined as those with 1 or 2 of the items; and robust individuals were defined as those with no items present.

Other variables

Demographic characteristics (gender, age, race/ethnicity, education level) were self-reported in the NHANES questionnaire. Diabetes status was determined by a "yes" or "no" answer to the question, "Other than during pregnancy, have you ever been told by a doctor or health professional that you have diabetes or sugar diabetes?" The answer of "borderline" was considered equivalent to "yes." Hypertension status was determined by a "yes" or "no" answer to the question, "Have you ever been told by a doctor or other health professional that you had hypertension, also called high blood pressure?" History of stroke was determined by a "yes" or "no" answer to the question, "Has a doctor or other health professional ever told you that you had a stroke?" History of congestive heart failure (CHF) was determined by a "yes" or "no" answer to the question, "Has a doctor or other health professional ever told you that you had congestive heart failure?" Smoking status was coded as ever or never and determined from the questions, "Have you smoked at least 100 cigarettes in your entire life?" and "Do you now smoke cigarettes?" Supplement use was determined by a "yes" or "no" answer to the question, "Have you used or taken any vitamins, minerals, or other dietary supplements in the past month?" Participants' height and weight were measured by trained health technicians using standardized techniques and equipment. BMI was calculated (in kg/m^2). The serum creatinine concentration was determined as part of the routine biochemistry profile with a Synchron LX20 (CV = 1.1%–2.2%; Beckman Coulter, Inc.) until 2007 and a UniCel DxC 800 Synchron (CV = 1.1%–5.4%; Beckman Coulter, Inc.) from 2008 onwards.

Statistical analysis

The 2005/2006, 2007/2008, and 2009/2010 cycles were combined, and 6-y sampling weights were constructed by using one-third of the 2-y sampling weight (WTMEC2YR) constructed by the NHANES to account for oversampling in complex survey design, survey nonresponse, and poststratification. All analyses accounted for complex survey design as per NCHS analytic guidelines (32).

Rao–Scott chi-square tests were used to evaluate the association between HL and categorical variables; *t* tests were used to evaluate the association between HL and continuous variables; and *F* tests were used to evaluate the association between pure tone hearing levels and the main exposures of interest categorized as quartiles. The 3 primary outcome variables are low-frequency, speech-frequency, and high-frequency HL. The 5 main exposures of interest were vitamin D status, PTH, total calcium, femoral neck BMD, and total spine BMD. Among other variables of interest (gender, age, BMI, education level, race/ethnicity, hypertension, diabetes, history of stroke, history of CHF, smoking status, supplement use, frailty, and creatinine concentration), gender, age, race/ethnicity, supplement use, frailty, and variables found to be associated with HL at the $P < 0.10$ level were included as covariates for each of the 5 main exposures of interest. Multivariable logistic regression was used to examine the relations between HL and vitamin D status as a binary variable and as a variable with 3 categories, PTH as a binary variable, and total calcium, femoral neck BMD, and total spine BMD as continuous variables, respectively, adjusting for covariates. For multivariable models, $P < 0.05$ was considered statistically significant. All analyses were conducted using SAS Survey procedures in SAS 9.4 (SAS Institute).

Results

Characteristics

The study sample included 1123 participants aged ≥70 y sampled from the US population. The weighted mean age of US adults aged ≥70 y was 76.4 y (**Table 1**) and 59.3% were female (**Table 2**). The majority of US adults aged ≥70 y had HL; 67.1%, 80.6%, and 92.6% had low-frequency, speech-frequency, and high-frequency HL, respectively. In addition, 64.1% had hypertension, 20.0% had diabetes, 7.4% had a history of CHF, and 8.9% had a history of stroke. Approximately half (49.5%)

TABLE 1 Characteristics of study population by HL status[1]

