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The cognitive maladaptation hypothesis: how sensory deprivation could contribute to cognitive decline

Abstract

This article reviews several established hypotheses explaining the possible relationship between hearing loss and cognitive decline, while suggesting a similar process may occur in other sensory systems (i.e., vision). Building on these existing frameworks, the article introduces an alternative perspective—the Cognitive Maladaptation Hypothesis. This hypothesis posits that prolonged sensory deprivation, in some cases can induce maladaptive neuroplastic changes in the brain. These changes involve the brain recalibrating to distorted or degraded sensory input, which increases the likelihood of perceptual and cognitive errors and accelerates the progression of neurocognitive impairment. The article emphasizes the critical need for early intervention, such as the use of hearing aid technology, to mitigate these maladaptive changes and preserve cognitive integrity and function. By critically examining existing theories and introducing a new neuroplasticity-based perspective, this article provides professionals with meaningful insight into the complex connection between sensory loss and cognitive decline.

Keywords: hearing loss, cognitive decline, harbinger hypothesis, cognitive load hypothesis, cascade hypothesis, common cause hypothesis, cognitive maladaptation hypothesis

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Hannah A Glick,[1] Douglas L Beck,[2] Keith Darrow,[3] Jung Trinh[4]
[1]Assistant Professor, University of Northern Colorado, USA
[2]Senior Director of Audiology, Essilor Luxottica Inc and Adjunct Clinical Professor of Communicative Disorders & Sciences, State University of NY at Buffalo, USA
[3]Professor, Worchester State University, USA
[4]Dung Trinh,MD., Chief Medical Officer Healthy Brain Clinic Board of Directors, Alzheimer's Orange County and Los Angeles Speakers Bureau. Otsuka, Eisai, USA

Correspondence: Dr. Douglas L. Beck Au.D. Senior Director of Audiology, Essilor Luxottica Inc.
Email dbeck@essilorluxottica.com

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Abbreviations: ARHL, age-related sensorineural hearing loss; NCD, mild neurocognitive disorder; MND, major neurocognitive disorder

Introduction

Age-related sensorineural hearing loss (ARHL) is one of the most prevalent chronic conditions in older adults, affecting approximately 73 million people in the USA.[1] While the negative consequences of hearing loss on communication are well known, growing evidence indicates that auditory deprivation, as in untreated ARHL, is also a significant and potentially modifiable risk factor for cognitive decline.[2] As the global population ages and the rates of dementia continue to significantly rise, understanding this relationship is becoming increasingly important for clinical care, research, and public health.

Cognitive disorders are marked by deficits in cognitive domains such as complex attention, executive function, learning and memory, language, perceptual-motor skills, or social cognition. These deficits must represent a decline from a previous level of functioning and must be observable by others or documented through objective assessment. Mild Neurocognitive Disorder (NCD)—commonly referred to as mild cognitive impairment (MCI) in clinical practice is characterized by modest cognitive decline in one or more of the above domains that does not interfere with a person's independence in daily activities, though greater effort, compensatory strategies, and accommodations which may be required. In contrast, Major Neurocognitive Disorder (MND) (formerly termed dementia) involves significant cognitive decline that does interfere with independence in everyday activities, such as managing finances, medications, or personal care. The decline must be substantial enough to impair occupational, social, or functional performance. The most common etiological subtype is Alzheimer's disease, but other types include vascular dementia, Lewy body dementia, and frontotemporal dementia, among others.[3]

Multiple longitudinal studies have shown that individuals with untreated hearing loss are considered "at risk" and have an increased likelihood of developing mild cognitive impairment (MCI) and dementia. The co-occurrence of other health comorbidities including low education, depression, hypertension, brain injury, obesity, high LDL cholesterol, diabetes, excessive alcohol consumption, smoking, physical inactivity, social isolation, and depression may further elevate long-term risk for cognitive decline.[2] These findings highlight the critical need to better understand the mechanisms linking auditory deprivation to cognitive impairment.

This article reviews several prevailing hypotheses which attempt to explain this association: the Harbinger Hypothesis, Cognitive Load Hypothesis, Cascade Hypothesis, and Common Cause Hypothesis—each proposing a different pathway through which hearing loss may influence cognitive function.

