Hearing Loss and Cognitive Decline in Older Adults

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Background: Whether hearing loss is independently associated with accelerated cognitive decline in older adults is unknown.

Methods: We studied 1984 older adults (mean age, 77.4 years) enrolled in the Health ABC Study, a prospective observational study begun in 1997-1998. Our baseline cohort consisted of participants without prevalent cognitive impairment (Modified Mini-Mental State Examination [3MS] score, ≥80) who underwent audiometric testing in year 5. Participants were followed up for 6 years. Hearing was defined at baseline using a pure-tone average of thresholds at 0.5 to 4 kHz in the better-hearing ear. Cognitive testing was performed in years 5, 8, 10, and 11 and consisted of the 3MS (measuring global function) and the Digit Symbol Substitution test (measuring executive function). Incident cognitive impairment was defined as a 3MS score of less than 80 or a decline in 3MS score of more than 5 points from baseline. Mixed-effects regression and Cox proportional hazards regression models were adjusted for demographic and cardiovascular risk factors.

Results: In total, 1162 individuals with baseline hearing loss (pure-tone average >25 dB) had annual rates of decline in 3MS and Digit Symbol Substitution test scores that were 41% and 32% greater, respectively, than those among individuals with normal hearing. On the 3MS, the annual score changes were −0.65 (95% CI, −0.73 to −0.56) vs −0.46 (95% CI, −0.55 to −0.36) points per year ($P = .004$). On the Digit Symbol Substitution test, the annual score changes were −0.83 (95% CI, −0.94 to −0.73) vs −0.63 (95% CI, −0.75 to −0.51) points per year ($P = .02$). Compared to those with normal hearing, individuals with hearing loss at baseline had a 24% (hazard ratio, 1.24; 95% CI, 1.05-1.48) increased risk for incident cognitive impairment. Rates of cognitive decline and the risk for incident cognitive impairment were linearly associated with the severity of an individual's baseline hearing loss.

Conclusions: Hearing loss is independently associated with accelerated cognitive decline and incident cognitive impairment in community-dwelling older adults. Further studies are needed to investigate what the mechanistic basis of this association is and whether hearing rehabilitative interventions could affect cognitive decline.

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THE PREVALENCE OF DEMENTIA is projected to double every 20 years because of the aging of the world population.[1] Therefore, identifying factors and understanding mechanistic pathways that lead to cognitive decline and dementia in older adults represent a public health priority. The results of some studies have suggested that hearing loss is independently associated with poorer cognitive functioning[2-5] and incident dementia,[6,7] possibly through the effects of hearing loss on cognitive load or mediation through reduced social engagement.[6] However, cross-sectional[8] and prospective[9] studies have reported conflicting results that may be explained by variations in the study populations and in the methods used for hearing and cognitive assessments.

Hearing loss is prevalent in almost two-thirds of adults older than 70 years and remains undertreated.[10,11] Determining if hearing loss is independently associated with cognitive decline is an important first step toward understanding whether the use of hearing rehabilitative interventions could help mitigate cognitive decline. Using standardized audiometric and cognitive tests, we investigated the association of hearing loss with cognitive trajectories and incident cognitive impairment during a 6-year period in a community-based biracial cohort of older adults without prevalent cognitive impairment.

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METHODS

STUDY POPULATION

Participants were enrolled in the Health ABC (Health, Aging, and Body Composition) Study,[12,13] a prospective observational investigation started in 1997-1998 that enrolled 3075 well-functioning community-dwelling older adults aged 70 to 79 years. Study participants were recruited from a random sample of Medicare beneficiaries of white and black race/ethnicity living within zip codes in Pittsburgh, Pennsylvania, and Memphis, Tennessee, who were within a 1-hour drive of the examination site. Only individuals of white and black race/ethnicity were recruited because an original study objective was to examine race/ethnicity differences in body composition variables, and resources were insufficient to include other races/ethnicities. To be eligible, participants had to report no difficulty with walking a quarter mile, climbing 10 steps without resting, or performing basic activities of daily living.