Variable	Overall		Low-frequency HL ($n = 954$)			Speech-frequency HL ($n = 1024$)			High-frequency HL ($n = 1120$)		
	n	Weighted mean ± SEM	Yes	No	P value[2]	Yes	No	P value[2]	Yes	No	P value[2]
Mean age, y	1123	76.4 ± 0.13	77.4 ± 0.16	74.7 ± 0.20	<0.01	77.0 ± 0.14	74.2 ± 0.31	<0.01	76.5 ± 0.13	73.7 ± 0.65	<0.01
Mean BMI, kg/m^2	1097	28.0 ± 0.19	28.0 ± 0.26	28.5 ± 0.42	0.38	28.0 ± 0.18	28.5 ± 0.56	0.35	28.0 ± 0.18	28.8 ± 1.15	<0.01
Creatinine, mg/dL	1069	1.06 ± 0.01	1.09 ± 0.02	1.00 ± 0.02	<0.01	1.08 ± 0.02	0.99 ± 0.02	<0.01	1.06 ± 0.01	0.95 ± 0.05	<0.01
Vitamin D, ng/mL	1074	27.5 ± 0.60	27.5 ± 0.76	27.9 ± 0.54	0.56	27.4 ± 0.71	28.0 ± 0.62	0.45	27.5 ± 0.63	26.9 ± 1.39	0.71
Total calcium, mg/dL	1069	9.53 ± 0.02	9.51 ± 0.02	9.58 ± 0.03	0.05	9.52 ± 0.02	9.56 ± 0.04	0.28	9.52 ± 0.02	9.70 ± 0.09	0.05
Parathyroid hormone, pg/mL	482	56.1 ± 1.39	58.4 ± 1.95	52.6 ± 3.20	0.17	57.9 ± 1.50	52.9 ± 3.38	0.19	56.2 ± 1.54	54.8 ± 7.73	0.97
Femoral neck BMD, g/cm^2	925	0.71 ± 0.01	0.70 ± 0.01	0.73 ± 0.01	<0.01	0.71 ± 0.01	0.71 ± 0.01	0.62	0.71 ± 0.01	0.68 ± 0.02	0.15
Total spine BMD, g/cm^2	541	0.98 ± 0.01	0.97 ± 0.01	1.00 ± 0.02	0.07	0.99 ± 0.01	0.98 ± 0.02	0.62	0.98 ± 0.01	0.94 ± 0.04	0.42

[1]Values are weighted means ± SEMs unless otherwise indicated. BMD, bone mineral density; HL, hearing loss.
[2]The t test was used to generate P values for continuous variables.

had never smoked, and 73.7% reported use of a supplement. Furthermore, 23.2% had total 25(OH)D concentrations < 20 ng/mL, 22.8% had PTH concentrations > 65 pg/mL, and only 3.2% had total calcium concentrations > 10.2 mg/dL.

Bivariate comparisons

At the bivariate level, older age ($P < 0.01$) and a higher creatinine concentration ($P < 0.01$) were associated with a higher prevalence of all types of HL (Table 1). Prefrail and frail status ($P = 0.01$) and total 25(OH)D < 20 ng/mL ($P < 0.01$) were associated with higher prevalence of low-frequency HL (Table 2).

Female gender ($P < 0.01$) was associated with lower prevalence of speech-frequency and high-frequency HL, and a higher education level was associated with lower prevalence of low-frequency ($P = 0.02$) and speech-frequency ($P < 0.01$) HL (Table 2). Supplement use was also associated with lower prevalence of low-frequency ($P < 0.01$) and speech-frequency ($P < 0.01$) HL. Compared with non-Hispanic white race, non-Hispanic black race and other race ($P < 0.01$) were associated with lower prevalence of speech-frequency HL. Hypertension ($P = 0.03$) was also associated with lower prevalence of speech-frequency HL.

The associations of smoking status ($P = 0.05$) with higher prevalence of speech-frequency HL and history of CHF ($P = 0.10$) with higher prevalence of low-frequency HL were not statistically significant.

Pure tone hearing levels

The associations of vitamin D concentrations, PTH, total calcium, femoral neck BMD, and total spine BMD with pure tone hearing levels at 500, 1000, 2000, 3000, 4000, 6000, and 8000 Hz were evaluated using the average threshold for both right and left ears (**Supplemental Table 1**). Higher concentrations of PTH were associated with greater thresholds from 1000 Hz to 8000 Hz (all $P < 0.05$). Higher concentrations of calcium were associated with lower thresholds from 2000 to 8000 Hz (all $P < 0.05$). Higher levels of femoral neck BMD were associated with lower thresholds at 500 ($P < 0.01$) and 1000 Hz ($P < 0.01$), and higher levels of total spine BMD were associated with lower thresholds at 500 ($P = 0.02$) and 4000 Hz ($P = 0.01$).

Multivariable logistic regression models

For the outcome low-frequency HL, 846, 375, 846, 716, and 408 participants (Figure 1) were included in multivariable analyses for vitamin D status, PTH, total calcium, femoral neck BMD, and total spine BMD, respectively (**Table 3**). Total 25(OH)D < 20 ng/mL was found to be associated with greater odds of low-frequency HL (OR: 2.02; 95% CI: 1.28, 3.19). 20 < 25(OH)D < 30 ng/mL was not associated with significantly different odds of low-frequency HL compared with 25(OH)D ≥ 30 ng/mL. A decrease in femoral neck BMD (OR: 4.55; 95% CI: 1.28, 16.67) and a decrease in total spine BMD (OR: 6.25; 95% CI: 1.33, 33.33) by 1 unit were both associated with greater odds of low-frequency HL.

