Hearing Loss and Cognition in the Baltimore Longitudinal Study of Aging

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Objective: To determine the relationship between hearing loss and cognitive function as assessed with a standardized neurocognitive battery. We hypothesized a priori that greater hearing loss is associated with lower cognitive test scores on tests of memory and executive function. *Method:* A cross-sectional cohort of 347 participants ≥55 years in the Baltimore Longitudinal Study of Aging without mild cognitive impairment or dementia had audiometric and cognitive testing performed in 1990–1994. Hearing loss was defined by an average of hearing thresholds at 0.5, 1, 2, and 4 kHz in the better-hearing ear. Cognitive testing consisted of a standardized neurocognitive battery incorporating tests of mental status, memory, executive function, processing speed, and verbal function. Regression models were used to examine the association between hearing loss and cognition while adjusting for confounders. *Results:* Greater hearing loss was significantly associated with lower scores on measures of mental status (Mini-Mental State Exam), memory (Free Recall), and executive function (Stroop Mixed, Trail Making B). These results were robust to analyses accounting for potential confounders, nonlinear effects of age, and exclusion of individuals with severe hearing loss. The reduction in cognitive performance associated with a 25 dB hearing loss was equivalent to the reduction associated with an age difference of 6.8 years. *Conclusion:* Hearing loss is independently associated with lower scores on tests of memory and executive function. Further research examining the longitudinal association of hearing loss with cognitive functioning is needed to confirm these cross-sectional findings.

Keywords: hearing loss, cognition, aging, dementia

We have previously demonstrated that audiometric hearing loss is independently associated with incident all-cause dementia in the Baltimore Longitudinal Study of Aging (BLSA) and that these results were robust to sensitivity analyses adjusting for known confounders, nonlinear effects, and other potential biases (Lin et al., 2011). Mechanistic pathways hypothesized to explain this observed association include a shared pathologic etiology, the effects of hearing loss on cognitive load and cognitive reserve, and/or mediation through social isolation and loneliness (Lin et al., 2011). Most likely, a number of these hypothesized pathways coexist and contribute to the development of cognitive impairment.

Regardless of the mechanism, a first step in exploring the pathway from hearing loss to dementia is to demonstrate that hearing loss is selectively associated with those cognitive measures and domains known to decline prior to dementia onset. Results from longitudinal studies have generally demonstrated that

decrements in both measures of memory (Elias et al., 2000; Grober, Hall, Lipton, et al., 2008; Linn et al., 1995; Rubin et al., 1998) and executive function (Chen et al., 2001; Fabrigoule et al., 1998; Grober, Hall, Lipton, et al., 2008; Rapp & Reischies, 2005; Royall, Chiodo, & Polk, 2004) precede subsequent dementia with accelerated declines in episodic memory and executive function observed 7 years and 3 years, respectively, before diagnosis (Grober, Hall, Lipton, et al., 2008; Hall, Lipton, Sliwinski, & Stewart, 2000; Hall et al., 2001). In contrast, measures of verbal intelligence do not decline until shortly before dementia diagnosis (Grober, Hall, Lipton, et al., 2008).

In the present study, we investigated the association of hearing loss with cognitive function using a standardized neurocognitive battery in a cross-sectional cohort of BLSA participants without mild cognitive impairment or dementia. This neurocognitive battery included tests of mental status (Mini-Mental State Exam [MMSE]), memory (Free and Cued Selective Reminding test [FCSRT]), executive function/attention (Trail Making B, Stroop Mixed), processing and psychomotor speed (Trail Making A, Stroop Colors & Words), and verbal ability and language (Category & Letter Fluency, American Version of the Nelson Adult Reading Test [AMNART]). We hypothesized a priori that greater hearing loss is associated with lower cognitive test scores on tests of memory and executive function.