In addition, we introduce a new theoretical framework: the Cognitive Maladaptation Hypothesis. This hypothesis suggests that prolonged sensory deprivation, particularly in the early stages of hearing loss, may lead to maladaptive neuroplastic changes that impair cognitive function and accelerate cognitive decline. By examining both established and emerging theories, this article aims to provide professionals with a clearer understanding of the interplay between hearing and cognition—and highlight the importance of early intervention to preserve both auditory and cognitive health.

Existing hypotheses explaining the association between hearing loss and cognitive decline

A growing body of research has established a strong association between ARHL and cognitive decline in older adults. As this connection becomes more evident, multiple theoretical models have been proposed to explain the underlying mechanisms. These hypotheses aim to disentangle whether sensory loss is merely

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correlated with cognitive dysfunction or whether it plays a direct or indirect causal role. The following sections summarize four of the most widely discussed theories in the literature: the Harbinger Hypothesis, Cognitive Load Hypothesis, Cascade Hypothesis, and Common Cause Hypothesis. Each offers a unique perspective on how hearing loss may influence cognitive trajectories.

The Harbinger Hypothesis, as the name implies, proposes that sensory impairments such as hearing or vision loss are early warning signs—or 'harbingers'—of cognitive decline and may reflect underlying neurodegenerative changes already in progress. Additionally, the presence of sensory deficits may artificially worsen performance on cognitive assessments due to inadequate access to verbal or visual instructions, rather than reflecting true cognitive dysfunction. This theory emphasizes the methodological challenge in accurately evaluating cognition in individuals with unaddressed sensory deficits. For example, individuals with hearing loss may misinterpret test questions or instructions, leading to reduced performance that mimics cognitive impairment.[4] This hypothesis suggests that some portion of the observed association between sensory loss and cognitive decline may be attributable to misattribution or measurement artifacts, particularly when hearing or vision is not properly accounted for in test design or administration. Although this hypothesis does not propose a biological mechanism of cognitive decline, the Harbinger Hypothesis highlights the importance of addressing sensory deficit prior to performing cognitive assessment or utilizing adapted or alternative cognitive tests designed for adults with sensory impairment.

The Cognitive Load Hypothesis is based on cognitive load theory.[5] This hypothesis posits that sensory deprivation (i.e. hearing or vision loss) places a greater burden on cognitive resources. When greater attentional and cognitive resources are required to interpret poor-quality sensory information, there is reduced cognitive spare capacity to perform other tasks, which may have a downstream negative effect on cognitive functioning (i.e., working memory). Neuroimaging studies have shown that individuals with hearing loss demonstrate increased activation in frontal cortical regions during auditory tasks, suggesting compensatory recruitment of brain areas typically dedicated to executive control.[6,7] Moreover, adults with normal hearing under degraded listening situations and adults with hearing loss exhibit increased situational and short-term cognitive load, as evidenced by pupil dilation and eye tracking, which indicates that the difficulty of processing degraded auditory signals places greater demands on cognitive resources.[8] These findings suggest that as speech (and other sensory/afferent information) becomes more challenging to comprehend, the brain relies more heavily on higher-order cognitive processes to compensate for the reduced or degraded cognitive input. This increased cognitive load required could lead to chronic effortful listening and cognitive fatigue, perhaps contributing to long-term cognitive decline.[9]

The Cascade Hypothesis provides a psychosocial and behavioral explanation for the relationship between sensory impairment and cognitive decline. This hypothesis posits that sensory deprivation (i.e. hearing and vision loss) leads to decreased social engagement—cognitively and socially stimulating activities that reduce long-term risk for cognitive decline. In turn, this can elevate an adult's risk for depression, loneliness, and social isolation. Both qualitative and quantitative research studies have observed increased loneliness among adults with hearing loss compared to those with normal hearing.[10] This may lead to a reduction in both the quantity and quality of cognitive stimulation. Over time, this reduction in environmental and social enrichment may result in fewer opportunities for neurocognitive resilience and plasticity, thus accelerating the onset or progression of dementia. This hypothesis highlights the indirect, yet significant, pathway through which sensory loss could affect cognitive health via emotional and social mechanisms.

