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Do self-reported hearing and visual impairments predict longitudinal dementia in older adults?

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Abstract

Background: Sensory impairments have been associated with dementia in older adults. However, the contribution of different impairments and how they interact in the development of dementia is not clear. We examined the independent and interaction effects of hearing impairment (HI) and visual impairment (VI) on incident dementia.

Design: Multi-centric population-based prospective cohort study.

Setting: Data were taken from the AgeDifferent.de platform, pooling participants aged 75 and older from the German LEILA75+ and AgeCoDe/AgeQualiDe cohorts.

Participants: Older adults ($N = 3497$) with mean age 79.8 years, 67.2% female.

Alexander Pabst and Jonathan Bär shared first authorship.

Preliminary results of this article were presented at the following meetings: AAIC 2019 (oral presentation).

[Correction added on 5 May 2021, after first online publication: Degree of Jonathan Bär has been changed.]

Measurements: Standardized interviews and questionnaires were used to assess self-reported HI and VI at baseline and all-cause dementia in 9 follow-ups, spanning over 20 years.

Methods: Competing risk regression models were conducted to test the main and interaction effects of HI and VI on dementia incidence, adjusting for established risk factors of dementia and accumulated mortality.

Results: HI and VI at baseline were reported by 30.3% and 16.6% of individuals, respectively. Adjusting for baseline information on sociodemographics, substance use, cognitive functioning and morbidity, and controlling for accumulated mortality risk, HI (sHR 1.16, 95% CI 1.04–1.30, $p = 0.011$) but not VI (sHR 1.07, 95% CI 0.90–1.28, $p = 0.462$) was significantly associated with incident dementia. There was no interaction between HI and VI (sHR 1.09, 95% CI 0.81–1.46, $p = 0.567$).

Conclusions: Hearing impairment is associated with an increased incidence of all-cause dementia in older adults. There is no excess risk or risk compensation through the additional presence or absence of visual impairment. Early prevention measures for hearing impairment might help to reduce the long-term risk of dementia.

KEYWORDS

cohort, dementia incidence, hearing, sensory impairment, vision

INTRODUCTION

Dementia remains a serious challenge for health care systems around the globe. Around 50 million people are currently living with dementia and an estimated 152 million people will be affected by 2050.[1] Dementia is today considered a major source of global disability and dependency in older adults. Yet, a decline in dementia incidence was found in western high-income countries.[2] It is estimated that more than one-third of dementia cases could be prevented by taking precautionary measures that address modifiable risk factors.[3,4] Such evidence-based measures of dementia prevention include regular physical activity, a healthy diet, and the management of cardiovascular risk factors such as diabetes mellitus, obesity, smoking, and hypertension.[5]

Evidence for preventive measures regarding other risk factors in older people, such as increasing sensory impairments, is less clear. Meta-analyses reported a significant association between hearing impairment (HI) and increased dementia risk.[6-8] At the same time, however, the longitudinal preventive effect of hearing aids is questioned.[1] In addition, little is known about the role of visual impairment (VI) on dementia risk and corresponding studies showed inconsistent findings. While Rogers and Langa[9] found a longitudinal effect of VI on dementia incidence among older adults, this was not the case in other studies.[10,11] Moreover, the individual contributions of different sensory modalities and their interplay in the development of dementia are not entirely clear. The concurrent existence of HI and VI has been associated more strongly with cognitive decline and dementia than the presence of a single impairment in some studies.[12-14] Another longitudinal study, however,

Key Points

- Self-reported hearing impairment is a robust risk factor for the increased incidence of all-cause dementia in adults aged 75 and older.
- There is no excess risk or risk compensation through the additional presence or absence of visual impairment.
- Early prevention measures for hearing impairment might help reduce the long-term risk of dementia.

Why Does this Paper Matter?