Method

Study Participants

Subjects were participants in the BLSA, an ongoing prospective study of the effects of aging that was initiated in 1958 by the National Institute on Aging (Shock et al., 1984). The BLSA cohort

is comprised of community-dwelling volunteers who travel to the National Institute on Aging (NIA) in Baltimore biennially for 2.5 days of intensive testing. From 1990–1994, audiometric testing was done in conjunction with neurocognitive evaluation, and the present investigation is based on a cross-sectional cohort of participants ($n = 347$) who were ≥55 years when examined in 1990–1994, had concurrent audiometric and neurocognitive testing, and did not have prevalent dementia or mild cognitive impairment at the time of evaluation. The National Institute on Aging and the Johns Hopkins School of Medicine Institutional Review Boards approved this study, and all participants gave written informed consent.

Diagnosis of Dementia

The protocol for adjudication of dementia in the BLSA has been used continuously since 1986 and has been described previously (Kawas, Gray, Brookmeyer, Fozard, & Zonderman, 2000). If subjects were determined to have clinically significant cognitive decline (typically memory) but did not meet criteria for dementia, they were classified as suspected dementia which corresponds to the current diagnosis of mild cognitive impairment (Petersen et al., 1999).

Cognitive Testing

Neurocognitive testing was performed by an experienced examiner accustomed to working with older adults and using a standardized protocol and neurocognitive battery. Cognitive test data available for the cohort under investigation include: MMSE ($n = 340$; Folstein, Folstein, & McHugh, 1975; FCSRT ($n = 343$); Trail Making A and B ($n = 338$); Stroop ($n = 314$); Letter and Category Fluency ($n = 345$); and AMNART ($n = 235$). Reduced numbers of participants assessed with the Stroop and AMNART reflect changes in the test battery in 1993.

FCSRT. The FCSRT measures memory under conditions that control attention and cognitive processing (Grober, Hall, McGinn, et al., 2008). It has been used in five major longitudinal aging studies (Grober, Buschke, Crystal, Bang, & Dresner, 1988a; Lindenberger, 1999; Petersen et al., 1995; Sarazin et al., 2007; Tuokko, Vernon-Wilkinson, Weir, & Beattie, 1991) and also in the Alzheimer's Disease Cooperative Study Instrumentation Protocol (Ferris et al., 2006). The FCSRT has been shown to be sensitive to early dementia and preclinical dementia in several cohorts (Grober, Buschke, Crystal, Bang, & Dresner, 1988b; Grober, Hall, McGinn, et al., 2008; Grober & Kawas, 1997; Grober, Lipton, Hall, & Crystal, 2000; Lindenberger, 1999; Petersen, Smith, Ivnik, Kokmen, & Tangalos, 1994; Tounsi et al., 1999; Tuokko et al., 1991) and is not associated with education (Ivnik et al., 1997) or race (Grober et al., 1988b; Grober, Hall, McGinn, et al., 2008). The FCSRT begins with a study phase in which subjects are asked to search a card containing four pictures (e.g., grapes) for an item that goes with a unique category cue (e.g., fruit). After all four items are identified, immediate recall of just those four items is tested. The search is performed again for items not retrieved by cued recall. The search procedure is continued until all 16 items are identified and retrieved in immediate recall. The study procedure is followed by three trials of recall each consisting of free recall followed by cued recall for items not retrieved by free recall.

The sum of the three free recall trials is a measure of learning and memory and is the measure used in the present analyses.

Trail Making Test. The Trail Making Test involves drawing lines to connect consecutively numbered circles (Part A) and then connecting dots containing numbers and letters arrayed randomly on a page in alternating sequence (Part B) [Reitan, 1958]. The dependent measure used in the present investigation is the reciprocal of the time (speed) taken by the subject to complete the task expressed in seconds. This procedure allows us to interpret larger scores as being associated with better cognitive performance (e.g., a score of 1/30 s is greater than a score of 1/60 s).

Stroop Test. The Stroop Test was administered in three formats. Stroop Words required the participant to read aloud the name of a color printed in black ink, and Stroop Colors required the participant to name the color of a printed series of x's in one of three colors (red, blue, green). Stroop Mixed was a color-word interference task in which participants were asked to report the color in which each color word was printed when the color word was printed in ink of a different color. Scores were reported as the number of responses correctly named in 45 s.

