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A clinical perspective: untreated hearing loss and cognitive decline

Abstract

Untreated hearing loss has been recognized as a potentially modifiable risk factor for cognitive decline. Research demonstrates that many "at-risk" adults with untreated hearing loss experience earlier onset and faster cognitive deterioration ("at-risk" as used here includes people with additional risk factors such as older adults, people with greater hearing loss, people with cardiovascular disease, diabetes, and/or polypharmacy issues, etc). Auditory decline begins decades before it is detected by conventional hearing tests. Sub-clinical changes such as cochlear synaptopathy, speech-in-noise problems, and extended high-frequency loss can disrupt auditory–cognitive processing even when conventional hearing thresholds from 250 to 8000 Hz appear normal. Early symptoms like tinnitus and difficulty understanding speech in noise may signal underlying neural vulnerability. Sub-clinical (not visible on a conventional hearing test) auditory deficits may increase cognitive load, as the brain must work harder to process degraded sound. Over time, this sustained effort may reduce neural efficiency and may contribute to cognitive maladaptation and may accelerate atrophy in brain regions involved in memory and executive function. Social isolation, anxiety, depression, and reduced communication are common consequences of untreated hearing loss, each of which may compound cognitive risk. Emerging evidence suggests that hearing loss is not only a marker but may also be a mechanism of cognitive decline. Degradation of speech-in-noise performance and the presence of tinnitus are associated with increased dementia risk and may serve as early biomarkers. Intervention studies indicate that hearing aids may slow cognitive decline in at-risk patients, with trials like ACHIEVE showing nearly a 50% reduction in cognitive decline over three years. Overall, early detection via comprehensive auditory testing (not hearing screenings) is critical. Diagnosing and treating hearing loss promptly (mid-life) may preserve cognitive function, may reduce dementia risk, and may support long-term brain health before the negative consequences of long-term untreated hearing loss appear.

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Introduction

Hearing loss is the most prevalent sensory impairment.[1,2] In recent years, there has been significant attention given to the correlation of untreated hearing loss in adults and an increased risk of cognitive decline. Multiple outcomes-based correlational studies have indicated that for many people at risk of cognitive decline, hearing aid amplification may be beneficial regarding slowing or reducing cognitive decline.[3–10] Age-related hearing loss (ARHL) and the consequences of ARHL (i.e., social isolation, lack of communication ability, anxiety, depression etc) are increasingly recognized as contributors to cognitive decline and dementia. Large epidemiological studies demonstrate correlations between untreated hearing loss and accelerated cognitive decline in at-risk individuals. At-risk people includes those who are older, have less education, have greater degrees of hearing loss, have co-morbidities (diabetes, pre-diabetes, cardiovascular issues etc.) and/or poly-pharmacy issues.[6,13–18]

Age-related auditory decline (i.e., presbycusis) begins years or decades before conventional threshold-based measures (i.e., 250 to 8000 Hz) indicate hearing impairment. Sub-clinical auditory pathology, including cochlear synaptopathy (the loss of synapses between inner hair cells and auditory nerve fibers) and extended high-frequency threshold elevation may disrupt auditory–cognitive integration despite normal conventional pure-tone sensitivity. Functional symptoms such as tinnitus, difficulty understanding speech in noise and reduced working memory capacity frequently precede conventional threshold loss and may reflect early cochlear or neural dysfunction within auditory and attentional networks.[19–29]

Mid-life detection of sub-clinical (i.e., not detectable via conventional threshold measures) auditory anomalies allows timely diagnosis and treatment, prior to the consequences of untreated hearing loss, including anxiety, depression, social isolation, attenuated communicative ability, and possible cognitive decline.

Unfortunately, mid-life audiometric changes are unlikely to be detected via conventional audiometry or hearing screenings which typically assess only loudness thresholds across a limited range of pure-tone frequencies. To identify and treat auditory deficits, comprehensive audiometric evaluations are required, including extended high frequency assessment, speech in noise testing, otoacoustic emissions, acoustic reflexes, and tympanometry.[30,31] A thorough medical case history is essential, as cardiovascular disease, diabetes, smoking, traumatic brain injury, and other common health conditions are independently associated with increased risk of hearing loss.[32–34] Validated self-report instruments such as the Hearing Handicap Inventory for the Elderly[35] and the Tinnitus Handicap Inventory[36] further quantify the functional and psychosocial impact of auditory symptoms which conventional pure-tone thresholds alone cannot capture.

