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Cognitive, Health, and Sociodemographic Predictors of Longitudinal Decline in Hearing Acuity Among Older Adults

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Background. We aimed to investigate predictors of change in pure-tone hearing thresholds in older adults.

Methods. Data were drawn from a pooled sample from the Dynamic Analyses to Optimise Ageing (DYNOPTA) project (N = 4,221, mean age = 73.6, range: 50–103 years). Pure-tone hearing thresholds were tested for frequencies between 0.5 and 8 kHz, on up to four occasions over a period of 11 years. Linear mixed models tested for predictors of change in hearing.

Results. Hearing loss for high-range frequencies preceded decline in low-range frequencies. Men had higher baseline hearing thresholds, but women experienced faster rates of decline in hearing for mid- to high-range frequencies. The estimated rate of change for a 75-year-old adult was 0.91 decibel hearing level (dB HL) per year for pure-tone thresholds averaged over frequencies ranging between 0.5 and 4 kHz in the better ear. Baseline age ($\beta = 0.03$, $p < .01$), hypertension ($\beta = 0.15$, $p < .01$), and probable cognitive impairment ($\beta = 0.40$, $p = .01$) were independent predictors of annual rate of change in hearing thresholds. Incidence of probable cognitive impairment was also associated with higher hearing thresholds. Other known correlates for prevalence of hearing impairment, including low education, noise damage, diabetes, and history of stroke were independently associated with baseline levels of hearing but were not predictive of change in hearing thresholds.

Conclusions. Faster rates of decline in hearing are predicted by probable cognitive impairment and hypertension.

Key Words: Presbycusis—Age-related hearing loss—Cognitive impairment—The Australian Longitudinal Study of Ageing—The Blue Mountains Eye Study.

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AGE-RELATED hearing loss is highly prevalent among older adults (1–4). It features among the leading causes of years lived with disability and is considered a substantial contributor to global burden of disease (5). Cross-sectional studies have identified diabetes (6,7), cardiovascular disease, hypertension, and blood pressure (8) as risk factors for hearing loss. Hearing loss has also been linked with poor physical and mental health, falls (9), mortality (10,11), and lower cognitive functioning or dementia (12–19). However, longitudinal analyses have failed to show an association between many of these risk factors with incidence of age-related hearing loss (20–22).

Divergent patterns of predictors for prevalence versus rates of decline in hearing have been suggested to arise from methodological factors. These include insufficient statistical power, differences in the rate of onset, and age dependency of hearing loss (20). Alternatively, the common practice of dividing ranges of averaged hearing thresholds into conventional categories of hearing loss (eg, no impairment, mild impairment, moderate impairment) may obscure true associations between risk factors for change in hearing acuity. We address these issues by employing growth curve techniques to examine hearing trajectories in a larger representative sample of older adults than has previously been available. Other studies investigating longitudinal changes in continuous measures have primarily focused on mapping age and sex trajectories of individual pure-tone frequencies (23–27). This study aims to extend the current understanding of age-related hearing loss by additionally investigating sociodemographic and health-related risk factors for change in hearing thresholds.

Methods

Participants

Data were drawn from the Australian Longitudinal Study of Ageing (ALSA) (28) and the Blue Mountains Eye Study

(BMES) (12) as part of the Dynamic Analyses to Optimise Ageing (DYNOPTA) project (29). ALSA drew a random sample of adults aged 70 years and older from the electoral role for the Adelaide metropolitan area of South Australia. ALSA oversampled men aged 85 years and older and also recruited spouses aged 65 years and older, or others over 70 years who were cohabiting with the sampled participant. Data collection pertinent to the aims of this study occurred within ALSA at wave 1 (1992), wave 3 (1994), wave 6 (2000–2001), and wave 7 (2003–2004). BMES attempted to recruit all adults aged 49 years and older from two postcodes in Blue Mountains region west of Sydney, Australia. Data collection pertinent to the aims of this study occurred within BMES occurred at wave 2 (1997–1999) and wave 3 (2002–2004). We define the baseline sample as pooled data from wave 1 of ALSA and wave 2 of BMES.