Fluency. In the Letter Fluency task, subjects generated words that began with the letters F, A, and S for 1 min each (Spreen & Benton, 1969). In the Category Fluency test, subjects had 1 minute each to generate exemplars of animals, fruits, and vegetables (Rosen, 1980). The dependent measures were the mean numbers of words generated across the three letters and the three categories, respectively.

AMNART. The AMNART was used to estimate verbal IQ. It consists of 50 words that cannot be pronounced phonetically (e.g., depot, naïve) [Nelson & O'Connell, 1978]. Estimated verbal IQ was computed using number of errors on the AMNART and years of education according to the following formula: $118.56 - [.88 * (\text{number of errors})] + (.56 * \text{years of education})$.

Audiometry

From 1990–1994, audiometry was performed in the BLSA study using a semi-automated testing device (Virtual Equipment Co., Audiometer Model 320) in a sound-attenuating chamber under unaided conditions (Industrial Acoustics Company, Model 400-A) which met prevailing standards for maximal permissible ambient noise levels during air conduction audiometry (American National Standards Institute [ANSI], 1977). A speech-frequency pure tone average (PTA) of air-conduction thresholds at 0.5, 1, 2, and 4 kHz was calculated for each ear, and the PTA in the better-hearing ear was used for subsequent analyses because this ear would be the principal determinant of hearing and speech perception ability on an everyday basis. All thresholds are expressed in dB HL (ANSI, 1989).

Other Covariates

A diagnosis of diabetes was established based on a fasting glucose >125 mg/dL, a pathologic oral glucose tolerance test, or a positive history of a physician diagnosis plus treatment with oral antidiabetic drugs or insulin. The diagnosis of hypertension was established based on a systolic blood pressure >140 and/or diastolic blood pressure ≥90 mmHg or treatment with antihypertensive medications. Race (white/black/other), education (in years),

hearing aid use (yes/no) and smoking status (current/former/never) were based on self-report. Depressive symptoms were assessed using the Center for Epidemiologic Studies Depression Scale (CES-D) [Radloff, 1977]. The CES-D is a 20-item inventory of depressive symptoms, and each response is scored from zero to 3 based on the frequency of occurrence of the symptom. The range of scores is zero to 60, where higher CES-D scores indicate increased frequency and severity of depressive symptoms.

Statistical Analyses

Locally weighted scatterplot smoothing (lowess) was used to graphically explore the association of hearing loss and age with cognitive scores and to identify nonlinear data trends. Linear regression was then used to model the association between cognitive scores and hearing loss while adjusting for age and other covariates. A robust variance estimator was used to account for heteroscedasticity seen in the model residuals (Harrell, 2001). Age was modeled using a cubic spline when appropriate to account for possible nonlinear effects of age on cognitive scores. To account for ceiling effects observed in the distribution of scores from the MMSE and AMNART, scores from these cognitive tests were converted into a five-level ordinal categorical variable using cut-points that approximately divided scores into five equal sized bins. Ordinal logistic regression was then performed, and β-coefficients from these analyses can be interpreted as the log odds of the next higher category of cognitive function associated with a 10-db increase in hearing loss (i.e., negative β's indicate poorer cognitive function with increasing hearing loss). Subjects with missing data for noncognitive variables were excluded from analyses, and this represented $\leq 2\%$ of the study sample in all analyses except for the one analysis that incorporated hearing aid use. Significance testing for all analyses was two-sided with a Type I error of 0.05. The statistical software used was Stata 11.1 (StataCorp, College Station, TX).

Results

Demographics for the study population considered in this report are presented in Table 1. From 1990–1994, 347 participants $\geq$55 years had concurrent audiometric and neurocognitive testing and were assessed as being free from prevalent dementia or cognitive impairment. This cohort was predominantly white (93.1%) and well-educated (mean years of education, 16.6).