TABLE 2 Characteristics of study population and prevalence of HL by characteristics[1]

	Overall		Low-frequency HL ($n = 954$)		Speech-frequency HL ($n = 1024$)		High-frequency HL ($n = 1120$)	
Variable	n	Weighted % (SEE)	Weighted % (SEE)	P value[2]	Weighted % (SEE)	P value[2]	Weighted % (SEE)	P value[2]
Total population	1123		67.1 (2.53)		80.6 (2.24)		92.6 (1.16)	
Gender				0.95		<0.01		<0.01
Female	572	59.3 (1.24)	67.0 (3.02)		76.3 (3.15)		95.4 (1.17)	
Male	551	40.7 (1.24)	67.2 (2.87)		86.5 (2.09)		98.8 (0.47)	
BMI, kg/m^2				0.09		0.80		—
<18.5	17	1.5 (0.49)	80.9 (9.76)		88.6 (8.54)		100 (0)	
18.5 to <25	322	30.3 (1.29)	70.7 (3.83)		81.1 (3.27)		96.8 (1.24)	
25 to <30	405	36.4 (1.38)	62.0 (3.74)		79.3 (3.08)		96.0 (1.64)	
≥30	353	31.8 (1.66)	68.7 (2.74)		81.3 (2.61)		97.3 (0.80)	
Education				0.02		<0.01		0.71
Less than high school	405	29.4 (2.23)	74.4 (2.66)		87.1 (2.33)		97.3 (1.24)	
High school graduate or GED	288	28.6 (1.65)	67.5 (4.33)		81.9 (3.35)		97.0 (1.24)	
Some college or AA	237	23.0 (1.62)	63.6 (4.39)		74.0 (4.10)		97.2 (1.07)	
College graduate or more	191	19.0 (2.05)	59.4 (4.67)		76.2 (3.36)		95.2 (1.96)	
Race/ethnicity				0.05		<0.01		0.24
Non-Hispanic white	791	84.6 (1.73)	68.1 (2.79)		82.2 (2.47)		97.0 (0.87)	
Non-Hispanic black	155	7.5 (1.11)	53.5 (5.40)		64.2 (4.97)		94.1 (1.79)	
Other	177	7.9 (1.31)	69.0 (5.65)		78.4 (4.33)		97.4 (1.01)	
Hypertension				0.19		0.03		0.13
Yes	736	64.1 (1.51)	65.2 (3.32)		78.5 (2.68)		98.1 (0.92)	
No	384	35.9 (1.51)	70.4 (2.98)		84.2 (2.40)		96.1 (0.87)	
Diabetes				0.22		0.16		0.18
Yes	253	20.0 (1.23)	62.3 (4.96)		76.2 (4.14)		94.9 (1.90)	
No	870	80.0 (1.23)	68.3 (2.60)		81.7 (2.30)		97.3 (0.75)	
History of CHF				0.10		0.29		0.76
Yes	89	7.4 (0.89)	76.3 (5.52)		86.5 (5.66)		95.9 (3.38)	
No	1021	92.6 (0.89)	66.2 (2.63)		80.0 (2.22)		96.8 (0.71)	
History of stroke				0.48		0.74		0.98
Yes	104	8.9 (0.99)	71.7 (7.79)		82.0 (5.52)		96.7 (2.76)	
No	1014	91.1 (0.99)	66.8 (2.26)		80.4 (2.16)		96.8 (0.72)	
Smoking status				0.21		0.05		0.36
Ever	586	50.5 (2.1)	65.0 (2.52)		83.2 (2.33)		97.4 (0.71)	
Never	537	49.5 (2.1)	69.2 (3.44)		77.8 (2.94)		96.2 (1.18)	
Supplement use				<0.01		<0.01		0.22
Yes	764	73.7 (1.03)	64.9 (2.70)		78.5 (2.55)		96.4 (0.95)	
No	359	26.3 (1.03)	73.5 (3.13)		86.3 (2.55)		98.0 (0.80)	
Frailty				0.01		0.41		—
Robust	410	39.9 (1.68)	61.3 (3.50)		78.3 (2.93)		97.6 (0.81)	
Prefrail	586	54.7 (1.64)	70.2 (2.71)		82.1 (2.66)		95.6 (1.36)	
Frail	62	5.5 (0.99)	68.2 (5.99)		81.2 (5.92)		100 (0)	
25(OH)D, ng/mL				<0.01		0.14		0.21
<20	284	23.2 (1.82)	75.3 (3.45)		84.4 (2.52)		97.7 (0.93)	
20 to <30	409	39.1 (1.92)	64.7 (2.51)		79.6 (2.69)		95.5 (0.97)	
≥30	381	37.7 (2.74)	66.5 (3.71)		78.7 (2.97)		97.4 (1.15)	
25(OH)D < 20 ng/mL				<0.01		0.06		0.29
Yes	284	23.2 (1.82)	75.3 (3.45)		84.4 (2.52)		97.7 (0.93)	
No	790	76.8 (1.82)	65.6 (2.83)		79.2 (2.53)		96.4 (0.84)	
PTH > 65 pg/mL				0.27		0.53		0.97
Yes	113	22.8 (1.88)	70.5 (4.94)		80.5 (4.36)		95.5 (2.77)	
No	369	77.2 (1.88)	63.5 (4.86)		77.3 (4.06)		95.6 (1.47)	
Total calcium > 10.2 mg/dL				0.18		0.41		0.06
Yes	36	3.2 (0.55)	55.7 (9.46)		73.6 (9.65)		89.9 (6.65)	
No	1033	96.8 (0.55)	68.3 (2.74)		80.6 (2.26)		96.9 (0.67)	

[1]GED, General Educational Development; AA, Associate in Arts; CHF, congestive heart failure; HL, hearing loss; PTH, parathyroid hormone; 25(OH)D, 25-hydroxyvitamin D.