Measures

Audiometric testing was conducted by a trained interviewer in each study. Hearing loss was assessed by uncorrected pure-tone thresholds in each ear at frequencies of 0.5, 1, 2, 3, 4, 6, and 8 kHz using calibrated portable audiometers for ALSA participants and in a sound-treated booth for BMES participants. Outcome variables used in analyses reported in this study were pure-tone thresholds in the better ear and a pure-tone average (PTA) of low- to mid-range frequencies important for speech perception (0.5, 1, 2, and 4 kHz) in the better ear. Thresholds ranged between 0 and 120 decibel hearing level (dB HL), thresholds of 120 dB HL were treated as outliers and coded as missing values. In BMES, frequencies of 3 kHz were only tested in participants with a difference of 20 dB HL between the 2 and 4 kHz frequencies.

Medical conditions were obtained by self-report of clinician diagnoses and included: diabetes, hypertension, history of stroke, and history of heart attack. Corrected visual acuity was tested with a logMAR chart at a distance of 3 m, with visual impairment defined by values greater than 0.3 logMAR. A score of 23 or less on the Mini-Mental State Examination (30) was used as an indicator of probable cognitive impairment. Smoking status was also obtained by self-report.

Information on workplace noise exposure was collected in ALSA with the question "Have you ever worked in a noisy environment where you had to shout to be heard?" and in BMES with the question "Have you ever worked in a noisy industry or noisy farm environment?" To identify cases with likely noise induced hearing loss, high-frequency audiometric noise notches were defined using the criteria described by Coles and colleagues (31). These criteria have been shown to have strong agreement with expert consensus (32).

Analysis

For descriptive purposes, the mean and standard deviation $PTA_{0.5,\,1,\,2,\,4\,kHz}$ were calculated for 10-year age groups and for each covariate. Linear mixed models were used to estimate trajectories of hearing thresholds in the better ear. All analyses included random effect variance components for the intercept and slope (time) with an unstructured covariance matrix. The optimal scaling of time was ascertained by comparing Bayesian Information Criterion (BIC) values for models that indexed time as linear and quadratic functions of age, with models that indexed time over a "years in study" metric adjusting for age at baseline with an interaction term between age at baseline and years in study. Better model fit is indicated by lower BIC values. Age and sex trajectories of hearing thresholds in the better ear were then estimated for each tone frequency and $PTA_{0.5,\,1,\,2,\,4\,kHz}$. Model coefficients were used to graph the mean trajectories for men and women aged 60, 75, and 90 years at baseline. The predicted mean ages at which the $PTA_{0.5,\,1,\,2,\,4\,kHz}$ trajectory crossed thresholds of 25 and 40 dB HL were estimated for men and women by solving the model equation for "time."

Interaction terms between baseline predictors and time tested between-person differences in hearing trajectories. We included baseline predictors of age (mean centered to 75 years), sex (female = 1), and indicators of probable cognitive impairment, diabetes, stroke, hypertension, visual impairment, and smoking status. Time invariant predictors were workplace noise exposure, high-frequency audiometric noise notches, and sociodemographics. For those baseline conditions that were significantly associated with change in hearing thresholds, we also included an indicator of post-baseline incidence to test if incident medical conditions were also associated with hearing loss. A four-stage procedure was employed to evaluate predictors of change in $PTA_{0.5,\,1,\,2,\,4\,kHz}$. In the first stage, we conducted a series of univariate models that estimated unadjusted associations between each predictor variable with baseline hearing levels and longitudinal hearing trajectories. In the second step, we ran the same set of univariate models adjusting for age at baseline. We then estimated a full multivariate model that included all covariates. In the final step, BIC were used to evaluate the multivariate model, which was refined by excluding model terms that did not contribute to the overall model fit. In order to determine the extent to which noise damage confounded inferences concerning age-related hearing loss, multivariate analyses were repeated excluding all participants who reported 5 years of workplace-related noise exposure or were identified to have high-frequency noise notches. All analyses were conducted using Stata version 10 (33).