Exploratory analysis of the cross-sectional association of cognitive scores with hearing loss demonstrated that scores from all cognitive tests except AMNART and Letter Fluency generally declined linearly with increasing levels of hearing loss (see Figure 1). After adjusting for age, associations between greater hearing loss and lower scores on all cognitive tests were significant or approached significance except for Trail Making A and Letter Fluency (see Table 2). Stronger associations were observed between hearing loss and measures of memory (FCSRT Free Recall, $p < .001$) and executive function (Stroop Mixed, $p = .006$).

We performed additional analyses incorporating additional covariates to test the robustness of our results. Results from a model adjusting for age, sex, education, diabetes, smoking, and hypertension demonstrated that greater hearing loss was significantly associated with lower scores on the MMSE, CSR Free Recall, and

Table 1

Demographic Characteristics, Baltimore Longitudinal Study of Aging 1990–1994

Characteristic	Cohort ($n = 347$)
Age, mean years (*SD*)	71.0 (7.2)
Hearing loss, mean dB (*SD*)	25.5 (13.7)
Hearing loss category, *n* (%)	
Normal (<25 dB)	205 (59.1)
Mild (25–40 dB)	99 (28.5)
Moderate (41–70 dB)	40 (11.5)
Severe ($\geq$71 dB)	3 (0.9)
Sex, *n* (%)	
Female	122 (35.2)
Race, *n* (%)	
White	323 (93.1)
Black	19 (5.5)
Other	5 (1.4)
Education, mean years (*SD*)	16.6 (2.9)
Smoking, *n* (%)	
Never	121 (34.9)
Former	217 (62.5)
Current	9 (2.6)
Hypertension, *n* (%)	214 (61.7)
Diabetes, *n* (%)	65 (18.7)
Hearing aid use *n* (%)[a]	46 (13.3)

[a] Data on hearing aid use were missing for 32 participants.

Stroop Mixed and that associations between hearing loss and Trail Making A and B approached significance (see Table 2). Overall, in this fully adjusted model, stronger associations were observed between hearing loss and cognitive measures of memory and executive function than with tests of psychomotor/processing speed and verbal function.

A cubic spline model was used to account for possible nonlinear effects of age in those cognitive measures (e.g., FCSRT Free Recall) which demonstrated possible nonlinear associations with age (data not shown), and the results from these analyses were not substantially different from models adjusting for age as a linear variable (cf. Table 2). Excluding participants with a history of a previous stroke ($n = 7$) also did not substantially change the main findings (cf. Table 2). We also performed an analysis excluding those participants with the greatest hearing loss (severe hearing loss category, $n = 3$) to ensure that results were not determined by a few strongly influential data points. In this analysis, the results from the fully adjusted models in Table 2 also remained substantially unchanged with the exception that the associations of hearing loss with Trail Making A and B were no longer significant ($p > .1$). Adjustment for CES-D scores also did not affect the significance of the results presented in Table 2 (data not shown).

In order to examine the role of hearing aids on cognition, we performed an analysis with an interaction term between hearing aid use and having hearing loss >25 dB. In this model, hearing aid use was not associated with scores on any of the cognitive tests (data not shown).