[2]The Rao–Scott chi-square test was used to generate P values for categorical variables. P values may not be available because of empty cells.

TABLE 3 Multivariable logistic regression models for the outcome of low-frequency hearing loss[1]

Variable	Model 1[2] OR (95% CI) ($n = 846$)	Model 2[3] OR (95% CI) ($n = 846$)	Model 3[4] OR (95% CI) ($n = 375$)	Model 4[5] OR (95% CI) ($n = 846$)	Model 5[6] OR (95% CI) ($n = 716$)	Model 6[7] OR (95% CI) ($n = 408$)
25(OH)D < 20 ng/mL	2.02 (1.28, 3.19)	—	—	—	—	—
25(OH)D, ng/mL						
≥30	—	1.0 (ref)	—	—	—	—
20 to <30	—	0.99 (0.74, 1.33)	—	—	—	—
<20	—	2.01 (1.18, 3.41)	—	—	—	—
PTH > 65 pg/mL	—	—	1.06 (0.58, 1.93)	—	—	—
Total calcium (1-unit increase)	—	—	—	0.68 (0.45, 1.03)	—	—
Femoral neck BMD (1-unit decrease)	—	—	—	—	4.55 (1.28, 16.67)	—
Total spine BMD (1-unit decrease)	—	—	—	—	—	6.25 (1.33, 33.33)
Female gender	0.93 (0.65, 1.33)	0.93 (0.64, 1.34)	1.24 (0.70, 2.18)	1.02 (0.70, 1.48)	0.80 (0.50, 1.27)	0.64 (0.33, 1.23)
Age	1.19 (1.13, 1.25)	1.19 (1.13, 1.25)	1.16 (1.08, 1.24)	1.18 (1.12, 1.24)	1.17 (1.11, 1.24)	1.20 (1.12, 1.30)
BMI, kg/m^2						
<18.5	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)
18.5 to <25	0.55 (0.17, 1.75)	0.55 (0.17, 1.75)	0.18 (0.02, 1.37)	0.50 (0.15, 1.68)	0.75 (0.18, 3.10)	0.65 (0.13, 3.14)
25 to <30	0.40 (0.11, 1.50)	0.40 (0.11, 1.50)	0.21 (0.02, 2.09)	0.38 (0.10, 1.45)	0.55 (0.10, 2.97)	0.44 (0.07, 2.77)
>30	0.77 (0.23, 2.58)	0.77 (0.23, 2.58)	0.38 (0.04, 3.19)	0.73 (0.20, 2.60)	1.36 (0.26, 6.98)	1.11 (0.15, 8.00)
Education						
Less than high school	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)
High school graduate or GED	0.85 (0.48, 1.50)	0.85 (0.48, 1.50)	0.55 (0.23, 1.27)	0.84 (0.49, 1.45)	1.01 (0.53, 1.91)	0.91 (0.48, 1.71)
Some college or AA	0.57 (0.32, 1.02)	0.57 (0.32, 1.02)	0.45 (0.14, 1.51)	0.56 (0.31, 1.01)	0.61 (0.31, 1.19)	0.67 (0.31, 1.46)
College graduate or more	0.63 (0.35, 1.12)	0.63 (0.35, 1.12)	0.45 (0.16, 1.27)	0.63 (0.36, 1.11)	0.79 (0.49, 1.26)	0.72 (0.41, 1.28)
Race/ethnicity						
Non-Hispanic white	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)
Non-Hispanic black	0.28 (0.16, 0.50)	0.28 (0.16, 0.50)	0.23 (0.09, 0.60)	0.36 (0.20, 0.64)	0.37 (0.19, 0.69)	0.34 (0.16, 0.70)
Other	0.94 (0.51, 1.74)	0.94 (0.51, 1.73)	2.12 (0.67, 6.68)	0.95 (0.51, 1.75)	0.98 (0.51, 1.89)	0.83 (0.34, 2.04)
Frailty						
Robust	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)
Prefrail	1.09 (0.83, 1.42)	1.09 (0.83, 1.42)	1.01 (0.66, 1.56)	1.15 (0.87, 1.51)	1.16 (0.84, 1.59)	0.82 (0.58, 1.17)
Frail	0.74 (0.35, 1.59)	0.74 (0.34, 1.60)	0.52 (0.17, 1.58)	0.80 (0.36, 1.75)	0.97 (0.38, 2.44)	0.67 (0.17, 2.60)
Supplement use	0.73 (0.48, 1.10)	0.73 (0.48, 1.10)	0.44 (0.22, 0.90)	0.63 (0.44, 0.91)	0.66 (0.42, 1.03)	0.25 (0.10, 0.60)
CHF	1.52 (0.66, 3.49)	1.52 (0.67, 3.48)	1.57 (0.41, 5.95)	1.43 (0.61, 3.36)	1.37 (0.60, 3.17)	1.88 (0.45, 7.94)
Creatinine	1.41 (0.67, 2.96)	1.41 (0.67, 2.96)	1.69 (0.58, 4.98)	1.47 (0.74, 2.91)	1.42 (0.74, 2.73)	1.17 (0.75, 1.83)