Results

Description of Sample Characteristics

The baseline sample profile is described in Table 1. The pooled sample comprised 4,221 participants (46.3% men) with a mean age of 73.6 years (*SD* = 8.9, range = 50–103). A total of 366 participants were classified with probable cognitive impairment at baseline, with a further 274 incident

Table 1. Baseline Sample Profile, 3,526 Australian Adults Aged 50 and Older

			PTA (dB)
	N	%	Mean (*SD*)
Sex			
Men	1,633	46.3	30.6 (15.7)
Women	1,893	53.7	26.0 (14.7)
Age (y)			
50–59	285	8.1	15.2 (11.3)
60–69	861	24.4	20.8 (13.4)
70–79	1,562	44.3	28.7 (13.1)
80–89	750	21.3	38.5 (14.2)
90+	68	1.9	46.6 (17.0)
Hearing loss			
Normal	1,718	48.7	16.0 (5.9)
Mild	1,140	32.3	32.4 (4.2)
Moderate–severe	668	18.9	52.0 (11.8)
Qualification			
Secondary only	1,647	46.7	29.7 (15.2)
Postsecondary	1,442	40.9	26.5 (15.1)
Tertiary	242	6.9	25.9 (14.2)
Occupation			
Tradesperson	440	12.5	32.9 (17.1)
Plant, machine operators, and drivers	129	3.7	31.6 (16.1)
Laborers and related workers	231	6.6	31.7 (16.1)
Other	2,726	77.3	26.9 (14.7)
Smoking status			
Never	1,741	49.4	27.7 (15.7)
Former	1,458	41.3	28.8 (14.8)
Current	291	8.3	26.9 (15.7)
Workplace noise exposure			
<1 y	2,339	66.3	27.1 (14.8)
1–5 y	323	9.2	29.5 (16.0)
5+ y	864	24.5	30.6 (16.1)
Hearing aid			
Yes	401	11.4	49.7 (15.1)
Hearing restricts social life			
Never	2,143	60.8	25.2 (13.9)
Sometimes	431	12.2	38.3 (14.4)
Often	209	5.9	47.3 (20.1)
Medical conditions (self-report)			
Diabetes	252	7.1	31.4 (16.6)
Stroke	151	4.3	32.4 (16.3)
Heart attack	353	10.0	31.3 (15.3)
Hypertension	1,234	35.0	27.4 (14.7)
Any circulatory condition	1,729	49.0	29.0 (15.3)
Measured conditions			
Systolic > 145 mmHg	2,334	66.2	28.3 (15.2)
Diastolic > 95 mmHg	428	12.1	26.1 (15.2)
Visual acuity > 0.3 logMAR	507	14.4	35.0 (15.9)
MMSE < 24	218	6.2	38.9 (16.7)

Note: logMAR = logarithm of the minimum angle of resolution; MMSE = Mini-Mental State Examination; PTA = Pure-tone average (dB) of 0.5, 1, 2, 4 kHz in the better ear. Column percentages are based on the number of participants who gave a valid response, rows may not sum to whole sample due to missing data.

cases in subsequent waves. There were 211 participants identified with high-frequency audiometric noise notches at any time (mean baseline age = 69.9, 75.4% men), and 851 participants reported workplace-related noise exposure for 5 or more years.

The average time intervals between successive waves were 3.8 (*SD* = 1.8), 6.1 (*SD* = 0.2), and 3.1 (*SD* = 0.2) years, with participants providing an average of 2 waves of data. Prior to the commencement of wave 2, 16.6% of participants were lost to attrition and a further 6.4% were deceased. The BMES sample (n = 2,334) only provided data for waves 1 and 2. Within the ALSA sample, 44.5% of baseline participants were deceased at wave 3, this increased to 58.8% at wave 4.

Audiometric testing was completed by 3,526 participants at baseline ($PTA_{0.5,\,1,\,2,\,4\,kHz}$ Mean (M) =28.2 dB, SD = 15.2) and 3,011 participants at wave 2 (M = 30.1 dB, SD = 15.5). Based on the ALSA sample, $PTA_{0.5,\,1,\,2,\,4\,kHz}$ data were available for 525 participants at wave 3 (M = 37.0 dB, SD = 14.3) and 391 participants at wave 4 (M = 38.6 dB, SD = 15.3).