Auditory decline as an early signal of cognitive risk

Hearing loss has been identified among the most significant potentially modifiable risk factors for dementia.[5,37–40] Longitudinal cohort studies and meta-analyses demonstrate that at-risk individuals with untreated hearing loss often experience earlier onset and more

rapid progression of cognitive decline compared to those with normal hearing or those treated with amplification.[4,6,41] Although these correlations are well-established, the mechanistic pathways linking auditory dysfunction to cognitive decline appear multifactorial, involving neural, cognitive, and psychosocial processes.[42–44]

Many common conditions in the aging population, including cardiovascular disease, diabetes, polypharmacy, and prior traumatic brain injury are independently associated with increased risk of hearing loss, suggesting that auditory decline may emerge earlier and progress more rapidly in medically complex individuals. Clinicians managing these conditions should recognize hearing loss as a potentially early manifestation of systemic disease and as an independent contributor to cognitive burden. Untreated hearing loss increases demand on attention, working memory, and executive function, resources which may already be compromised in medically vulnerable populations.

Processing degraded, distorted, and attenuated auditory input requires increased and sustained engagement of cognitive resources, often increasing cognitive load. Over time, increased listening effort can reduce neural efficiency and may promote cognitive maladaptation[45] such that erroneous, attenuated and/or distorted incoming sensory signals interfere with previously established cognition, knowledge and neural pathways. Neuroimaging studies demonstrate that hearing loss is associated with reduced gray matter in auditory and temporal regions, alterations in cortical organization, and changes in functional connectivity within networks supporting memory and executive function.[18,46–48]

These findings suggest that auditory system integrity plays a foundational role in maintaining cognitive resilience, and that early auditory decline may serve as both a marker and a mechanism of broader vulnerability.

Sub-clinical auditory decline

Age-related auditory decline does not begin with the onset of traditionally measured hearing loss. Degenerative changes in the auditory system may start as early as the pre-teenage years[49] and population-based data indicates that the majority of adults who report hearing difficulty identify symptom onset between the ages of 30 and 59[50] with extended high-frequency threshold elevation detectable as early as the late 20s.[51,52] During this sub-clinical stage, the cochlea and auditory nerve undergo progressive neural degeneration, beginning with cochlear synaptopathy and followed by the gradual degeneration of ascending auditory nerve fibers.[53]

These changes significantly degrade the quantity and quality of acoustic information transmitted to the brain. Despite the loss of up to 70-80% of auditory nerve fibers, conventional audiometric thresholds may remain normal.[54,55] However, temporal resolution, extended high-frequency sensitivity, and word recognition in noise tend to progressively deteriorate.[20,56] Indeed, middle-aged individuals with normal conventional audiograms frequently report difficulty understanding speech in noise, such as following conversations in restaurants, social gatherings, and group settings, and/or they experience tinnitus.[57,58] These functional deficits, frequently termed 'hidden hearing loss,' are not captured by conventional pure-tone audiometry or hearing screenings, all of which underscore the need for comprehensive audiometric evaluations.

Functional auditory symptoms as early biomarkers of cognitive decline

Functional symptoms of sub-clinical ARHL may serve as early indicators of neural vulnerability to cognitive decline.