[1]BMD, bone mineral density; PTH, parathyroid hormone; 25(OH)D, 25-hydroxyvitamin D; GED, General Educational Development; AA, Associate in Arts; CHF, congestive heart failure.

[2]Logistic regression model containing vitamin D status as a binary variable and variables with $P < 0.10$ in bivariate analyses.

[3]Logistic regression model containing vitamin D status as a categorical variable and variables with $P < 0.10$ in bivariate analyses.

[4]Logistic regression model containing PTH status as a binary variable and variables with $P < 0.10$ in bivariate analyses.

[5]Logistic regression model containing total calcium and variables with $P < 0.10$ in bivariate analyses.

[6]Logistic regression model containing femoral neck BMD and variables with $P < 0.10$ in bivariate analyses.

[7]Logistic regression model containing total spine BMD and variables with $P < 0.10$ in bivariate analyses.

PTH concentration > 65 pg/mL (OR: 1.06; CI: 0.58, 1.93) and increases in total calcium (OR: 0.68; 95% CI: 0.45, 1.03) were not found to be associated with low-frequency HL.

For the outcome speech-frequency HL, 916, 409, 916, 774, and 444 participants (Figure 1) were included in multivariable analyses for vitamin D status, PTH, total calcium, femoral neck BMD, and total spine BMD, respectively (**Table 4**). Total 25(OH)D < 20 ng/mL was found to be associated with greater odds of speech-frequency HL (OR: 1.96; 95% CI: 1.12, 3.44). 20 < 25(OH)D < 30 ng/mL was not associated with significantly different odds of speech-frequency HL compared with 25(OH)D ≥ 30 ng/mL. PTH concentrations > 65 pg/mL (OR: 1.13; CI: 0.49, 2.58), increases in total calcium (OR: 1.00; 95% CI: 0.56, 1.76), decreases in femoral neck BMD (OR: 1.15; 95% CI: 0.18, 7.14), and decreases in total spine BMD (OR: 4.00; 95% CI: 0.71, 25.00) were not found to be associated with speech-frequency HL.

For the outcome high-frequency HL, 1007, 450, 1007, 850, and 495 participants (Figure 1) were included in multivariable analyses for vitamin D status, PTH, total calcium, femoral neck BMD, and total spine BMD, respectively (**Table 5**). Neither vitamin D status, PTH status, total calcium, femoral neck BMD, nor total spine BMD were found to be associated with high-frequency HL, and CIs were wide.

Analyses restricted to male ($n = 415$), female ($n = 431$), and non-Hispanic white ($n = 612$) subgroups yielded similar results, although CIs were wider and, in some cases, not significant. Analyses restricted to non-Hispanic black ($n = 104$) and other race ($n = 130$) subgroups yielded no significant results, and CIs were wide owing to the small sample sizes.

TABLE 4 Multivariable logistic regression models for the outcome of speech-frequency hearing loss[1]