We bolster growing evidence that early treatment of modifiable risk factors of dementia such as hearing impairment significantly contributes to reducing the burden of disease in adults aged 75 and older.

found no covariate-adjusted independent effect of the dual sensory impairment on cognitive decline.[15]

Another limitation that all previous studies have in common is that they did not consider the cumulative risk of mortality when assessing the longitudinal risk of dementia. However, it is essential to consider competing events when analyzing survival data in older individuals.[16] In particular, mortality can occur when monitoring long-term changes in older people. This competing event, death, impedes the occurrence of the event of interest, dementia. Failing to consider the competing risk of mortality will likely overestimate the absolute risk of dementia and may bias the association with sensory impairments among older adults.[17]

In sum, no firm conclusions can currently be drawn on the impact and interaction of HI and VI on the risk of dementia. On the one hand, the combination of HI and VI could be associated with an excess risk of dementia. On the other hand, compensatory effects could exist, in the sense that the impact of the impaired sensory modality is attenuated by the unimpaired other sensory modality.[18] The present study aimed to systematically examine independent and interaction effects of HI and VI on incident all-cause dementia. In particular, we investigated (1) whether HI and VI were significant individual risk factors of incident dementia when adjusting for cumulative mortality, (2) whether HI and VI were independent risk factors of dementia, controlling for one another and other known dementia-related risk factors, and (3) whether there was an interaction of HI and VI on dementia in the sense of an excess risk or risk compensation.

METHODS

Study design and sample

The sample was taken from the platform "Healthy Aging: Gender-specific trajectories into the latest life" (AgeDifferent.de), which pooled data from two prospective German old-age cohorts. The population-based LEILA75+ study[19] sampled community-dwelling participants aged 75 years and older from the local registry of Leipzig, and included five follow-ups scheduled every 18 months and a final sixth follow-up five years after the fifth follow-up. The multicenter AgeCoDe/AgeQualiDe studies[20,21] collected data from a cohort of dementia-free primary-care patients of age 75 years and older in six German cities (Hamburg, Bonn, Düsseldorf, Leipzig, Mannheim, and Munich). AgeQualiDe is a continuation and extension of the AgeCoDe study, such that the AgeQualiDe baseline corresponds to AgeCoDe study follow-up seven. A total of nine follow-up assessments of the AgeCoDe/AgeQualiDe studies have been carried out at an 18-month interval. Written informed consent was obtained from all individuals before participation in the original cohort studies. The studies were further approved by the local ethics committees.

The present study used pooled data from baseline and all follow-up assessments of both cohorts, spanning a 20-year observation period from 1997 to 2017 (Figure 1). The overall baseline sample included data from $N = 5019$ participants ($n = 1692$ from LEILA75+, $n = 3327$ from AgeCoDe/AgeQualiDe). For analysis, participants with incomplete baseline interviews ($n = 427$), a dementia diagnosis ($n = 291$), missing information on covariates at baseline ($n = 68$), or without at least one follow-up assessment ($n = 736$) were excluded. The resulting analytical sample comprised 3497 participants (69.7%) at baseline; a flowchart of sample selection is shown in Figure 2.

Dementia incidence

Assessment of dementia was based on the *S*tructured *I*nterview for the *D*iagnosis of dementia of the *A*lzheimer type, *M*ultiinfarct dementia, and dementia of other etiology (SIDAM).[22] It contains a test battery with standardized tasks to evaluate several cognitive domains,

