REVIEW

Sensorineural Hearing Loss: Impact on Cognitive Function and Emotional well-being

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Abstract. This paper explores the impact of sensorineural hearing loss on cognitive function and emotional well-being, highlighting the complex connections between hearing loss, cognitive decline, and depressive disorders. Hearing loss, particularly in older age, is often a symptom of progressive neurodegenerative dysfunction and not just an isolated condition, with significant consequences for mental and cognitive health. Recent research indicates that hearing loss is related to a heightened risk of dementia and depression. Interventions - especially through the early use of hearing aids - appear to mitigate cognitive decline. The relationship is multifaceted, comprising age-related factors and structural brain alterations. Additionally, hearing loss in older adults is closely connected with depressive symptoms, and auditory aids have been proven to enhance cognitive performance and emotional health. Ultimately, the paper emphasizes the relevance of early diagnosis and intervention strategies for managing the disruptive effects of hearing loss on cognition and emotional health.

Keywords: *sensorineural hearing loss; presbycusis; cognitive decline; depression; cochlear implants.*

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INTRODUCTION

Sensorineural hearing loss (SNHL) constitutes the most frequent type of hearing impairment, with presbycusis—defined by bilateral, high-frequency hearing loss related to aging—being the most common variant. Epidemiological data suggest that hearing loss affects roughly one in four individuals above the age of 60, approximately 50% of those over 75, and 80% of adults older than 85 [1,2]. The World Health Organization states that, at a global level, more than 430 million people live with severe hearing loss, and this number will increase to 2.5 billion by 2050, including over 700 million individuals who will require hearing-related interventions. The estimated annual economic burden exceeds 1 trillion USD. Despite its high prevalence, hearing loss often remains undiagnosed and undertreated, especially in older populations [3].

The broader impact of hearing loss extends beyond auditory perception, affecting communication, social interaction, and cognitive function. This was eloquently expressed by Helen Keller, who, having been both deaf and blind from infancy, observed: “Blindness separates people from things; deafness separates people from people” [4]. Though anecdotal, her reflection captures a key insight supported by empirical research: hearing loss is associated with isolation, depression, and cognitive depreciation [5-7]. These effects are particularly significant in presbycusis, where peripheral degeneration

(cochlear hair cell loss, auditory nerve dysfunction) is often compounded by central hearing alterations in processing [8,9].

The link between hearing loss and cognitive health is multifactorial and operates in both directions, encompassing mechanisms such as auditory deprivation, heightened mental effort, neuroanatomical changes, and decreased social interaction [10]. Importantly, even slight to moderate degrees of hearing impairment have been associated with an elevated risk of developing dementia and mood disorders [11]. These understandings point out the need for diagnosis and intervention, especially in aging populations.

Standard evaluation relies on pure-tone audiometry, yet patients may present with disproportionate speech perception deficits, indicating central auditory dysfunction. Additional tools such as speech-in-noise testing, dichotic listening, and electrophysiological measures provide a more comprehensive assessment [7].

This review explores the anatomical, functional, and psychosocial pathways connecting sensorineural hearing loss to neurocognitive and affective outcomes and summarizes current evidence on diagnostic and therapeutic approaches.

ANATOMICAL AND FUNCTIONAL PATHWAYS

The auditory pathway starts when sound waves are directed by the outer ear and set the tympanic membrane and ossicles in motion, transmitting mechanical energy to the cochlea. Within the cochlea, this energy is transformed into electrical signals by sensory hair cells. These signals travel through the auditory nerve and reach the brainstem, where they are first processed by the cochlear nuclei and the superior olivary complex. The information then ascends to the midbrain's inferior colliculus, passes through the medial geniculate bodies (part of the thalamus), and afterwards reaches the primary auditory cortex (the superior temporal gyrus/ Heschl's gyrus) [12,13].

The primary auditory cortex is responsible for decoding basic auditory features like frequency, intensity, and timing. Information is then relayed to higher-order cortical regions, including the medial temporal lobe, where semantic interpretation and sound recognition occur. Prefrontal areas integrate emotional and contextual aspects of auditory stimuli, while the precuneus and hippocampus contribute to episodic memory, self-referential processing, and associative meaning [6,14].

Recent findings highlight that auditory perception relies not only on the primary auditory cortex but also on a broader network of cortical regions (Figure 1). Among these are multimodal integrative areas such as the dorsolateral prefrontal cortex, posterior cingulate, and parietal lobes, which contribute to the coordination of auditory input with visual, motor, and affective information—facilitating the construction of complex, contextually meaningful representations [10,14,15].