Modeling of Time

Linear mixed models that indexed time over a years in study metric and adjusted for baseline age (BIC = 54,272.0) provided a better description of longitudinal change in $PTA_{0.5,\,1,\,2,\,4\,kHz}$ and were preferable to models that indexed time using an "age" metric (BIC = 55,195.9). This was consistent with previous recommendations regarding the optimal scaling of time in longitudinal analyses with broad age cohorts (34). All subsequent results index time over a years in study metric.

Trajectories of Hearing Thresholds for Men and Women

The estimated age-related trajectories for each of the seven pure-tone frequencies and $PTA_{0.5,\,1,\,2,\,4\,kHz}$ in men and women are presented in Figure 1. An increase in hearing thresholds over time indicates a decline in hearing acuity. Relative to high-range frequencies, change in hearing thresholds for low-range frequencies began later and accelerated with age. Age-related changes in frequencies greater than 4 kHz were observed for adults of all ages, whereas frequencies of 0.5 and 1 kHz did not show marked increases in pure-tone thresholds until individuals were aged in their 70s. There were no sex differences in rate of change in hearing for $PTA_{0.5,\,1,\,2,\,4\,kHz}$ and low-range frequencies. However, women had lower intercepts and faster increases in thresholds greater than 3 kHz. Sex differences in intercepts and slopes were greatest for mid-range frequencies. For adults aged 75 years at baseline, the estimated mean $PTA_{0.5,\,1,\,2,\,4\,kHz}$ trajectory crossed a threshold of 25 DB HL (often defined as mild hearing impairment) at ages 67.8 years for men and 71.1 years for women. The estimated mean $PTA_{0.5,\,1,\,2,\,4\,kHz}$ trajectory crossed a threshold of 40 dB HL (often defined as moderate hearing impairment) at ages 83.2 years for men and 86.5 years for women.

Predictors of Hearing Loss

Table 2 shows the results from the series of univariate-, age-, and multivariate-adjusted linear mixed models for $PTA_{0.5,\,1,\,2,\,4\,kHz}$ in the better ear. In the age-adjusted univariate models, all baseline covariates reliably predicted initial