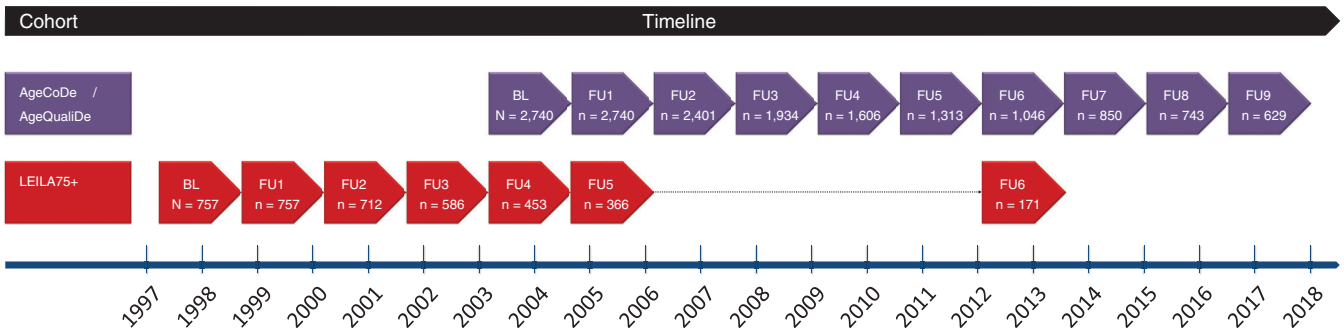

FIGURE 1 Timeline of study waves and corresponding analytical sample sizes by cohort