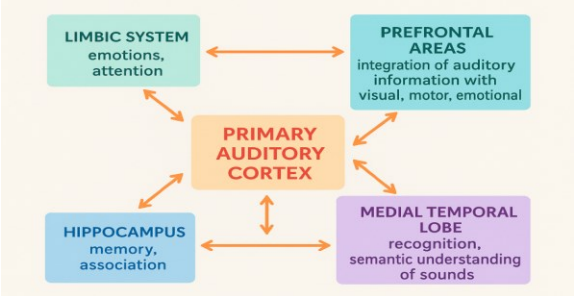

Figure 1. Diagram of the primary auditory cortex and its connections. Bidirectional arrows show its links to the limbic system, precuneus and parietal areas, prefrontal areas, hippocampus, and medial temporal lobe.

CLINICAL MANIFESTATIONS

SNHL, including presbycusis, is increasingly understood not merely as an isolated auditory condition but as part of a broader neurodegenerative process. While the term "hearing loss" typically refers to elevated auditory thresholds (>25 dB HL), this reflects only one component of a more complex clinical picture involving both peripheral and central auditory pathways (table 1) [6,10,16].

Auditory Manifestations. Patients often experience reduced speech clarity - especially in noisy environments - due to high-frequency consonant loss. Tinnitus affects ~40% of individuals and may involve phantom-like perceptions, with pain reported in ~20%. Some also report sound intolerance or recruitment, a hypersensitivity to loud sounds [7,17].

Cognitive and Emotional Effects. SNHL imposes increased cognitive load, diverting resources toward auditory processing and leading to working memory strain and reduced cognitive reserve [1,9]. These factors may increase vulnerability to dementia and depression in later life, particularly among individuals experiencing social isolation, bothersome tinnitus, or impairments of the vestibular system [7,11].

Social and Behavioral Changes. Diminished hearing often leads to avoidance of social interactions, particularly in noisy settings, resulting in isolation and reduced participation in community activities. These effects are more pronounced in institutional settings and may differ by gender [2].

Motor and Frailty Components. Vestibular deficits, present in ~20% of SNHL cases, increase the risk of falls, slower gait, and physical frailty. Fear of falling further limits activity, promoting sedentarism and muscular deconditioning [18,19].

Table 1. Summary of clinical domains and manifestations associated with SNHL.

DOMAIN	MANIFESTATIONS
Auditory	Elevated hearing thresholds, reduced sound clarity, presence of tinnitus, abnormal loudness perception
Cognitive/Emotional	Increased working memory demands, diminished cognitive reserve, heightened risk of dementia and depression
Social	Isolation, low engagement, reduced communication
Motor/Frailty	Vestibular dysfunction (~20%), falls, reduced gait speed, diminished mobility

SNHL AND COGNITIVE DECLINE

A large number of studies, transversal as well as longitudinal, have highlighted a significant association between hearing loss and cognitive loss, especially in connection with dementia. For instance, the longitudinal study of aging known as Baltimore, which followed 639 individuals for 17 years, revealed a 5 times higher chance of developing dementia in individuals affected by severe SNHL compared to peers with normal hearing [1].

Complementary findings from the NHANES and Health ABC cohorts support this link, demonstrating that worsening hearing loss correlates with reduced cognitive processing speed and executive domain [11,20].

These data underscore the potential of hearing loss as a modifiable risk factor, a view echoed in the 2024 Lancet Commission Report, which identifies untreated hearing loss as accounting for approximately 7% of global dementia cases, making it the largest modifiable contributor [21]. Timely measures - such as the use of hearing aids and strategies to minimize noise-related damage - may play an important role in lowering the risk of developing dementia.

In terms of study methodology, input parameters usually involve the classification of hearing loss (HL) based on audiometric threshold averages: <25 dB HL = normal hearing; 25–40 dB = mild loss; 41–70 dB = moderate; >70 dB = severe. Cognitive outcomes are often assessed using composite z-scores derived from a battery of ≥16 standardized neuropsychological tests (e.g., MMSE, Digit Symbol, Trail Making, Stroop). Statistical analyses frequently include Pearson or Spearman correlations and multiple linear regression models adjusting for age, education, and comorbidities, with significance levels set at $\alpha = 0.05$ [11,20].