Audiometric testing was administered in year 5 (2001-2002) of the Health ABC Study. Participants were followed up for 6 years. Of 2206 participants who underwent hearing testing, 1984 older adults (mean age, 77.4 years) had no evidence of cognitive impairment (defined by a Modified Mini-Mental State Examination [3MS] score of ≥80), and these participants comprised our analytic (baseline) cohort. Some participants did not undergo audiometric testing in year 5 for various reasons (eg, attrition from death, dropout, or missed study visit). All study participants signed a written informed consent, and this study was approved by the institutional review boards of the study sites.

AUDIOMETRY

Audiometric assessments were performed in a sound-treated booth. Air conduction thresholds in each ear were obtained from 0.25 to 8 kHz with headphones (TDH 39; Telephonics Corporation) using an audiometer (MA40; Maico Diagnostics) calibrated to American National Standards Institute standards (S3.6-1996). All thresholds are reported as decibels of hearing level. A pure-tone average (PTA) of hearing thresholds at 0.5 to 4 kHz was calculated for the better-hearing ear. Hearing loss was defined as a PTA exceeding 25 dB per the definition of impairment by the World Health Organization[14] (the level at which hearing loss begins to impair daily communication).

COGNITIVE ASSESSMENTS

The 3MS and the Digit Symbol Substitution (DSS) test were administered in year 5 (2001-2002), year 8 (2004-2005), year 10 (2006-2007), and year 11 (2007-2008). The 3MS is a global test with components for orientation, concentration, language, praxis, and memory.[15] The maximum score is 100, and 3MS scores of less than 80 are considered indicative of cognitive impairment (a cut point of 80 is 91% sensitive and 97% specific for dementia).[15] The DSS test is a nonverbal test of psychomotor speed and executive function[16] in which participants code a series of numbers with the corresponding symbol in 90 seconds. We defined incident cognitive impairment as a 3MS score of less than 80 or a decline in 3MS score of more than 5 points from baseline.[17-19]

OTHER COVARIATES

At enrollment, participants reported their age, sex, race/ethnicity, and education. Prespecified algorithms based on self-report and physician diagnoses, recorded medications, and laboratory data were used to define the presence of hypertension (based on clinic measure, medications, or self-report) and diabetes mellitus (based on fasting blood glucose level, medications, or self-report). Stroke history, smoking status (current, former, or never), and hearing aid use ("Do you wear a hearing aid?") were based on interviewer-administered questionnaires. Risk factors for cognitive decline not known to be associated with hearing loss (eg, alcohol use and hyperlipidemia) were excluded as covariates in the analytic model. In sensitivity analyses, participants were defined as remaining dementia free if they did not use any dementia medications (memantine hydrochloride and acetylcholinesterase inhibitors) or have any dementia-related hospitalizations in a review of inpatient records.[20] This limited diagnostic definition has been used previously[20] and was used in the present analyses only to exclude potentially influential data points from patients with possibly more advanced dementia. Depressive symptoms at baseline were assessed with the 20-item Centre for Epidemiological Studies Depression Scale.[21]

STATISTICAL ANALYSIS

Baseline characteristics of the study participants were compared using the Wilcoxon rank sum test and the χ^2 test. We created linear mixed-effects models to assess the association of hearing loss with repeated measures of the 3MS and DSS test over time, with individual-specific cognitive score and annual rate of change over time modeled as random effects. In linear mixed-effects models, an interaction term of hearing loss × time was included to assess whether hearing loss at baseline affected the individual rate of change in 3MS and DSS test scores. Discrete-time Cox proportional hazards regression models were used to study the time to incident cognitive impairment. Unless otherwise specified, all models were adjusted for demographic risk factors (age, sex, race/ethnicity, education, and study site) and for cardiovascular risk factors (smoking status, hypertension, diabetes, and stroke history) as time-constant covariates. Regression model assumptions were checked with residual plots and histograms. Participants with missing covariate data (<1% of the analytic cohort in all analyses) were excluded from analyses. Significance testing for all analyses was 2-sided, with a type I error of .05. Statistical software (SAS 9.2; SAS Institute) was used.

RESULTS

At baseline, participants with hearing loss were more likely to be male, older, and of white race/ethnicity and to have a history of smoking than participants with normal hearing (**Table 1**). Individuals with hearing loss primarily had mild hearing loss (PTA > 25 to 40 dB; 762 participants [65.6%]) or moderate hearing loss (PTA > 40 to 70 dB; 386 participants [33.2%]) rather than severe hearing loss (PTA > 70 dB; 14 participants [1.2%]).