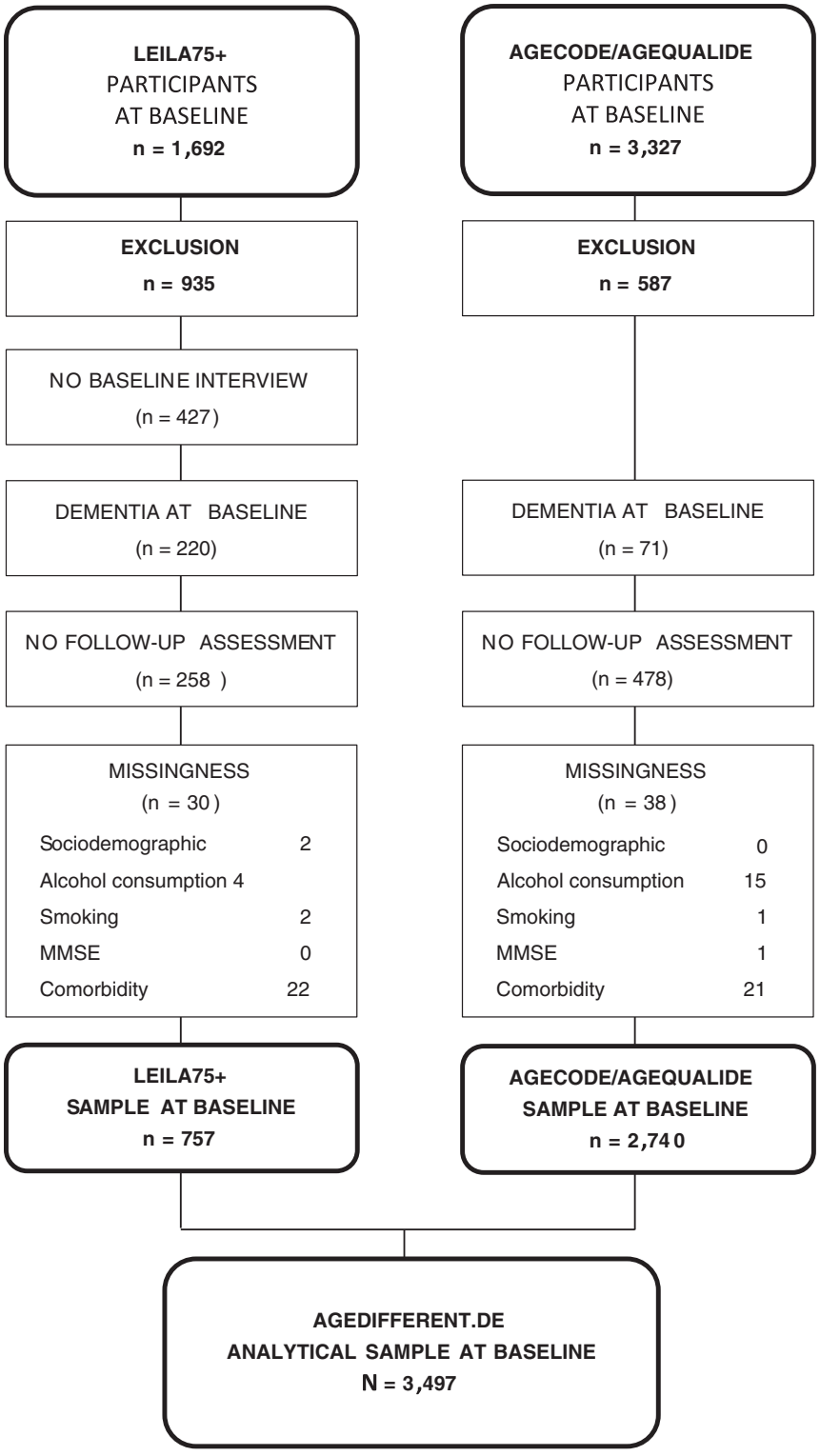

FIGURE 2 Flowchart of analytical sample at baseline

including the 30 items of the Mini-Mental State Examination (MMSE).[23] Dementia was diagnosed according to DSM-IV (Diagnostic and Statistical Manual of Mental Disorders; 4th ed.) criteria in the AgeCoDe/AgeQualiDe studies and according to DSM-III-R criteria in the LEILA75+ study, both of which are implemented as standardized diagnostic algorithms in the SIDAM. If SIDAM results were unavailable, we assigned dementia diagnoses in the AgeCoDe/AgeQualiDe studies when ratings were ≥4 on the Global Deterioration Scale[24] and/or >8 on the Blessed Dementia Rating scales.[25] In the LEILA75+ study, the Clinical Dementia Rating Scale[26] was used to obtain dementia diagnoses when SIDAM could not be assessed. All interviews were conducted face-to-face by

trained research assistants. In both cohorts, dementia diagnoses were finally validated in conferences of interviewers with geriatric experts (psychologists, geriatricians, or geriatric psychiatrists), yielding a consensus diagnosis for each incident dementia case.

Sensory impairment

In this study, we defined impairment as the individual's experience of limitations in sensory functioning, based on the self-reported grade of performance in hearing/seeing assessed at the baseline interview. In particular, we assessed HI by asking participants "Do you have difficulty hearing?," and performance should be rated on a grading scale consistent with the WHO[27] proposed rating classification for HI: (1) no impairment, (2) slight impairment, (3) moderate impairment, and (4) severe/profound impairment. An equivalent self-report measure was used for grading VI. Trained interviewers additionally validated the participants' self-assessment of HI and VI. Since less than 2% and less than 4% reported at least moderate impairment in hearing and seeing, respectively, responses 2–4 were collapsed into one category to form binary indicators of HI and VI.

Covariates

Baseline information on several covariates was collected to control for possible confounding effects. Socioeconomic characteristics were gender, age, years of education (<10 years vs. ≥10 years), and marital status (married vs. unmarried, divorced or widowed). We further included indicators of substance use since it is known to contribute to dementia.[3] First, individuals were classified as current smokers, ex-smokers, or non-smokers. Second, we estimated the prevalence of current alcohol consumption as drinking at least once a week. The sum score of the MMSE was included to control for the baseline level of cognitive functioning. In addition, we assessed depression symptoms with the German versions of the Center for Epidemiologic Studies Depression Scale (CES-D)[28] in LEILA75+ and the Geriatric Depression Scale (GDS-15)[29] in AgeCoDe/AgeQualiDe. We used established cutoff scores (CES-D score ≥ 23; GDS-15 score ≥ 10) to indicate clinically relevant depression.[30] Finally, we considered several indicators of comorbidity known to be associated with dementia, that is, cardiac diseases, stroke or transient ischemic attack (TIA), and diabetes mellitus. Medical diagnoses were obtained from structured interviews with proxy informants (LEILA75+) and from standardized questionnaires completed by the participants' GPs (AgeCoDe/AgeQualiDe).

Statistical analyses

Differences in sensory impairment and covariates between individuals who progressed/did not progress to dementia during follow-up were tested using Pearson chi-square tests (nominal) or Wilcoxon two-sample tests (ordinal or interval). Main and interaction effects of sensory impairments on the incidence of dementia were evaluated using competing risk (CR) regression models. These models are an extension of the standard approach for analyzing survival data (e.g., using Cox models), with the advantage that the accumulated competing risk of mortality over time is additionally taken into account when estimating failure probabilities.[16,17] Results of CR models were expressed as subdistribution hazard ratios with corresponding 95% confidence intervals, which can be interpreted similarly to hazard ratios in Cox regression.