To assess the magnitude of the reduction in cognitive performance associated with hearing loss, we estimated the difference in chronological age that would be equivalent to the cross-sectional effect of a 25-dB increase in hearing thresholds (analogous to shifting from normal hearing to a mild hearing loss) on cognitive scores. Cognitive tests that were associated with both age and
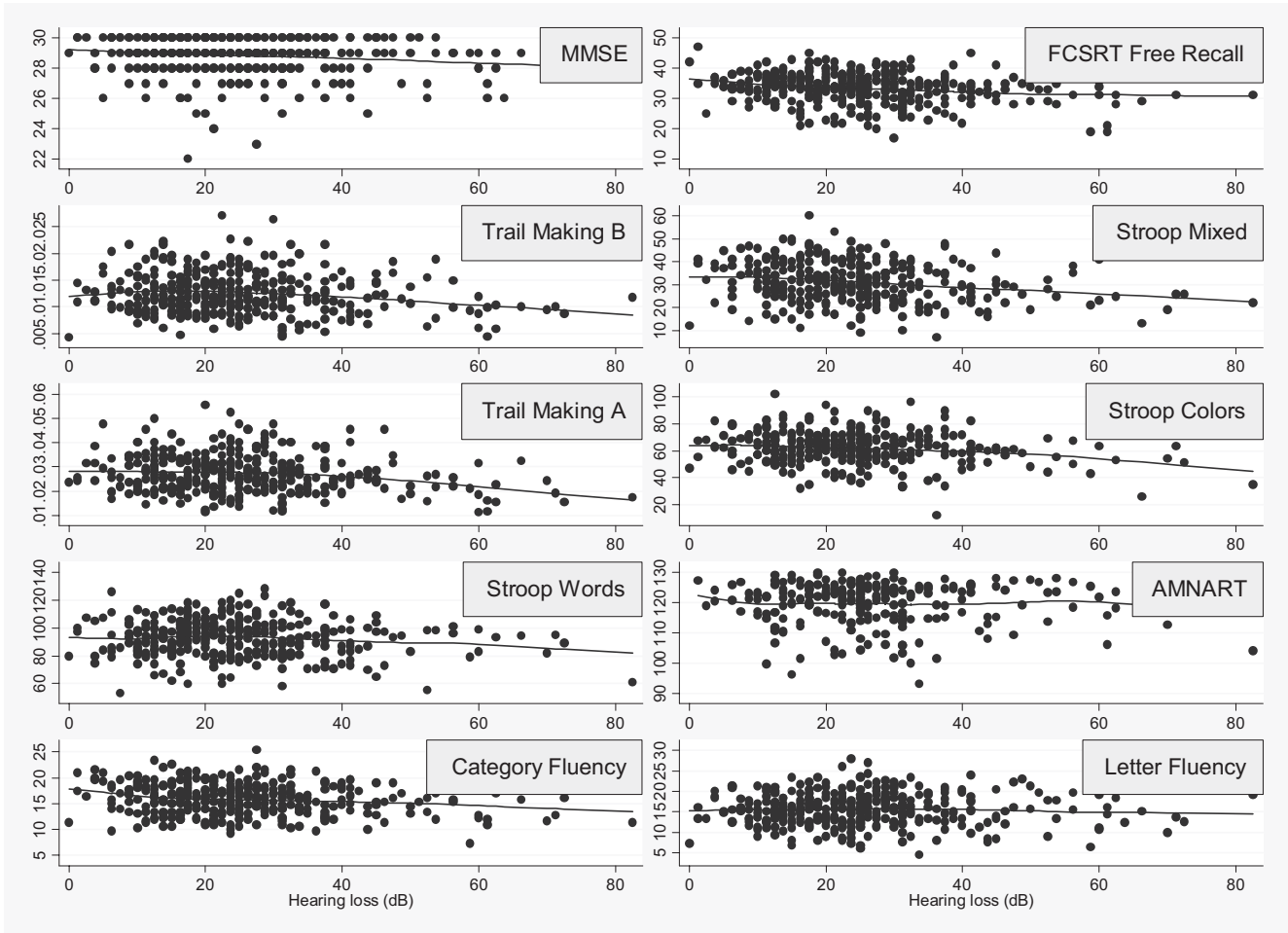
Figure 1. Cross-sectional association of hearing loss and cognitive scores, Baltimore Longitudinal Study of Aging, 1990–1994.

hearing loss in fully adjusted models include Stroop Mixed and Trail Making A and B (see Table 3). The difference in age equivalent to the cognitive reduction associated with a 25-dB increase in hearing loss is 6.8, 5.8, and 6.7 years for Stroop Mixed, Trail Making A, and Trail Making B, respectively.

Discussion

In this cross-sectional study of adults who were free of prevalent dementia or mild cognitive impairment, hearing loss was independently associated with tests of memory and executive function, and these results were robust to analyses accounting for confounders, nonlinear effects of age, and excluding participants with severe hearing loss. The magnitude of the reduction in cognitive performance associated with hearing loss is clinically significant with the reduction associated with a 25-dB hearing loss being equivalent to an age difference of 6.8 years on tests of executive function.