In mixed-effects models adjusted for demographic and cardiovascular risk factors, hearing loss was associated with lower baseline 3MS scores (**Table 2**). On average, individuals with hearing loss had cognitive scores at baseline that were −0.75 (95% CI, −1.17 to −0.33) points lower on the 3MS and −0.92 (95% CI, −1.94 to 0.10) points lower on the DSS test than individuals with normal hearing. The association of other covariates with baseline cognitive scores is summarized in the eTable (http://www.jamainternalmed.com).

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Table 1. Demographic and Clinical Characteristics of the Baseline (Year 5) Study Cohort by Hearing Status[a]

Characteristic	Normal Hearing (n = 822)	Hearing Loss (n = 1162)	*P* Value
Age, mean (SD), y	76.8 (2.7)	77.9 (2.8)	<.001
Race/ethnicity, No. (%)			
Black	345 (42.0)	299 (25.7)	<.001
White	477 (58.0)	863 (74.3)	
Male sex, No. (%)	310 (37.7)	641 (55.2)	<.001
Education, No. (%)			
<12th grade	132 (16.1)	207 (17.8)	.50
High school graduate	276 (33.7)	399 (34.3)	
≥Some college	411 (50.2)	556 (47.8)	
Study site, No. (%)			
Memphis, Tennessee	348 (42.3)	563 (48.5)	.008
Pittsburgh, Pennsylvania	474 (57.7)	599 (51.5)	
Smoking status, No. (%)			
Current	43 (5.2)	68 (5.9)	.002
Former	369 (44.9)	606 (52.4)	
Never	409 (49.8)	483 (41.7)	
Hypertension, No. (%)	639 (77.7)	892 (76.8)	.63
Diabetes mellitus, No. (%)	140 (17.0)	226 (19.4)	.18
Stroke history, No. (%)	57 (6.9)	111 (9.6)	.04
Hearing aid use, No. (%)	6 (0.7)	251 (21.6)	<.001
Pure-tone average, mean (SD), dB	18.1 (5.0)	38.7 (10.7)	<.001
CES-D, mean (SD) score	4.70 (4.11)	4.85 (4.28)	.43

Abbreviation: CES-D, Centre for Epidemiological Studies Depression Scale.
[a]Hearing loss is defined as a speech-frequency pure-tone average of hearing thresholds at 0.5, 1, 2, and 4 kHz in the better-hearing ear exceeding 25 dB. Some variable totals do not sum to column totals because of missing data.

Table 2. Adjusted Mean Baseline Differences and Annual Rates of Change in Modified Mini-Mental State Examination (3MS) and Digit Symbol Substitution (DSS) Test Scores for Individuals With Normal Hearing vs Hearing Loss in Multivariate Mixed-effects Models[a]

Variable	Score (95% CI): Normal Hearing (n = 822)	Score (95% CI): Hearing Loss (n = 1162)	*P* Value
Baseline score difference			
3MS	1 [Reference]	−0.75 (−1.17 to −0.33)	<.001
DSS	1 [Reference]	−0.92 (−1.94 to 0.10)	.08
Annual change in score			
3MS	−0.46 (−0.55 to −0.36)	−0.65 (−0.73 to −0.56)	.004
DSS	−0.63 (−0.75 to −0.51)	−0.83 (−0.94 to −0.73)	.02

[a]Hearing loss is defined as a speech-frequency pure-tone average of hearing thresholds at 0.5, 1, 2, and 4 kHz in the better-hearing ear exceeding 25 dB. All models are adjusted for age, sex, race/ethnicity, education, study site, smoking status, hypertension, diabetes mellitus, and stroke history.