To systematically test the effect of HI and VI on the incidence of dementia, we tested four consecutive models, each of which adjusting for gender and baseline information on age, marital status, school education, alcohol consumption, smoking status, cognitive functioning, cardiac diseases, stroke/TIA, depression, and diabetes mellitus. First, we evaluated the individual risk of any sensory impairment by testing the effect of HI on the incidence of dementia without considering VI (model 1), and vice versa (model 2). Next, we estimated the effects of HI and VI simultaneously to test whether the effects are independent of each other (model 3). Finally, we examined a model incorporating the interaction of HI and VI with dementia to test whether the effect of one impairment is modified by the presence/absence of the other impairment (model 4). We report the "relative excess risk due to interaction" (RERI) to evaluate the strength of the complementary associations of both forms of sensory impairment. All analyses have been performed in Stata 16.0 SE (StataCorp LP, College Station, TX).

RESULTS

Descriptive statistics

Of the $N = 3497$ individuals included in the analyses at baseline, $n = 902$ (25.8%) developed dementia during follow-up. This corresponds to an incidence rate of 37.4 (95% CI 32.7–44.1) cases per 1000 person-years. The average time from baseline to dementia onset was 5.5 years (SD 3.4), while the mean follow-up time among all individuals was 7.1 years (SD 4.0). Baseline characteristics of the analytical sample by the incidence of dementia status are shown in Table 1. The mean age of participants was

TABLE 1 Distribution of sociodemographics, cognitive functioning, substance use, and comorbidity at baseline by incident dementia at follow-up

	Total	Incident dementia			
		Yes	No		
	N = 3497	*n* = 902	*n* = 2595	Chi-square/z	*p*
Gender, *n* (%)					
Female	2349 (67.2)	662 (73.4)	1687 (65.0)	21.33	<0.001
Age, *mean* (SD)	79.8 (3.9)	81.0 (4.1)	79.4 (3.7)	−10.20	<0.001
Marital status, *n* (%)					
Unmarried/divorced/widowed	2105 (60.2)	606 (67.2)	1499 (57.8)		
Married	1392 (39.8)	296 (32.8)	1096 (42.2)	27.78	<0.001
School education, *n* (%)					
<10 years	2290 (65.5)	599 (66.4)	1691 (65.2)		
≥10 years	1207 (34.5)	303 (33.6)	904 (34.8)	0.46	0.498
MMSE, *mean* (SD)	27.5 (1.9)	26.8 (2.1)	27.7 (1.7)	11.85	<0.001
Smoking status, *n* (%)					
Non-smoker	2186 (62.5)	593 (65.7)	1593 (61.4)		
Ex-smoker	1063 (30.4)	256 (28.4)	807 (31.1)		
Current smoker	248 (7.1)	53 (5.9)	195 (7.5)	6.19	0.045
Alcohol consumption, *n* (%)					
No current drinker	1727 (49.4)	492 (54.6)	1235 (47.6)		
Current drinker	1770 (50.6)	410 (45.5)	1360 (52.4)	12.95	<0.001
Cardiac diseases, *n* (%)					
Yes	1359 (38.9)	347 (38.5)	1012 (39.0)	0.08	0.779
Stroke/TIA, *n* (%)					
Yes	313 (9.0)	113 (12.5)	200 (7.7)	19.09	<0.001
Depression, *n* (%)					
Yes	141 (4.0)	44 (4.9)	97 (3.7)	2.25	0.134
Diabetes mellitus, *n* (%)					
Yes	767 (21.9)	212 (23.5)	555 (21.4)	1.75	0.186

Note: Pearson chi-square tests (nominal) or Wilcoxon two-sample tests (ordinal or interval) were used for bivariate comparisons between individuals with/without incident dementia.

Abbreviations: MMSE, Mini-Mental State Examination (score range: 0–30).