Our results contribute to the literature examining the association between hearing loss and cognition. Our findings are consistent with some prior research demonstrating significant associations between greater hearing loss and poorer cognitive function on both

verbal (Granick, Kleban, & Weiss, 1976; Gussekloo, de Craen, Oduber, van Boxtel, & Westendorp, 2005; Helzner et al., 2005; Lindenberger & Baltes, 1994; Ohta, Carlin, & Harmon, 1981; Tay et al., 2006; Thomas et al., 1983; Uhlmann, Larson, Rees, Koepsell, & Duckert, 1989; Valentijn et al., 2005) and nonverbal cognitive tests (Granick et al., 1976; Lindenberger & Baltes, 1994; Valentijn et al., 2005) and in both cross-sectional and prospective studies (Peters, Potter, & Scholer, 1988; Valentijn et al., 2005). In contrast, other studies have not found similar associations (Anstey, Luszcz, & Sanchez, 2001; Gennis, Garry, Haaland, Yeo, & Goodwin, 1991). While some heterogeneity in study results is explained by the choice of cognitive tests, one key limitation across multiple studies is the variability in how hearing loss was measured and how audiometric data were analyzed (e.g., choice of pure tone thresholds used to define hearing loss). Most studies utilized portable or screening audiometers (Anstey et al., 2001; Gussekloo et al., 2005; Lindenberger & Baltes, 1994; Valentijn et al., 2005) or tested participants under varying environmental conditions (e.g., home-based testing; Lindenberger & Baltes, 1994), while some did not adequately describe their audiometric testing protocol (Gennis

Table 2

Regression Models of Cognitive Scores per 10dB of Hearing Loss, Baltimore Longitudinal Study of Aging 1990–1994

	Adjusted for age		Adjusted for all covariates[c]	
	β^a (95% CI)	p^d	β^a (95% CI)	p^d
Mental status				
MMSE	-0.16^b ($-0.29 - -0.03$)	*	-0.17^b ($-0.30 - -0.04$)	*
Memory				
Free Recall from FCSRT	-0.76 ($-1.19 - -0.33$)	***	-0.58 ($-1.0 - -0.15$)	**
Executive function				
Trail Making Test Part B, Speed	-2.6×10^{-4} ($-5.6 \times 10^{-4} - -4.8 \times 10^{-5}$)	+	-3.0×10^{-4} ($-5.9 \times 10^{-4} - 1.1 \times 10^{-6}$)	+
Stroop Mixed	-1.0 ($-1.7 - -0.30$)	**	-0.91 ($-1.7 - -0.16$)	*
Processing and psychomotor speed				
Trail Making Test Part A, speed	-5.0×10^{-4} ($-1.1 \times 10^{-3} - 1.2 \times 10^{-4}$)		-6.4×10^{-4} ($-1.3 \times 10^{-3} - 9.4 \times 10^{-7}$)	+
Stroop Colors	-1.0 ($-2.1 - -0.06$)	+	-0.78 ($-1.9 - 0.29$)	
Stroop Words	-1.0 ($-2.2 - 0.19$)	+	-0.91 ($-2.2 - 0.36$)	
Verbal function and language				
Category Fluency	-0.27 ($-0.52 - -0.009$)	*	-0.09 ($-0.35 - 0.17$)	
Letter Fluency	0.15 ($-0.83 - 1.1$)		0.25 ($-0.79 - 1.3$)	
American Version of the Nelson				
Adult Reading Test	-0.18^b ($-0.37 - 0.012$)	+	-0.12^b ($-0.32 - 0.07$)	

[a] β-coefficient represents the expected difference in cognitive scores associated with a 10-db increase in hearing loss. Negative β's indicate poorer cognitive function with increasing hearing loss.   [b] β-coefficient represents the log odds of the next higher category of cognitive function associated with a 10db increase in hearing loss. Negative β's indicate poorer cognitive function with increasing hearing loss.   [c] Covariates include age, sex, race, education, diabetes, smoking, and hypertension.
[d] + $p < .10$.   * $p < .05$.   ** $p < .01$.   *** $p < .001$.

et al., 1991; Ohta et al., 1981; Thomas et al., 1983). The effect of biased or imprecise assessments of hearing thresholds would likely decrease sensitivity to detect associations due to increased variance. Strengths of our current study are the use of a standardized audiometric testing protocol performed in a sound-attenuating chamber, a definition of hearing loss adjudicated by the World Health Organization (www.who.int/pbd/deafness/hearing_impairment_grades/en/index.html"), and a standardized neurocognitive battery evaluating multiple cognitive domains.