Variable	Model 1[2] OR (95% CI) ($n = 916$)	Model 2[3] OR (95% CI) ($n = 916$)	Model 3[4] OR (95% CI) ($n = 409$)	Model 4[5] OR (95% CI) ($n = 916$)	Model 5[6] OR (95% CI) ($n = 774$)	Model 6[7] OR (95% CI) ($n = 444$)
25(OH)D < 20 ng/mL	1.96 (1.12, 3.44)	—	—	—	—	—
25(OH)D, ng/mL						
≥30	—	1.0 (ref)	—	—	—	—
20 to <30	—	1.05 (0.71, 1.57)	—	—	—	—
<20	—	2.02 (1.15, 3.54)	—	—	—	—
PTH > 65 pg/mL	—	—	1.13 (0.49, 2.58)	—	—	—
Total calcium (1-unit increase)	—	—	—	1.00 (0.56, 1.76)	—	—
Femoral neck BMD (1-unit decrease)	—	—	—	—	1.15 (0.18, 7.14)	—
Total spine BMD (1-unit decrease)	—	—	—	—	—	4.00 (0.71, 25.00)
Female gender	0.44 (0.26, 0.73)	0.45 (0.27, 0.75)	0.44 (0.20, 0.99)	0.45 (0.27, 0.75)	0.37 (0.21, 0.65)	0.25 (0.12, 0.51)
Age	1.20 (1.13, 1.28)	1.20 (1.13, 1.29)	1.17 (1.07, 1.28)	1.20 (1.12, 1.27)	1.19 (1.12, 1.27)	1.23 (1.12, 1.36)
Education						
Less than high school	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)
High school graduate or GED	0.63 (0.36, 1.09)	0.61 (0.35, 1.07)	0.64 (0.26, 1.57)	0.62 (0.36, 1.05)	0.87 (0.48, 1.56)	0.79 (0.42, 1.48)
Some college or AA	0.29 (0.15, 0.55)	0.28 (0.15, 0.53)	0.27 (0.07, 1.06)	0.29 (0.16, 0.54)	0.34 (0.17, 0.69)	0.39 (0.13, 1.24)
College graduate or more	0.38 (0.21, 0.70)	0.37 (0.20, 0.68)	0.29 (0.10, 0.81)	0.38 (0.21, 0.70)	0.50 (0.28, 0.88)	0.36 (0.17, 0.77)
Race/ethnicity						
Non-Hispanic white	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)
Non-Hispanic black	0.22 (0.11, 0.44)	0.23 (0.11, 0.44)	0.21 (0.05, 0.80)	0.27 (0.13, 0.53)	0.29 (0.13, 0.63)	0.32 (0.13, 0.84)
Other	0.59 (0.28, 1.26)	0.59 (0.28, 1.28)	2.65 (0.56, 12.51)	0.61 (0.29, 1.28)	0.62 (0.30, 1.30)	0.65 (0.23, 1.86)
Frailty						
Robust	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)
Prefrail	1.02 (0.69, 1.51)	1.03 (0.69, 1.53)	0.85 (0.46, 1.57)	1.06 (0.71, 1.60)	1.10 (0.72, 1.68)	1.02 (0.58, 1.78)
Frail	1.11 (0.61, 2.00)	1.13 (0.63, 2.02)	1.29 (0.68, 2.47)	1.16 (0.67, 2.00)	1.67 (0.69, 4.07)	1.76 (0.53, 5.91)
Supplement use	0.62 (0.39, 1.00)	0.61 (0.39, 0.95)	0.40 (0.21, 0.77)	0.52 (0.35, 0.77)	0.61 (0.37, 1.00)	0.25 (0.13, 0.50)
Hypertension	0.61 (0.39, 0.97)	0.61 (0.38, 0.96)	0.60 (0.37, 0.97)	0.63 (0.41, 0.98)	0.58 (0.32, 1.05)	0.51 (0.24, 1.05)
Ever smoker	1.36 (0.94, 1.96)	1.46 (1.00, 2.13)	1.49 (0.90, 2.49)	1.34 (0.93, 1.92)	1.28 (0.86, 1.92)	1.75 (1.05, 2.91)
Creatinine	1.21 (0.52, 2.81)	0.91 (0.45, 1.87)	1.20 (0.38, 3.74)	1.19 (0.57, 2.49)	1.16 (0.54, 2.50)	0.98 (0.51, 1.88)

[1]BMD, bone mineral density; PTH, parathyroid hormone; 25(OH)D, 25-hydroxyvitamin D; GED, General Educational Development; AA, Associate in Arts.

[2]Logistic regression model containing vitamin D status as a binary variable and variables with $P < 0.10$ in bivariate analyses.

[3]Logistic regression model containing vitamin D status as a categorical variable and variables with $P < 0.10$ in bivariate analyses.

[4]Logistic regression model containing PTH status as a binary variable and variables with $P < 0.10$ in bivariate analyses.

[5]Logistic regression model containing total calcium and variables with $P < 0.10$ in bivariate analyses.

[6]Logistic regression model containing femoral neck BMD and variables with $P < 0.10$ in bivariate analyses.

[7]Logistic regression model containing total spine BMD and variables with $P < 0.10$ in bivariate analyses.

Discussion

This study investigated the relation between vitamin D status, PTH status, total calcium, BMD, and HL among the elderly in a nationally representative sample. Low vitamin D status, defined as total 25(OH)D < 20 ng/mL, was found to be associated with low-frequency and speech-frequency HL, and lower femoral neck BMD and lower total spine BMD were found to be associated with greater odds of low-frequency HL. No association was found between PTH status, systemic calcium concentrations, and HL.