79.8 years (SD 3.9); 67.2% were female. Older and unmarried, divorced, or widowed individuals, females, and those with worse cognitive functioning or reports of stroke/TIA more frequently developed dementia during follow-up. In contrast, smoking and alcohol consumption were less frequently associated with later dementia.

Sensory impairment

A total of 30.3% of individuals reported HI at baseline, and 16.6% reported VI (Table 2). A combination of HI and VI was reported by 6.5%. Both forms of sensory impairment were more frequently reported by those who progressed to dementia during follow-up (HI 35.7% vs. 28.5%, $p < 0.001$; VI 19.0% vs. 15.7%, $p = 0.024$).

Effects of sensory impairment on incident dementia

Results of the CR models associating sensory impairment with incident dementia while controlling for the fact that mortality may also occur are shown in Table 3. Adjusting for all covariates, HI (model 1: sHR 1.16, 95% CI 1.04–1.30, $p = 0.011$) but not VI (model 2: sHR 1.07, 95% CI 0.90–1.28, $p = 0.462$) was significantly associated with incident dementia. The same was true when considering

TABLE 2 Distribution of hearing and visual impairment at baseline by incident dementia at follow-up

	Total	Incident dementia		Chi-square	*p*
		Yes *n* (%)	No *n* (%)		
HI					
No	2436 (69.7)	580 (64.3)	1856 (71.5)		
Yes	1061 (30.3)	322 (35.7)	739 (28.5)	16.51	<0.001
VI					
No	2918 (83.4)	731 (81.0)	2187 (84.3)		
Yes	579 (16.6)	171 (19.0)	408 (15.7)	5.07	0.024
HI and VI					
No	3268 (93.5)	823 (91.2)	2445 (94.2)		
Yes	229 (6.5)	79 (8.8)	150 (5.8)	9.70	0.002

Note: Pearson chi-square tests were used for bivariate comparisons between individuals with/without incident dementia.
Abbreviations: HI, hearing impairment; VI, visual impairment.

TABLE 3 Results of competing risk regression analyses of hearing and visual impairment at baseline and their interaction on incident dementia at follow-up

	Model	sHR	(95% CI)	Wald	*p*
Individual risk					
HI only	I	1.16	(1.04, 1.30)	6.55	0.011
VI only	II	1.07	(0.90, 1.28)	0.54	0.462
Independent risk					
HI	III	1.16	(1.03, 1.30)	6.07	0.014
VI		1.06	(0.89, 1.26)	0.38	0.538
Combined risk					
HI	IV	1.14	(1.00, 1.30)	3.74	0.053
VI		1.02	(0.82, 1.26)	0.03	0.862
HI × VI		1.09	(0.81, 1.46)	0.33	0.567

Note: Models I and II included HI and VI only, respectively. Model III included both HI and VI. Model IV included HI, VI and an interaction term between HI and VI. Each model additionally adjusted for gender and baseline information on age, marital status, school education, alcohol consumption, smoking status, cognitive functioning, cardiac diseases, stroke/TIA, depression, and diabetes mellitus.
Abbreviations: HI, hearing impairment; sHR: subdistribution hazard ratio; VI, visual impairment.

both risk factors simultaneously (model 3). In the interaction model, the main effects of HI and VI were not statistically significant (model 4: HI: sHR 1.14, 95% CI 1.00–1.30, $p = 0.053$; VI: sHR 1.02, 95% CI 0.82–1.26, $p = 0.862$). In addition, the interaction of HI with VI was also not significant (sHR 1.09, 95% CI 0.81–1.46, $p = 0.567$), indicating that the effect of one impairment was not modified by the presence of the other impairment. Likewise, the RERI was estimated at 0.11 (95% CI −0.23–0.45, $p = 0.536$), suggesting that the combined effect is not significantly different from the sum of the individual effects.

DISCUSSION

Our results showed that self-reported HI but not VI was significantly associated with incident dementia when adjusting for a wide range of dementia-related covariates and mortality. The interaction of HI and VI did not significantly predict incident dementia beyond the individual risks.