A number of 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, or vice versa there may be an overdiagnosis of hearing loss in individuals with subclinical cognitive impairment. Miscommunication is unlikely given that hearing loss (short of severe-to-profound hearing loss) minimally impairs face-to-face communication in quiet environments (i.e., during cognitive test-

ing) [Gordon-Salant, 2005] particularly in the setting of testing by experienced examiners who are accustomed to working with older adults. A previous study by Lindenberger and colleagues [Lindenberger, Scherer, & Baltes, 2001] also demonstrated that artificially induced hearing loss (through the use of occlusive headphones) did not acutely affect the results of neurocognitive testing using both verbal and nonverbal cognitive tests. Confounding by poor verbal communication is also unlikely since the cognitive tests of memory (Free Recall) and executive function (Stroop and Trail Making) associated with hearing loss in the present study do not rely heavily on presentation of verbal information. We also conducted a sensitivity analysis excluding individuals with severe hearing loss, and the significance of the association of hearing loss with MMSE, Free Recall, and Stroop Mixed remained unchanged.

An overdiagnosis of hearing loss is also unlikely since there is no evidence that subclinical cognitive impairment would affect the reliability of audiometric testing. Clinically pure-tone audiometry

Table 3

Regression Models of Cognitive Scores per Year of Age or 25 dB of Hearing Loss for Cognitive Tests Associated With Both Age and Hearing Loss

	Age (per year)		Hearing loss (per 25 dB)		Δ Age (years) equivalent to 25 dB of hearing loss
	β^a (95% CI)	p	β^b (95% CI)	p	
Stroop Mixed	-0.33 ($-0.48 - -0.18$)	<.001	-2.27 ($-4.14 - -0.40$)	.02	6.8
Trail Making					
Part A, speed	-0.00027 ($-0.00040 - -0.00015$)	<.001	-0.0016 ($-0.0032 - 2.35 \times 10^{-6}$)	.05	5.8
Part B, speed	-0.00011 ($-0.00018 - -0.000044$)	.001	$-.00074$ ($-0.0015 - 2.74 \times 10^{-6}$)	.05	6.7

[a] β-coefficient represents the expected difference in cognitive scores associated with a 1 year increase in age. Negative β's indicate poorer cognitive function with increasing age.   [b] β-coefficient represents the expected difference in cognitive scores associated with a 25 db increase in hearing loss (analogous to shifting from normal hearing to mild hearing loss). Negative β's indicate poorer cognitive function with increasing hearing loss.
[a,b] All models adjusted for age, sex, race, education, diabetes, smoking, and hypertension.

is performed in children even as young as 4 years. There is also no evidence to suggest that older compared to younger adults adopt a more conservative response bias in reporting detection of the auditory signal during pure tone audiometry (Marshall, 1991).

A shared neuropathologic etiology 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 (Pickles, 2008). Neuropathology associated with Alzheimer's disease (AD) has not been found in the peripheral auditory pathways (Baloyannis, Mauroudis, Manolides, & Manolides, 2009; Sinha, Hollen, Rodriguez, & Miller, 1993). The likelihood of another neurobiological process such as microvascular disease causing both hearing loss and dementia also cannot be fully excluded. However, risk factors for vascular disease such as diabetes, smoking, and hypertension were adjusted for in our models, and our results were robust to excluding individuals with a prior stroke.