Past studies investigating the relation between vitamin D status and HL have yielded conflicting results. The findings from our study confirmed the results of 2 smaller cross-sectional studies ($n = 638$ and $n = 68$, respectively). The first found that vitamin D deficiency and lower calcium concentrations were linked to hearing impairment among diabetic patients (22). The second linked vitamin D deficiency to the development of sudden SNHL (23). In addition, patients with vitamin D deficiency were found to exhibit a lower response rate to treatment with steroids than those with sufficient vitamin D concentrations (23). We were unable to detect an association between low vitamin D status and high-frequency HL, but this is likely due to the ubiquity of high-frequency HL in this population of US elderly adults, leaving a very small number of subjects without this outcome ($n = 32$).

In contrast to our findings, Kim et al. (12) found no association between vitamin D status and HL. However, their study sample was restricted to postmenopausal osteoporotic women, and their smaller sample size ($n = 324$) may have limited the power to detect a possible association. In univariate analyses, Kang et al. (24) found that subjects in the highest quartile of vitamin D concentrations (>22.45 ng/mL) had worse hearing at mid and high frequencies, among Korean adults aged 50–80 y, than the subjects in the lowest quartile of vitamin D concentrations (<17.96 ng/mL), but no significant association persisted in multivariable analyses.

TABLE 5 Multivariable logistic regression models for the outcome of high-frequency hearing loss[1]

Variable	Model 1[2] OR (95% CI) ($n = 1007$)	Model 2[3] OR (95% CI) ($n = 1007$)	Model 3[4] OR (95% CI) ($n = 450$)	Model 4[5] OR (95% CI) ($n = 1007$)	Model 5[6] OR (95% CI) ($n = 850$)	Model 6[7] OR (95% CI) ($n = 495$)
25(OH)D < 20 ng/mL	1.76 (0.76, 4.07)	—	—	—	—	—
25(OH)D, ng/mL						
≥30	—	1.0 (ref)	—	—	—	—
20 to <30	—	0.48 (0.18, 1.25)	—	—	—	—
<20	—	1.11 (0.42, 2.91)	—	—	—	—
PTH > 65 pg/mL	—	—	0.67 (0.10, 4.47)	—	—	—
Total calcium (1-unit increase)	—	—	—	0.57 (0.24, 1.38)	—	—
Femoral neck BMD (1-unit decrease)	—	—	—	—	0.09 (0.01, 2.86)	—
Total spine BMD (1-unit decrease)	—	—	—	—	—	0.96 (0.04, 25.00)
Female gender	0.24 (0.08, 0.70)	0.22 (0.08, 0.63)	0.16 (0.04, 0.67)	0.27 (0.09, 0.86)	0.31 (0.08, 1.16)	0.24 (0.07, 0.87)
Age	1.19 (1.08, 1.32)	1.19 (1.07, 1.31)	1.19 (1.06, 1.34)	1.18 (1.07, 1.30)	1.18 (1.06, 1.32)	1.13 (0.97, 1.33)
Race/ethnicity						
Non-Hispanic white	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)
Non-Hispanic black	0.41 (0.16, 1.10)	0.44 (0.16, 1.21)	1.68 (0.37, 7.59)	0.49 (0.18, 1.32)	0.37 (0.13, 1.07)	0.48 (0.16, 1.47)
Other	1.06 (0.36, 3.12)	1.14 (0.40, 3.28)	3.10 (0.40, 23.89)	1.06 (0.38, 3.00)	1.29 (0.43, 3.90)	5.54 (0.64, 47.76)
Prefrail/frail	0.62 (0.20, 1.91)	0.63 (0.21, 1.91)	0.83 (0.18, 3.79)	0.67 (0.22, 2.02)	0.71 (0.23, 2.20)	0.56 (0.15, 2.05)
Supplement use	0.57 (0.24, 1.38)	0.50 (0.21, 1.19)	0.55 (0.16, 1.93)	0.51 (0.21, 1.22)	0.38 (0.13, 1.11)	0.39 (0.11, 1.33)
Creatinine	1.39 (0.39, 4.88)	1.42 (0.36, 5.67)	2.33 (0.22, 24.57)	1.47 (0.44, 4.89)	1.77 (0.32, 9.92)	1.12 (0.25, 5.11)

[1]BMD, bone mineral density; PTH, parathyroid hormone; 25(OH)D, 25-hydroxyvitamin D; GED, General Educational Development; AA, Associate in Arts.
[2]Logistic regression model containing vitamin D status as a binary variable and variables with $P < 0.10$ in bivariate analyses.
[3]Logistic regression model containing vitamin D status as a categorical variable and variables with $P < 0.10$ in bivariate analyses.
[4]Logistic regression model containing PTH status as a binary variable and variables with $P < 0.10$ in bivariate analyses.
[5]Logistic regression model containing total calcium and variables with $P < 0.10$ in bivariate analyses.
[6]Logistic regression model containing femoral neck BMD and variables with $P < 0.10$ in bivariate analyses.
[7]Logistic regression model containing total spine BMD and variables with $P < 0.10$ in bivariate analyses.