Self-reported HI appeared to be a major risk factor of dementia, independent of sociodemographic and other health-related factors. This finding corresponds with the results of previous longitudinal studies using objective measures of HI.[31,32] Moreover, there is evidence of a predictive effect of HI on cognitive functioning when adjusting for VI.[33] Our study extends these earlier results by focusing specifically on incident dementia, including particularly individuals aged 85 and older, and accounting for the accumulated risk of mortality.

While individuals who later developed dementia reported VI more frequently, there was no covariate-adjusted effect on incident dementia. Similar findings were reported in a large population-based study from the United Kingdom among individuals aged 70 and older.[11] The authors argued that older individuals may have spent more time treating their VIs than younger individuals, making these impairments less relevant. The timely use of glasses is generally more common when getting older than the use of hearing aids, which can lead to early compensation of VIs.[34]

Interestingly, the insignificant interaction between HI and VI suggested that the risk of sensory impairment in one

domain does not multiply with the risk of sensory impairment in the other domain. In other words, there is no evidence for an excess risk of incident dementia in those reporting both forms of sensory impairment compared to those reporting one of these impairments only. Moreover, while compensatory effects were assumed in previous research,[18] the conditional effects of HI and VI in the interaction model were not significantly associated with incident dementia either. This means that the lack of sensory impairment in one domain did not compensate for the risk of dementia attributable to the impairment in the respective other domain. Overall, it can be concluded that HI was the only stable sensory risk factor of dementia. Moreover, this risk was neither increased by impaired vision, nor was it reduced by functioning vision. Therefore, early treatment of HI and hearing loss appears to be essential to prevent dementia.

Strengths of the present study include the large sample of older adults who provide longitudinal data for an observation period of 20 years. The use of structured clinical interviews with additional consensus conferences to obtain dementia status is another advantage of the LEILA75+ and AgeCoDe/AgeQualiDe cohort studies. In addition, using CR regression models allowed us to adjust for cumulative risk of mortality. This is an important concern in survival analyses and considered to yield more accurate risk associations with dementia.[17] The present study also has limitations. First, we used self-reported data to assess HI and VIs which could underestimate the prevalence of actual impairments and limit comparability with studies using objective measures. In particular, we did not use audiometric or ophthalmic measurements to evaluate participants' ratings of sensory impairments. Yet there is evidence suggesting good agreement between self-reported and objective measures of vision and audition.[35] Second, we did not consider the causes of sensory impairment, the time of onset, and the use of hearing or visual aids, as no data on these issues was available. In conclusion, we could not differentiate between sensory impairment ratings from participants who used or did not use compensating aids. Finally, we did not take into account possible changes in sensory impairments over time. However, sensitivity analyses revealed that reports on impairments were quite stable over time and changes had no substantial impact on incident dementia. Although we consider the found associations valid and reliable, we cannot completely rule out undiscovered mechanisms between increasingly declining sensory performance and longitudinal dementia.

Our results suggest that HI, but not VI, is a robust and independent risk factor for dementia in older adults. This finding has important implications for the prevention of dementia. While hearing loss is widespread in older adults and is now considered one of the main causes of years lived with disability worldwide,[36] 2019 WHO guidelines do not give any recommendations for the use of hearing aids as a means to reduce the risk of cognitive decline and dementia.[1] This is most likely due to the fact that the underlying biological mechanism that links HI to dementia is not entirely clear so far and needs further research.[3] Nonetheless, prevention efforts for dementia should focus on the benefits of healthcare solutions available for HI. In particular, multicomponent interventions targeting modifiable risk and protective factors of dementia should include treatment of HI.[37] At the global public health level, prevention efforts should target particularly low- and middle-income countries, which have the highest burden of HI and dementia.[1,36]

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CONFLICT OF INTEREST

None.

AUTHOR CONTRIBUTIONS

Study concept and design: Pabst, Bär, Maier, Wagner, Wiese, König, Riedel-Heller.

Acquisition of data: all.

Analysis and interpretation of data: Pabst, Bär, Riedel-Heller.

Drafting the manuscript: Pabst, Bär.

Critical revision of the manuscript for important intellectual content: all.

Final approval of the version to be published: all.

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