We investigated whether baseline hearing loss was associated with subsequent cognitive trajectories (Table 2). On the 3MS, individuals with hearing loss had annual rates of decline that were 41% greater than those among individuals with normal hearing (−0.65 vs −0.46 points per year, *P* = .004); a difference of −0.19 points per year is 0.23 of the SD of the estimated annual rates of decline among those with normal hearing (SD, 0.83). On average, during the 6-year follow-up period, individuals with hearing loss had adjusted 3MS scores that declined from 90.3 (95% CI, 89.8-90.8) at baseline to 86.4 (95% CI, 85.7-87.1) at the end of the follow-up period compared with 91.0 (95% CI, 90.5-91.6) at baseline and 88.3 (95% CI, 87.5-89.1) at the end of the follow-up period for individuals with normal hearing. On the DSS test, individuals with hearing loss had annual rates of decline that were 32% greater than those among individuals with normal hearing (−0.83 vs −0.63 points per year, *P* = .02); a difference of −0.20 points per year is 0.33 of the SD of the estimated annual rates of decline among those with normal hearing (SD, 0.60). On average, individuals with hearing loss had DSS test scores of 31.1 (95% CI, 29.9-32.3) at baseline and 26.1 (95% CI, 24.8-27.4) at the 6-year follow-up visit compared with 32.0 (95% CI, 30.7-33.4) at baseline and 28.3 (95% CI, 26.9-29.6) at the 6-year follow-up visit for individuals with normal hearing (**Figure**).

To exclude potentially influential data points, sensitivity analyses restricting the analytic cohort to those who were without severe hearing loss (n = 1970) or to those who remained dementia free during the follow-up period (n = 1749) did not substantively affect the results (Table 2). In the latter analysis of individuals who remained dementia free, accelerated annual rates of cognitive decline were still observed in individuals with hearing loss vs individuals with normal hearing (on the 3MS, −0.46 vs −0.30 points per year, *P* = .002; and on the DSS test, −0.72 vs −0.54 points per year, *P* = .03). We also investigated whether adjustment for depressive symptoms as a possible mediator in the association of hearing loss with cognition would attenuate the observed association. In these analyses adjusted for Centre for Epidemiological Studies Depression Scale scores at baseline, the magnitude of the association of hearing loss with accelerated cognitive decline was not substantively changed.

We explored whether hearing loss severity at baseline was associated with the magnitude of the observed rate of subsequent cognitive decline. Compared with the rate of 3MS score decline in individuals with normal hearing (−0.45; 95% CI, −0.55 to −0.36 points per year), the rates of 3MS score decline were significantly greater in individuals with mild hearing loss (−0.61; 95% CI, −0.72 to −0.51 points per year; *P* = .03) and in individuals with moderate or greater hearing loss (−0.71; 95% CI, −0.85 to −0.56 points per year; *P* = .005). Similarly, compared with the rate of DSS test score decline in individuals with normal hearing (−0.63; 95% CI, −0.75 to −0.51 points per year), the rates of DSS test score decline were also greater in individuals with mild hearing loss (−0.79; 95% CI, −0.92 to −0.65 points per year; *P* = .09) and in individuals with moderate or greater hearing loss (−0.92; 95% CI, −1.11 to −0.74 points per year; *P* = .01). Treating hearing loss as a continuous predictor variable yielded similar results. For the 3MS and DSS test, respectively, every 10 dB of hearing loss at baseline was associated with an incremental additional rate of decline of −0.07 (95% CI, −0.12 to −0.02 points per year; *P* = .003) and −0.06 (95% CI, −0.12 to −0.004 points per year; *P* = .04).

We analyzed the association of baseline hearing loss with incident cognitive impairment in 1626 individuals