A meta-analysis synthesizes relative risk (RR) estimates from multiple cohorts, showing an overall RR of ~2.4 for incident dementia in individuals with hearing loss. Despite this strong pooled effect, heterogeneity was substantial ($I^2 \approx 81.4\%$), reflecting variability in study design, populations, and cognitive assessments [22]. Some studies (e.g., Gates, 2002; Gates, 2011) reported particularly high and imprecise RR estimates, as evidenced by wide confidence intervals [23,24].

SNHL AND DEPRESSION

Several mechanisms may explain the discrepancy in research focus between depression and cognitive decline: SNHL directly impairs language processing and memory - core components of cognition - and often induces neuroplastic changes that burden compensatory cognitive circuits, potentially accelerating cognitive decline [25]. In contrast, depression may arise indirectly, particularly through social isolation or communication difficulties.

However, studies reveal significant associations between SNHL and depressive symptoms, especially among the elderly. One meta-analysis covering 35 studies with 147,000 people showed that those with hearing loss had a 47% higher likelihood of experiencing depression (OR = 1.47) [26]. Following a similar direction, another meta-analysis of 24 cohort studies identified sensorineural hearing loss as an independent predictor of depression (OR = 1.35), with particularly elevated risks noted in cases of sudden hearing loss and studies with follow-up periods of five years or longer [27].

Other authors have highlighted a bidirectional association between SNHL and both depression and anxiety, an association shaped by age, geographic region, and diagnostic criteria used [28]. Supporting this, the Health ABC study found that individuals with moderate hearing loss (>40 dB) had a 23% reduction in emotional vitality, including higher levels of anxiety and depressive symptoms [29].

Furthermore, data from the NHANES study showed that despite hearing loss by itself not beingstrongly linked to major depression - as assessed by the 9-item Patient Health Questionnaire - individuals who used hearing aids for at least five hours daily experienced a notable decrease in depressive symptoms [30]. These findings emphasize the role of hearing interventions in improving not only auditory function but also emotional well-being in aging populations.

PATHOPHYSIOLOGICAL MECHANISMS

To clarify the potential causal pathways linking age-related hearing loss and cognitive decline,

four major hypotheses have been proposed. Each offers a distinct perspective on the directionality and mechanisms of this association, ranging from cognitive influences on perception to shared neurodegenerative etiologies [31].

Cognitive load on perception. This hypothesis states that reduced cognitive capacity may hinder auditory processing, creating the appearance of hearing impairment. Difficulties such as slower processing speed or decreased attention could limit the brain's efficiency in interpreting sound, particularly in noisy environments. However, most studies do not support the idea that cognitive decline comes before hearing loss. And those that do often use memory tests that are affected by how well someone can hear [31,32].

Information degradation. This hypothesis suggests that when sound signals are weak or unclear, the brain has to work harder just to understand speech. That extra effort takes away mental energy from other tasks like memory, focus, or reasoning. As a result, people may show signs of cognitive decline, but these effects can often be improved [33,34]. Numerous studies support this theory, showing that older adults exhibit poorer memory and higher listening effort when decoding degraded or noisy speech [35,36].

Sensory deprivation. Chronic reduction in auditory input leads to deafferentation and structural atrophy in the primary (A1) and secondary (A2) auditory cortices. These areas may undergo cross-modal reorganization, being partially recruited by visual or tactile systems, which can disrupt the normal integration of auditory and cognitive processes [20,31]. While conceptually compelling, especially given cortical reorganization in long-term auditory deprivation, the effects of hearing loss early in life appear less severe cognitively [25,37].

An illustrative case of this neuroplastic adaptation was observed during the COVID-19 pandemic: the use of face masks - while essential for public health - made it harder to see visual cues like lip reading, which many people with hearing loss rely on to help them understand speech. This shows how losing input from more than one sense - like both hearing and sight -can put extra strain on the brain and make it harder for the brain to adapt and stay organized [38-42].

Common cause. A shared underlying pathology - such as cerebral microangiopathy, chronic inflammation, or oxidative stress - may simultaneously impair auditory and cognitive paths [36,37]. Studies support the hypothesis by demonstrating simultaneous decline across multiple sensory and cognitive domains (figure 2). However, some studies indicate that hearing loss contributes independently to cognition, implying that common causes cannot entirely explain the relationship [31].