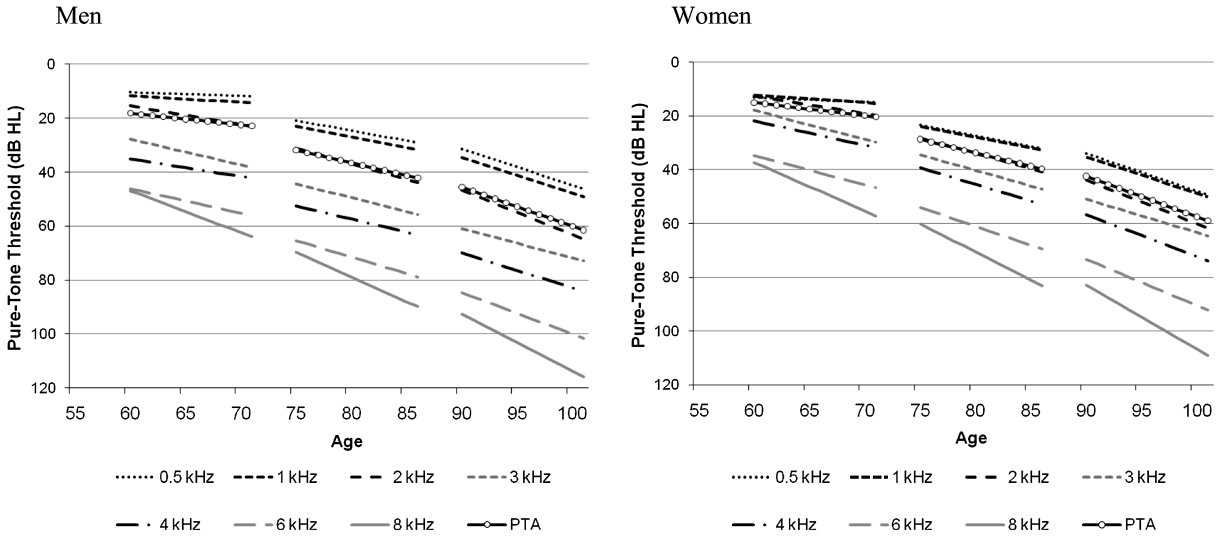

Figure 1. Unadjusted 11-year trajectories of pure-tone thresholds decibel hearing level (dB HL) for frequencies of 0.5, 1, 2, 3, 4, 6, and 8 kHz in the better ear, and $PTA_{0.5,\,1,\,2,\,4\,kHz}$ in the better ear, estimated for three cohorts of men (left panel) and women (right panel) aged 60, 75, and 90 years at baseline. The y-axis has been reversed so a negative gradient indicates a decline in hearing performance. Sample excludes participants with high-frequency noise notches. The better ear was defined by $PTA_{0.5,\,1,\,2,\,4\,kHz}$.

levels of $PTA_{0.5,\,1,\,2,\,4\,kHz}$. However, the only statistically significant predictors of rate of change were baseline age, sex, workplace noise exposure, and probable cognitive impairment. Faster increases in hearing thresholds were observed for older adults, women, and participants with probable cognitive impairment. Interestingly, noise notches were not associated with hearing trajectories, but participants reporting 5 years or more of workplace noise exposure showed slower increases in hearing thresholds.

In multivariate analyses, smoking, visual impairment, and postsecondary nontertiary qualifications did not contribute to overall model fit and were excluded from the final model. For an adult aged 75 years, the average $PTA_{0.5,\,1,\,2,\,4\,kHz}$ trajectory increased at a rate of 0.86 dB HL per annum, with annual increase in the rate of change of 0.03 dB HL. After adjusting for sociodemographic and health variables, there were no sex differences in rate of change in hearing, though probable cognitive impairment at baseline was associated with both poorer initial $PTA_{0.5,\,1,\,2,\,4\,kHz}$ levels (β_{level} = 3.91, 95% confidence interval [CI] = 2.05–5.77) and faster rates of change in $PTA_{0.5,\,1,\,2,\,4\,kHz}$ (β_{change} = 0.40, 95% CI = 0.12–0.68). Incident probable cognitive impairment was also associated higher $PTA_{0.5,\,1,\,2,\,4\,kHz}$ ($\beta_{incident}$ = 0.83, 95% CI = 0.12–1.55). Probable cognitive impairment at baseline was not associated with change in better ear thresholds for individual frequencies greater than 4 kHz. Multivariate analyses also revealed greater rates of change in thresholds for participants reporting clinically diagnosed hypertension at baseline (β_{change} = 0.15, 95% CI = 0.06–0.25). Excluding participants who reported 5 years or more of workplace noise exposure or who had high-frequency noise notches, resulted in only minor adjustments to model coefficients and the substantive findings remained unchanged (data not shown).

Discussion

This study reports on patterns and predictors of change in 11-year trajectories for hearing thresholds in older adults. Hearing loss for frequencies important for speech perception increased at an average rate of 0.91 dB/year. Unsurprisingly, these rates of hearing decline were accelerated for older ages. Half of all adults in the oldest old cohort, aged 85 years and older, had moderate hearing loss, and almost all of the oldest old cohort could be expected to have at least a mild degree of hearing loss. A key finding is that cognitive impairment was independently associated with lower levels and accelerated declines in peripheral hearing ability. Furthermore, incidence of cognitive impairment was also associated with poorer hearing. Thus, both between-person differences and within-person change in cognitive function were identified as risk factors for hearing loss. Hypertension was also found to be predictive of greater decline rates in hearing.