The mechanism by which vitamin D deficiency may cause HL is likely through its role as a key regulator of calcium absorption and bone metabolism. Vitamin D may affect calcium homeostasis locally within the inner ear, where calcium ions play an important role in the conduction of electrical impulses between nerve fibers and hair cell transduction (19, 20). Although we did not find an association between systemic calcium concentrations and HL, the majority of the study sample had systemic calcium concentrations within the normal range (Table 2), because calcium concentrations are tightly regulated. This lack of variability may have contributed to our null finding. In addition, small differences in systemic calcium concentrations may not reflect local calcium concentrations within the inner ear, where calcium status may carry more significant implications for HL. We also did not find an association between PTH and HL, although PTH concentrations were only available for the 2005/2006 cycle, which limited the sample size.

In an early case series, vitamin D deficiency was present in 10 cases of bilateral cochlear deafness (21). It was postulated that localized demineralization of the cochlea leading to structural changes of the otic capsule most likely played an important role (21). In this study, femoral neck and total spine BMD were used as a proxy for temporal bone BMD, based on the assumption that osteoporosis is a systemic disease. We found that a 1-unit decrease in femoral neck BMD and a 1-unit decrease in total spine BMD were both associated with greater odds of low-frequency but not speech-frequency or high-frequency HL. Studies linking femoral or spine BMD to HL have been primarily conducted in postmenopausal women (10–15). Helzner et al. (16), who investigated the relation between BMD and HL in patients over the age of 70 y, failed to find an association, except among black men. Studies investigating the relation between BMD and HL, including this one, are limited by the lack of data on BMD of the temporal bone and cochlear capsule. One study found that bone quality around the inner ear was poorly correlated to the BMD of the central skeleton (33). Nevertheless, our results indicate that BMD may play some role in low-frequency HL in this population.

Furthermore, there is molecular evidence suggesting that changes in mechanical properties of the cochlear bone, without other structural changes in the sensory neuroepithelia, may lead to SNHL. Mice overproducing transforming growth factor β (TGFβ) have SNHL, which is thought to be due to reduction in elastic modulus and hardness of the cochlear bones compared with wildtype mice; quite remarkably, with a reduction in production of TGFβ, the bone and hearing return to normal (34). Interactions are thought to occur between TGFβ and vitamin D in their regulation of osteoblasts (35, 36). The influence of vitamin D on bone metabolism may be a second mechanism by which vitamin D deficiency affects HL.

Vitamin D also has important roles in the regulation of vascular cell function, angiogenesis, and inflammation (37–39). Vitamin D is an inhibitor of angiogenesis, and vitamin D deficiency has been implicated in various ocular diseases. Vitamin D also regulates the immune system by reducing the expression of

proinflammatory cytokines and increasing the expression of anti-inflammatory cytokines (38, 40, 41). Inflammation has been hypothesized as one of the mechanisms driving obesity-induced HL which, like vitamin D deficiency, also primarily affects the low frequency (42, 43). Immunomodulatory properties and vascular effects are other mechanisms by which vitamin D deficiency may cause HL.

Overall, the strengths of this study include its utilization of a large, nationally representative sample, enabling adjustment for age, gender, education level, and many possible confounders, including creatinine, which has previously been linked to HL (44). The availability of audiometry data for elderly adults in whom HL is prevalent is also a strength, because self-report of HL can result in underestimation (45). However, our ability to detect any association between the main exposures of interest and high-frequency HL was hindered by the high prevalence of the outcome in this population of elderly adults. Other limitations include the cross-sectional design, precluding the determination of the temporal relation between exposure and outcome and limiting our ability to make any conclusions about causality, and the lack of available data for certain variables of interest, such as vitamin B-12 and season of 25(OH)D measurement, which may affect HL or vitamin D concentration. Nevertheless, it is more plausible that low vitamin D status causes HL, rather than the reverse. Finally, the proposed mechanisms by which low vitamin D status may cause HL, through altering calcium homeostasis within the inner ear and through demineralization of the cochlea, would lead to SNHL. In this study, SNHL was inferred based on normal otoscopy and adequate- or good-quality tympanograms with peaks >0.3 mL. It is possible that some individuals included in the study may have had conductive HL instead of SNHL.

In the elderly, low vitamin D status, but not PTH or total calcium, was found to be associated with HL. Decreases in BMD were found to be associated with greater odds of low-frequency HL. Our results contribute to the small body of literature linking low vitamin D concentrations and low BMD to HL.

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The authors' responsibilities were as follows—BS and AKL: designed the research, analyzed the data, and had primary responsibility for the final content; and all authors: wrote the paper and read and approved the final manuscript. AKL serves on the Medical Advisory Board for Advanced Bionics and on the Surgical Advisory Board for MED-EL. All other authors report no conflicts of interest.

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