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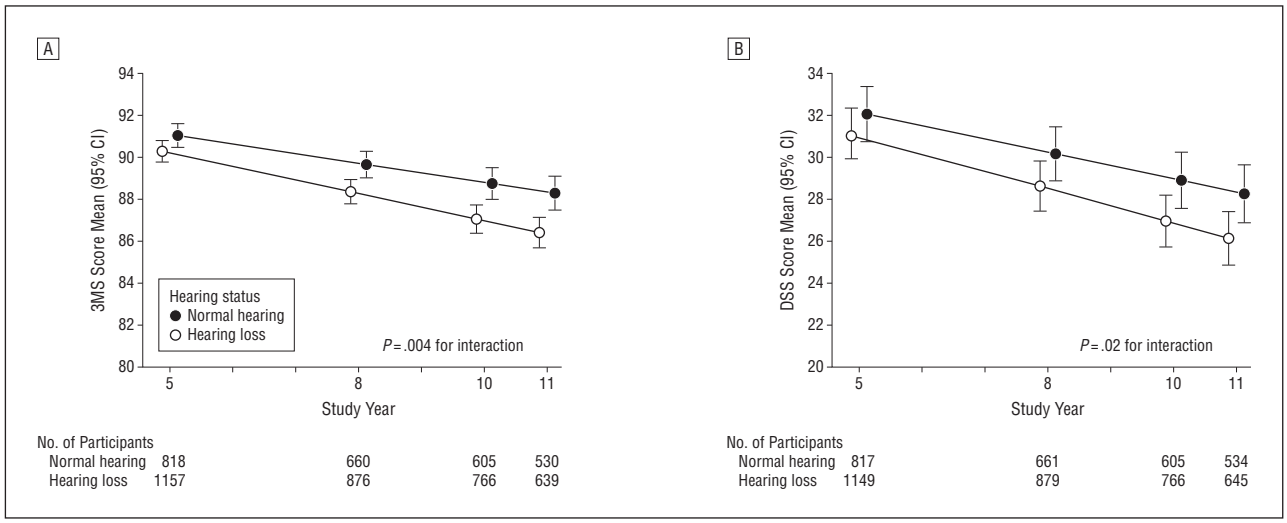

Figure. Multivariate mixed-effects models for adjusted mean scores by study year and by baseline hearing status. A, Modified Mini-Mental State Examination (3MS). B, Digit Symbol Substitution (DSS) test. Error bars indicate 95% CIs. All models are adjusted for age, sex, race/ethnicity, education, study site, smoking status, hypertension, diabetes mellitus, and stroke history. The interaction term is between hearing loss and time. Study year 5 is 2001-2002, study year 8 is 2004-2005, study year 10 is 2006-2007, and study year 11 is 2007-2008.

Table 3. Cox Proportional Hazards Regression Models for Incident Cognitive Impairment by Baseline Hearing Status in 1626 Individuals With at Least 1 Follow-up Visit[a]

Model[b]	Hazard Ratio (95% CI)	*P* Value
Hearing loss vs normal hearing[c]	1.24 (1.05-1.48)	.01
Hearing loss category[d]		
Mild	1.19 (0.99-1.44)	.06
≥Moderate	1.36 (1.08-1.70)	.008
Per 10 dB of hearing loss	1.07 (1.01-1.14)	.03

[a] Cognitive impairment was defined as a Modified Mini-Mental State Examination score of less than 80 or a decline in the Modified Mini-Mental State Examination score of more than 5 points from baseline. The analytic cohort was restricted to 1626 individuals with at least 1 follow-up visit after the baseline (year 5) assessment.

[b] All models are adjusted for age, sex, race/ethnicity, education, study site, smoking status, hypertension, diabetes mellitus, and stroke history.

[c] Hearing loss is defined as a pure-tone average exceeding 25 dB. Normal hearing is defined as a pure-tone average of 25 dB or less.

[d] Reference is normal hearing. Mild hearing loss is defined as a pure-tone average of greater than 25 to 40 dB. Moderate or greater hearing loss is defined as a pure-tone average exceeding 40 dB.

with at least 1 follow-up visit (**Table 3**). In total, 609 cases of incident cognitive impairment were recorded during the 6-year follow-up period. Individuals having hearing loss at baseline had a 24% (hazard ratio, 1.24; 95% CI, 1.05-1.48; *P* = .01) increased risk for incident cognitive impairment during the follow-up period compared with individuals having normal hearing. The magnitude of this association was linearly associated with the severity of an individual's hearing loss at baseline.