Finally, hearing loss may be causally associated with cognitive decline, possibly through social isolation, cognitive load, or a combination of these pathways. Communication impairments caused by hearing loss can lead to social isolation and loneliness in older adults (Strawbridge, Wallhagen, Shema, & Kaplan, 2000; Weinstein & Ventry, 1982), and epidemiologic (Barnes, Mendes de Leon, Wilson, Bienias, & Evans, 2004; Fratiglioni, Wang, Ericsson, Maytan, & Winblad, 2000) and neuroanatomic studies (Bennett, Schneider, Tang, Arnold, & Wilson, 2006) have demonstrated associations between loneliness and poor social networks with cognitive decline and dementia. Mechanisms that have been implicated in the association between loneliness and cognition included direct pathophysiologic effects of altered gene expression profiles and increased inflammation in lonely individuals (Cole, Hawkley, Arevalo, & Cacioppo; Cole et al., 2007) or through psychosocial pathways of social support and influence (Berkman, Glass, Brissette, & Seeman, 2000; Uchino, 2006).

The effect of hearing loss on cognitive load is suggested by studies demonstrating that under conditions where auditory perception is difficult (i.e., hearing loss), greater cognitive resources are dedicated to auditory perceptual processing to the detriment of other cognitive processes such as working memory (Pichora-Fuller, Schneider, & Daneman, 1995; Rabbitt, 1990; Rabbitt, 1968; Tun, McCoy, & Wingfield, 2009). Neuroimaging studies have demonstrated a compensatory recruitment of regions in the frontal and temporoparietal cortex to maintain auditory speech processing in older adults (Wingfield & Grossman, 2006), and this pattern of neural compensation may explain the general preservation of language comprehension that is seen even in older individuals with dementia (Rousseaux, Seve, Vallet, Pasquier, & Mackowiak-Cordoliani). The cognitive load induced by hearing loss could, therefore, result in a smaller pool of resources being available for other cognitive tasks under a resource capacity model proposed by Kahneman (Kahneman, 1973). Such a hypothesis is generally consistent with our observed results demonstrating that hearing loss was primarily associated with the more challenging cognitive tests that would be expected to overwhelm available resources (e.g., Free Recall, Stroop Mixed; Trails B) rather than cognitive tests focused on less complex speeded tasks or language.

In the current study, self-reported hearing aid use was not associated with higher scores on cognitive tests among participants with hearing loss, but data on other key variables (e.g., years of hearing aid use, type of hearing aid, hours worn per day, characteristics of subjects choosing to use hearing aids, use of other communicative strategies, adequacy of rehabilitation, etc.) that would affect the success of aural rehabilitation and affect any observed association were not available. Analogous to other putative risk factors for cognitive decline (e.g., microvascular disease), strategies aimed at prevention may need to take place many years before disease onset. Consequently, whether hearing devices and aural rehabilitative strategies could have an effect on cognitive decline remains unknown and will require further study.

A key limitation of our study is that our results are based on cross-sectional data rather than on longitudinal trajectories of cognitive function and hearing loss over time. Therefore, our estimates of the expected change in cognitive scores associated with hearing loss and age may be subject to bias by cohort effects or obscured by interindividual heterogeneity in participant characteristics. However, the relative homogeneity of our study cohort in both observed and likely unobservable characteristics may help limit these potential biases. Our results also demonstrated robust associations of hearing loss with cognitive domains of memory and executive function that were consistent with our a priori hypothesis, and this differential association of hearing loss with select cognitive domains may not be readily attributable to a particular bias.

Another limitation of our study is that our results may not be broadly generalizable because our cohort consisted of primarily white, well-educated adults. This potential limitation, however, could strengthen the internal validity of our findings given the relative homogeneity of the study cohort. Residual confounding by other medical or environmental factors is also possible but speculative based on our current knowledge of risk factors for hearing loss and cognitive decline that were adjusted for in our models.

If our results are confirmed longitudinally and in other independent studies, our findings potentially have significant implications for public health. Hearing loss is highly prevalent, and the effects of hearing loss are potentially treatable with rehabilitative devices and strategies that remain grossly underutilized (Lin, Thorpe, Gordon-Salant, & Ferrucci, 2011). Further research into whether such interventions could impact cognition and dementia are needed given the lack of past research in this area.

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