Tinnitus has been associated with abnormal neural activity across distributed auditory, attentional, and limbic brain networks. Neuroimaging studies demonstrate that tinnitus involves hyperactivity in auditory pathways and altered connectivity in limbic structures, attention networks, and the default mode networks, suggesting that chronic tinnitus may function as a biomarker of broader vulnerability, rather than an isolated auditory symptom.[59–61] Meta-analytic and population-based evidence confirms that tinnitus is associated with elevated risk of dementia and Alzheimer's disease.[62,63]

Speech-in-noise difficulty similarly reflects impaired neural encoding and reduced efficiency of communication between sensory and cognitive processing systems. Understanding speech in background noise requires peripheral auditory integrity and engagement of higher-order cognitive processes including attention, working memory, and executive control.[64]

Large population-based studies demonstrate that impaired speech-in-noise performance is independently associated with increased dementia risk. In the UK Biobank study ($n \approx 82{,}039$, 11-year follow-up), poor speech-in-noise ability was associated with a 61% hazard ratio increased risk of incident dementia compared to those with normal performance,[65] reinforcing the view that processing deficits may serve as observable biomarkers of neural strain across auditory, attentional, and cognitive networks.

Auditory deprivation, cognitive load, and vulnerability in aging

Several complementary mechanisms have been proposed to explain how auditory dysfunction may contribute to cognitive decline. Degraded auditory input increases cognitive load by diverting attentional and executive resources towards perceptual processing, reducing resources available for higher-order cognitive processes such as memory encoding and executive function.[64,66] Chronically increased listening effort may promote cognitive fatigue and maladaptive neural reorganization, particularly when compounded by sensory deprivation. Reduced afferent auditory input alters cortical organization and network connectivity, promoting cross-modal reorganization and functional changes within attention, memory, and executive networks. These neuroplastic changes may be adaptive or maladaptive, as proposed through the Cognitive Maladaptation Hypothesis.[45] Consistent with maladaptive reorganization, neuroimaging studies demonstrate accelerated brain atrophy in auditory and temporal regions among individuals with hearing loss.[46–48]

The cognitive consequences of auditory deprivation are not likely to be uniform across individuals. Those with pre-existing vulnerabilities, including cardio-vascular and metabolic disease, dual-sensory impairment,[67] polypharmacy, traumatic brain injury, or chronic neuropsychiatric conditions, often exhibit diminished cognitive reserve. In these populations, increased listening effort and auditory-related neural stress may interact synergistically with vascular, inflammatory, or neurodegenerative processes, accelerating network-level dysfunction.[14,18,68] From a clinical perspective, hearing

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loss and tinnitus may function as independent contributors to cognitive risk, and also as amplifiers of vulnerability in populations already burdened by co-morbid disease.

Hearing loss, dementia risk, and the importance of early intervention

Given the established role of hearing loss as a leading potentially modifiable risk factor for dementia, attention has increasingly turned to whether timely audiologic intervention can mitigate cognitive decline. Longitudinal observational data demonstrates that untreated hearing loss is often correlated with increased dementia risk and hearing aid use may be correlated with reduced or delayed cognitive decline.[3,4,41,69] Emerging longitudinal evidence suggests that earlier adoption of hearing intervention (i.e., diagnosis and treatment) in midlife may confer greater cognitive benefit among at-risk individuals.[37,41,70]

Beyond observational data, intervention studies provide converging evidence that amplification may slow cognitive decline in some individuals at-risk. The ACHIEVE trial demonstrated a 48% reduction in 3-year cognitive decline in at-risk adults.[6] The ENHANCE study similarly showed cognitive stability over 3 years in hearing aid users compared to age-matched controls.[7] Meta-analytic data further support these findings with hearing aid use associated with a 19% reduction in long-term cognitive decline risk[8] and potential benefit (i.e., delayed reduction) across global cognition, memory and executive function domains.[12]

Collectively, these findings reinforce the clinical imperative for timely (i.e., mid-life) hearing intervention, which may slow cognitive decline while preserving communication ability, social connectedness, and functional independence, all of which contribute to cognitive resilience in aging adults.[71,72]

Conclusion

Untreated hearing loss is among the most significant potentially modifiable risk factors for cognitive decline and dementia. Converging evidence demonstrates that timely diagnosis and treatment with amplification may slow this trajectory. Framing hearing loss as a chronic sensory disorder with downstream neurobiological consequences reframes early detection as a clinical imperative. Early identification and treatment of auditory decline represents an actionable clinical strategy for preserving cognitive resilience and reducing dementia risk across the lifespan.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

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