This study adds to the growing literature linking poor hearing with neurocognitive disorders (13–18) and age-related cognitive decline (19). Early hearing loss and rapid hearing decline have been suggested to be precursors of dementia and could be useful risk markers in dementia diagnosis (16,18), though the analyses presented here do not test this hypothesis. Rather than assessing hearing loss as a leading indicator of cognitive decline, we show that individuals with cognitive impairment experience faster declines in peripheral hearing. That cognitive impairment was not predictive of decline in high-frequency thresholds suggests underlying mechanistic pathways. However, the mechanism for this is unclear and cannot be identified from this study. The co-occurrence of cognitive impairment and hearing loss should be expected due to their associations with aging, but further explanation is warranted because their association

Table 2. Fixed Effects for Predictors of Baseline Levels and Longitudinal Trajectories of Hearing Thresholds ($PTA_{0.5, 1, 2, 4\ kHz}$) in the Better Ear Estimated From Univariate and Multivariate Linear Mixed Models

	Univariate Models		Age-Adjusted Models		Multivariate (full model)		Multivariate (final model)	
	β (SE)	*p*	β (SE)	*p*	β (SE)	*p*	β (SE)	*p*
Unadjusted								
Intercept (baseline)	27.91 (0.26)	<.01	29.92 (0.23)	<.01	27.82 (0.96)	<.01	28.69 (0.48)	<.01
Time (y)	0.84 (0.03)	<.01	0.97 (0.03)	<.01	0.89 (0.10)	<.01	0.86 (0.03)	<.01
Demographics								
$Age_{baseline}$*	0.91 (0.03)	<.01	0.91 (0.03)	<.01	0.87 (0.03)	<.01	0.89 (0.03)	<.01
$Age_{baseline}$ by time	0.03 (0.00)	<.01	0.03 (0.00)	<.01	0.03 (0.00)	<.01	0.03 (0.00)	<.01
Women	−4.64 (0.51)	<.01	−3.32 (0.44)	<.01	−2.04 (0.54)	<.01	−1.54 (0.48)	<.01
Women by time	0.09 (0.05)	0.07	0.12 (0.05)	.02	0.10 (0.05)	0.08	Dropped from model	
Cognitive Status								
$MMSE < 24_{baseline}$	11.75 (1.04)	<.01	5.16 (0.92)	<.01	3.34 (1.02)	<.01	3.91 (0.95)	<.01
$MMSE < 24_{baseline}$ by time	0.54 (0.15)	<.01	0.37 (0.14)	.01	0.47 (0.15)	<.01	0.40 (0.14)	.01
$MMSE < 24_{incidence}$	1.55 (0.36)	<.01	0.93 (0.36)	.01	0.87 (0.39)	.03	0.83 (0.36)	.02
Qualifications								
Secondary only	3.91 (1.04)	<.01	2.14 (0.90)	.02	2.37 (0.93)	.01	1.08 (0.45)	.02
Secondary only by time	0.05 (0.10)	.64	−0.05 (0.10)	.61	−0.12 (0.09)	.20	Dropped from model	
Postsecondary	0.87 (1.05)	.41	1.35 (0.90)	.14	1.23 (0.92)	.18	Dropped from model	
Postsecondary by time	−0.02 (0.10)	.83	−0.04 (0.10)	.68	−0.08 (0.09)	.42	Dropped from model	
Smoking Status								
Former smoker	1.19 (0.54)	.03	0.89 (0.46)	.05	−0.45 (0.51)	.38	Dropped from model	
Former smoker by time	−0.04 (0.05)	.41	−0.05 (0.05)	.31	0.03 (0.05)	.62	Dropped from model	
Current smoker	−1.03 (0.97)	.29	2.07 (0.83)	.01	0.24 (0.88)	.79	Dropped from model	
Current smoker by time	−0.14 (0.10)	.17	−0.03 (0.10)	.79	0.11 (0.10)	.24	Dropped from model	
Workplace noise exposure								
5 y or more	3.51 (0.61)	<.01	4.96 (0.51)	<.01	3.80 (0.59)	<.01	3.97 (0.57)	<.01
5 y or more by time	−0.23 (0.06)	<.01	−0.18 (0.06)	<.01	−0.07 (0.06)	.27	−0.13 (0.05)	.01
1–5 y	2.49 (0.90)	.01	3.88 (0.76)	<.01	3.48 (0.83)	<.01	3.27 (0.78)	<.01
1–5 y by time	−0.01 (0.09)	.87	0.01 (0.09)	.90	<.01 (0.08)	.97	Dropped from model	
Noise notch								
Notch	1.29 (0.59)	.03	1.61 (0.59)	.01	0.78 (0.57)	.17	1.24 (0.49)	.01
Notch by time	−0.01 (0.18)	.97	−0.01 (0.17)	.94	−0.04 (0.17)	.80	Dropped from model	
Medical conditions								
Hypertension	−1.38 (0.54)	.01	−0.93 (0.46)	.04	−0.77 (0.49)	.11	−0.79 (0.47)	.09
Hypertension by time	0.11 (0.05)	.04	0.10 (0.05)	.06	0.14 (0.05)	<.01	0.15 (0.05)	<.01
Diabetes	3.14 (1.01)	<.01	3.06 (0.86)	<.01	2.76 (1.14)	.02	2.09 (0.85)	.01
Diabetes by time	−0.09 (0.11)	.43	−0.06 (0.11)	.54	−0.23 (0.14)	.11	Dropped from model	
Stroke	4.67 (1.29)	<.01	3.28 (1.10)	<.01	2.66 (0.90)	<.01	2.56 (1.10)	.02
Stroke by time	−0.19 (0.16)	.22	−0.16 (0.15)	.29	−0.06 (0.10)	.56	Dropped from model	
Visual impairment	8.66 (0.72)	<.01	2.04 (0.66)	<.01	1.31 (0.67)	.05	Dropped from model	
Visual impairment by time	0.13 (0.08)	.08	−0.04 (0.08)	.59	−0.10 (0.07)	.15	Dropped from model	