We also examined whether hearing aid use among individuals with hearing loss was associated with cognitive trajectories. In these analyses restricted to individuals with moderate or greater hearing loss (individuals in whom hearing aid use was more common) and adjusted for demographic factors, 182 individuals using hearing aids compared with 218 individuals not using hearing aids had higher baseline cognitive scores on the 3MS (difference of 1.06; 95% CI, 0.16-1.97 points; *P* = .02) but not on the DSS test (difference of 0.96; 95% CI, −1.2 to 3.1 points; *P* = .38). Rates of cognitive decline were not significantly attenuated in individuals using hearing aids vs those not using hearing aids (on the 3MS, −0.62 [95% CI, −0.84 to −0.41] vs −0.77 [95% CI, −0.98 to −0.56] points per year, *P* = .36; and on the DSS test, −0.82 [95% CI, −1.06 to −0.58] vs −0.98 [95% CI, −1.22 to −0.75] points per year, *P* = .34). Hearing aid use was not significantly associated with lower risk for incident cognitive impairment (hazard ratio, 0.82; 95% CI, 0.58-1.16; *P* = .26).

COMMENT

Our results demonstrate that hearing loss is independently associated with accelerated cognitive decline and incident cognitive impairment in community-dwelling older adults. The magnitude of these associations is clinically significant, with individuals having hearing loss demonstrating a 30% to 40% accelerated rate of cognitive decline and a 24% increased risk for incident cognitive impairment during a 6-year period compared with individuals having normal hearing. On average, individuals with hearing loss would require 7.7 years to decline by 5 points on the 3MS (a commonly accepted level of change indicative of cognitive impairment[17-19]) vs 10.9 years in individuals with normal hearing.

Our results are consistent with prior research demonstrating significant associations between greater hearing loss and poorer cognitive function on verbal cognitive tests[3-5,7,8,22-26] and nonverbal cognitive tests[2,3,5,24,26] and in cross-sectional and prospective studies.[5,27] In contrast, other studies[8,9,28] have not found similar associations. A key limitation across these prior studies is the variability in how hearing loss was measured and how audiometric data were analyzed (eg, the choice of pure-tone thresholds used to define hearing loss). Most studies used portable or screening audiometers[5,8,26,28] or tested

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participants under varying environmental conditions (eg, home-based testing).[26] The effect of biased or imprecise assessments of hearing thresholds would likely decrease the sensitivity to detect associations because of increased variance. These prior studies also generally were conducted in study populations from which the observed results may not be generalizable. Strengths of the present study include that it was performed among a population-based cohort of community-dwelling older adults, our results are based on both verbal and nonverbal cognitive tests, and audiometric assessments of hearing used a definition of hearing loss adopted by the World Health Organization.[14]

Several mechanisms may be theoretically implicated in the observed association between hearing loss and cognition. Poor verbal communication associated with hearing loss may confound cognitive testing; vice versa, overdiagnosis of hearing loss may have occurred in individuals with subclinical cognitive impairment. Miscommunication is unlikely given that hearing loss (short of severe hearing loss) should minimally impair face-to-face communication in quiet environments (ie, during cognitive testing),[29] particularly with testing administered by experienced examiners accustomed to working with older adults. Our results were also consistent using both verbal (3MS) and nonverbal (DSS) tests and were not sensitive to the exclusion of individuals with severe hearing loss from the analytic cohort.

An overdiagnosis of hearing loss is also unlikely because no evidence exists that subclinical cognitive impairment would affect the reliability of audiometric testing. Behaviorally, pure-tone audiometry has been reliably performed in adults with early dementia[7] and is routinely performed in children as young as 4 years. Also, no evidence suggests that older adults compared with younger adults adopt a more conservative response bias in reporting the detection of the auditory signal during pure-tone audiometry.[30]

A shared neuropathologic origin underlying both hearing loss and cognitive decline is a possibility, but our study relied on a measure that primarily reflects peripheral hearing loss. Pure-tone audiometry is typically considered a measure of the auditory periphery because detection of pure tones relies on cochlear transduction and neuronal afferents to brainstem nuclei, without requiring significant higher auditory cortical processing.[31] Neuropathologic conditions associated with Alzheimer disease have not been found in the peripheral auditory pathways.[32,33]

Finally, hearing loss may be mechanistically associated with cognitive decline, possibly through social isolation or cognitive load. Communication impairments caused by hearing loss can lead to social isolation and loneliness in older adults,[34,35] and epidemiologic[36,37] and neuroanatomic[38] studies have demonstrated associations between loneliness and cognitive decline or dementia. The effect of hearing loss on cognitive load is suggested by the results of studies[39-42] demonstrating that under conditions where auditory perception is difficult (ie, in the case of hearing loss), greater cognitive resources are dedicated to auditory perceptual processing, to the detriment of other cognitive processes such as working memory. Neuroimaging studies[43,44] have demonstrated a compensatory recruitment of regions in the prefrontal and temporoparietal cortex to maintain auditory speech processing in older adults, and this pattern of neural compensation may explain the general preservation of language comprehension that is seen even in individuals with advanced dementia.[45]