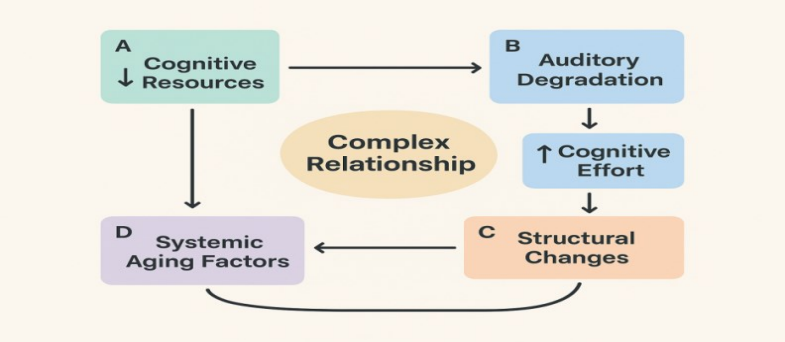

Figure 2. Illustration of the complex interplay between the four main hypotheses linking hearing loss and cognitive decline. Adapted and synthesized from Wayne & Johnsrude, 2015 [31].

THERAPEUTIC INTERVENTIONS

Multiple assistive technologies - hearing aids, and cochlear implants - act as modifiable factors in the prevention and management of dementia-related outcomes. In a longitudinal study published in JAMA Otolaryngology Head & Neck Surgery, 76% of patients who received cochlear implants reported an absence of depressive symptoms at 12 months after the procedure, compared to 59% before the procedure. Cognitive assessments revealed that before implantation, 44% of patients had abnormal global cognition scores. After 12 months, 81% improved, with most scores returning to normal [43]. Parallel findings from the ACHIEVE trial reported a nearly 50% drop in the risk of cognitive degeneration for the elderly who use hearing aids and simultaneously have other risk factors for dementia [44]. Moreover, a meta-analysis affirmed a 19% diminished risk of dementia and a 3% improvement in short-term cognition following hearing aid use [45]. These findings highlight the therapeutic relevance of hearing interventions not only in improving auditory function but also in contributing to broader neurocognitive and psychological health.

PUBLIC HEALTH PERSPECTIVE

While therapeutic interventions offer significant benefits, the best approach is to protect hearing before damage occurs - a key part of healthy aging. Hearing loss due to noise exposure remains a preventable yet under-addressed public health issue [46]. Experimental studies have shown that exposure to sounds louder than 85 dBA can result in permanent damage to the connections and nerve cells in the cochlea, even if the initial hearing loss appears to be temporary [47]. Prolonged exposure to high-intensity noise leads to cochlear synaptopathy (damage to the connections between hair cells and nerves) and eventual loss of auditory nerve fibres with implications for both auditory acuity and cognitive processing [48]. Public health campaigns such as It's a Noisy Planet, Protect Their Hearing [49] and International Noise Awareness Day [50], have emphasized that environmental sounds - such as fireworks (140–160 dBA), ambulance sirens (110–129 dBA), and personal listening devices at high volume (94–110 dBA) - can all cause hearing damage in just a few minutes. Even sounds that seem harmless, like lawnmowers (80–100 dBA), can be risky after extended exposure (≥8 hours). Preventive strategies should focus on reducing daily sound exposure through the use of hearing protection, limiting volume on personal devices, incorporating quiet breaks, and increasing awareness - particularly among children and adolescents, who are more vulnerable to long-term damage. Public education, informed by both epidemiologic and experimental evidence, is essential to reduce the future burden of auditory and cognitive impairment linked to preventable hearing loss.

CONCLUSION

Sensorineural hearing loss is no longer viewed merely as a sensory deficit but as a multifaceted clinical condition with profound implications for cognitive function and emotional well-being. A growing body of evidence supports its role as both a marker and a modifiable contributor to neurodegenerative trajectories, including dementia and depression. Through anatomical, functional, and psychosocial pathways, hearing loss burdens central neural resources, accelerates cortical reorganization, and fosters social disengagement - all of which contribute to a cognitive and affective decline in aging individuals. Crucially, timely auditory rehabilitation through hearing aids and cochlear implants has been shown to mitigate these adverse outcomes, reinforcing the need for proactive clinical screening and intervention. Moreover, the importance of prevention - especially noise exposure reduction - must not be understated, as it represents a critical, yet underutilized, strategy in safeguarding auditory and cognitive health. In light of current demographic trends and the projected global rise in age-related hearing loss, integrating auditory health into public health and dementia prevention policies is both an urgent necessity and a scientifically grounded opportunity. Continued interdisciplinary research, combining audiology, neurology, and public health, will be essential in translating this understanding into effective, equitable, and sustainable care.

Author Contributions

E.A.I. and V.S. performed the conceptual design, literature review, and drafted the initial

manuscript. C.A.S. and D.M. provided expert clinical input, revised the manuscript critically. All authors contributed equally to the interpretation of the data, final editing, and approved the final version.

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