Notes: $MMSE < 24_{baseline}$ = baseline probable cognitive impairment; $MMSE < 24_{incidence}$ = incidence of probable cognitive impairment post-baseline. Random effects for intercept and slope are not shown. Reference group for each variable: Men: No cognitive impairment, tertiary qualified, never smoker, less than 1 year noise exposure, absent noise notch, no reported hypertension, no reported diabetes, no reported stroke, and no visual impairment.

*$Age_{baseline}$ is centered to 75 years.

remains after statistically controlling for the effects of age. A third variable not properly adjusted for in this study, such as cerebral microangiopathy, is the most likely explanation for the association between cognition and hearing decline. As dementia pathology is not believed to affect the inner ear or cochlea (35), the current findings could simply be accounted for by top-down processing effects and reflect a more cautious or impaired decision-making process regarding tone perception judgments. Older adults, particularly those with poor executive functioning, may show a response bias whereby greater certainty is required before they acknowledge an audible tone. To a lesser extent, these findings could partially be explained by difficulties experienced by people with sensory loss when completing standard neuropsychological assessments. However, such explanations can generally be discounted as it is possible to conduct audiometric testing in young children, and trained clinical interviewers should be sensitive to hearing limitations of study participants (16).

A combination of histological, electrophysical, and molecular mechanisms in both the peripheral and central nervous system underlie hearing loss (36). It is likely that any biological mechanism underlying a link between dementia and hearing loss occurs centrally upstream of the cochlea (18). For example, Alzheimer's disease pathology has been observed in auditory system pathways such as the ventral

nucleus of the medial geniculate body and in the auditory cortex, but not in cochlear nuclei (35). As unaided pure-tone thresholds were used in this study, we are unable to draw direct inferences about the association between cognitive function and central auditory processing. Our understanding of the temporal interrelations between hearing and cognition would be improved by longitudinal analyses of specific cognitive domains, hearing thresholds, and hearing measures that better asses central presbycusis and neural loss, such as dichotic listening or synthetic sentence identification tasks (2).