In the present study, hearing aid use was associated with slightly attenuated rates of cognitive decline and risk for cognitive impairment among individuals with hearing loss, but these results were not significant. Our study cohort may have been underpowered to detect a significant association, and data on other key variables (eg, years of hearing aid use, adequacy of hearing aid fitting and rehabilitation, etc) that would affect the success of hearing loss treatment and influence any observed association were unavailable. Contrary to popular perceptions, proper hearing rehabilitative treatment is complex, does not simply consist of using a hearing aid, and can vary substantially depending on the treating audiologist.[46] These observational results also must be interpreted with caution because individuals choosing to use a hearing aid likely differ significantly from those individuals not using a hearing aid in measured and unmeasured factors. Consequently, whether hearing rehabilitative strategies could affect cognitive decline remains unknown and will likely be determined only a randomized controlled trial.

A key limitation of our study is that we cannot determine the mechanistic basis of the observed association between hearing loss and cognitive decline. In particular, hearing loss may plausibly contribute to an overall cycle of multimorbidity and frailty or may synergistically interact with other known risk factors for dementia,[47-50] both of which could lead to cognitive decline in older adults. However, the hypothesized pathways underlying the association of hearing loss with cognition are not mutually exclusive; hence, multiple pathways (eg, shared neuropathologic conditions, cognitive load, and increased loneliness) could likely coexist and synergistically contribute to accelerated cognitive decline in individuals with hearing loss. Another limitation of our study is that hearing loss was measured only at baseline, and information was unavailable on the trajectory or the possible origin of the hearing loss. However, it is unlikely that this limitation would lead to a differential bias in our results. Residual confounding by other environmental or neuropathologic processes is also plausible but is speculative based on our knowledge of known risk factors for hearing loss and cognitive decline.

In conclusion, our results suggest that hearing loss is associated with accelerated cognitive decline and incident cognitive impairment in older adults. Further research is needed to investigate what the mechanistic basis of this observed association is and whether such pathways would be amenable to hearing rehabilitative interventions.

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Author Contributions: Dr Lin had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. *Study concept and design:* Lin and Simonsick. *Acquisition of data:* Yaffe, Harris, Purchase-Helzner, Satterfield, Ayonayon, and Simonsick. *Analysis and interpretation of data:* Lin, Yaffe, Xia, Xue, Ayonayon, Ferrucci, and Simonsick. *Drafting of the manuscript:* Lin. *Critical revision of the manuscript for important intellectual content:* Lin, Yaffe, Xia, Xue, Harris, Purchase-Helzner, Satterfield, Ayonayon, Ferrucci, and Simonsick. *Statistical analysis:* Xia and Xue. *Obtained funding:* Lin. *Administrative, technical, or material support:* Lin, Yaffe, Harris, Purchase-Helzner, Satterfield, Ayonayon, Ferrucci, and Simonsick. *Study supervision:* Lin and Simonsick.

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EDITOR'S NOTE

Evolving Insights About the Impact of Sensory Deficits in the Elderly

In this issue of the journal, we are presented with observational data about the high prevalence of dual (hearing and vision) sensory impairment, as well as evidence of an association between hearing impairment and cognitive decline, among the elderly. Because

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caring for older adults means focusing on improving and maintaining function, these articles show the potential for correcting hearing loss and visual loss so that elders can function better with appropriate treatment. Although there is insufficient evidence to recommend screening for both types of impairment, efficacious treatments exist for both, and physicians should be attentive to signs of either impairment. The finding that more input to the brain through better hearing may improve cognition is appealing and worth further study. More work is needed on assessing whether attention to case identification of sensory impairment, and appropriate treatment, improves patient-centered outcomes in older adults. With the expanding population of the elderly, multiple sensory deficits will likely become of sufficient population burden that we cannot afford to neglect them.

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