Our results support previous findings where risk factors for prevalence of hearing loss, including smoking, diabetes, and stroke (20–22), were not found to be predictive of incidence of hearing loss. Even cross-sectional associations between these factors and hearing loss remain in question. Recent analyses of 717 older adults in the National Health and Nutritional Examination Survey (4) failed to find independent associations between low-frequency, speech-frequency, or high-frequency thresholds with the same set of risk factors, regardless of whether thresholds were modeled as continuous or binary outcomes. This contrasts with our findings, as both diabetes and stroke were cross-sectionally associated with poor baseline hearing. These inconsistencies could arise from methodological differences and the larger sample available in DYNOPTA. Lin and colleagues (4) also speculate that smoking, diabetes, and other cardiovascular risk factors may only have weak associations with hearing loss that are mediated or obscured by other factors. It is therefore intriguing to note the opposite pattern of results for hypertension, which was not predictive of baseline hearing levels but was a risk factor for change. The relation between hypertension and hearing loss is uncertain. Although some researchers have identified hypertension as being linked with hearing loss (2), in particular systolic blood pressure (37), this was not the case in the National Health and Nutritional Examination Survey (4). This deserves further investigation.

Age-related declines in sensory functioning have multiple etiologies, ranging from genetic factors (38) to environmental exposures (36,39), but it has been argued recently that between-person differences in audiometric hearing thresholds can be primarily attributed to genetic variation (40). If so, then this may explain why there has been a failure to show an association between changes in hearing performance with many of the known risk factors for poor hearing. The inability to identify predictors for change in hearing and the equivocal cross-sectional findings suggest that rate of hearing decline may be a better indicator of putative normative or primary ageing processes and less influenced by disease than other functions. If higher intercepts reflect earlier onset of decline, this could indicate that hearing loss may begin at earlier ages for individuals with poor health, but the rate of hearing loss remains stable for most groups, with the exception of individuals with cognitive impairment or hypertension.

Paradoxically, there was no evidence of a relation between audiograms indicative of noise damage with hearing trajectories, yet noise exposure was predictive of more gradual declines in hearing. This is not completely inconsistent with a previous study that demonstrated slower hearing change for frequencies between 3 and 6 kHz, yet accelerated change for adjacent frequencies of 2 and 8 kHz, among individuals with noise notches (41). These findings were based on a younger sample of men and a different methodology to that employed in the current study. Our failure to identify high-frequency noise notches as a risk factor for change could be due to the difficulty in reliably identifying notches in older adults, particularly for ages when noise-induced hearing loss becomes concomitant with age-related hearing loss (31).

Our results are consistent with existing knowledge about the general progression of age-related hearing loss (2). Typically, age-related hearing loss begins with loss of the ability to perceive high frequencies, then gradually extends to low-range frequencies. High-frequency hearing loss has previously been reported to begin during the 50s (23), so it is likely that decline for high frequencies began before study commencement. Although men had poorer hearing levels for mid- and high-range frequencies, women experienced faster rates of hearing decline for these ranges. The lower initial levels for men probably reflect an earlier age onset of hearing loss.

Differential patterns of hearing loss occur across a spectrum of tone frequencies, which can be either independent of or related to age (42). Due to the time intervals between hearing measurements, we lacked the data to detect rapid declines that occurred independently of age effects over a short time frame. At least four distinct types of presbycusis have been classified, each characterized by a unique pattern of change (36,43), which we were also unable to investigate here. This study has not included ototoxic agents (3,36), and genetic data were not available. We also lacked clinical diagnoses of dementia. These caveats notwithstanding, ours is the largest data set to assess the predictors of hearing loss.

In summary, this study contributes to existing knowledge of the association between impaired cognitive function and hypertension with accelerated decline in hearing. Our findings highlight the need for researchers and clinicians to be aware of impaired cognitive functioning when assessing hearing performance, and conversely, of hearing limitations when diagnosing, screening, and managing individuals with dementia or other cognitive impairments. With the projected rise in the age-adjusted prevalence of hearing loss, its relation to health, well-being, and longevity, there is a need for greater awareness and a better understanding of the development of age-related hearing loss and its interaction with comorbid chronic health conditions.

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