The Common Cause Hypothesis proposes that age-related declines in both sensory and cognitive functions may stem from shared neurobiological mechanism, rather than one directly causing the other. These declines occur concurrently as manifestations of more widespread neurodegenerative changes, potentially driven by common structural, vascular, metabolic, and genetic factors. Neuroimaging studies have identified reduced and/or accelerated decline in temporal lobe volumes and gray matter density[11,12] as well as reduced white matter in cortical regions involved in cognitive processing.[12-15]

This hypothesis is further reinforced by findings that both ARHL and cognitive impairment are multifactorial and heterogeneous, often linked by common vascular risk factors such as atherosclerosis, smoking, and diabetes.[2,16,17] Additionally, oxidative stress, which affects both cerebral and auditory microcirculation, has been implicated in the deterioration of both systems.[18] Genetic research also suggests there may be a common biological risk factor linking sensory and cognitive decline. One example is the apolipoprotein E (APOE) e4 gene variant, which is strongly associated with a higher risk of developing Alzheimer's disease. Some studies have found that people who carry this gene may also be more likely to experience hearing loss[19] while other studies contradict these findings.[20,21]

Issues with existing hypotheses

While existing hypotheses each offer valuable frameworks for understanding the relationship between sensory loss and cognitive decline, several limitations and unanswered questions have emerged that warrant further consideration. First, the Harbinger Hypothesis primarily addresses the confounding factor of audibility influencing performance on cognitive tests. Although it is true that individuals with unaddressed hearing loss may perform more poorly on cognitive assessments due to difficulty perceiving instructions or test stimuli, this explanation does not account for studies that indicate that unaddressed hearing loss is associated with significantly poorer cognitive functioning even after addressing the issue of audibility.[22]

Similarly, while the Cognitive Load Hypothesis has been supported by neuroimaging and behavioral studies showing recruitment of additional cortical areas in individuals with hearing loss,[6,7] it primarily accounts for short-term strain on attentional and memory systems and does not fully explain how these situational changes could contribute to long-term cognitive decline. Moreover, the Cognitive Load hypothesis does not explain how the brain's efforts to compensate during listening tasks might lead to wider cognitive impairments that extend beyond just auditory processing.

The Cascade Hypothesis, which suggests that hearing loss contributes to cognitive decline through intermediary factors such as social isolation, loneliness, and depression, has its own set of limitations. While there is substantial evidence linking these psychosocial factors to elevated risk for cognitive decline,[2] these factors may act as mediators rather than primary causal agents.[23] In this context, sensory deprivation (i.e., hearing and vision loss) may initiate a cascade of psychosocial consequences that exacerbate pre-existing vulnerabilities contributing to long-term cognitive decline. Additionally, it is challenging to establish clear directionality, since cognitive impairment may also lead to reduced social engagement and communication difficulties, which could be misattributed as consequences of hearing loss. Not all adults with hearing loss

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experience social isolation, loneliness, or depression, and this hypothesis does not account for individual differences and cognitive resilience.

Lastly, several issues limit the appeal of the Common Cause Hypothesis. A primary concern is the lack of clarity regarding the exact neurobiological mechanisms that link age-related hearing loss (ARHL) and cognitive decline. While common risk factors, such as vascular changes and genetic influences have been proposed, a clear shared underlying neurobiological pathway has not been identified. Although neuroimaging studies have identified deprivation-induced structural and functional changes in auditory and cognitive processing regions in adults with untreated hearing loss, these observations do not establish a direct causal relationship between hearing loss and cognitive impairment. This hypothesis also does not fully account for those adults with hearing loss who develop cognitive decline in absence of other health issues, such as vascular disease.

Collectively, these limitations point to the need for an expanded and/or alternative theoretical framework that considers not only the direct and indirect effects of sensory deprivation but also the maladaptive neuroplastic changes that may occur in response to prolonged degraded sensory input. This gap in the literature provides the conceptual foundation for the Cognitive Maladaptation Hypothesis, which seeks to integrate existing knowledge with emerging neuroscience insights about how the brain responds—and potentially maladapts—to sensory loss over time.

Cognitive maladaptation hypothesis

The Cognitive Maladaptation Hypothesis (proposed by Beck & Darrow)[24] introduces a novel perspective on the relationship between sensory loss—particularly hearing and vision impairment—and cognitive decline. Building on well-established principles of neuroplasticity, this hypothesis suggests that the brain's adaptive responses to sensory deprivation, such as auditory or visual loss, can, under certain conditions and over time as untreated hearing and vision loss progresses, become maladaptive and contribute to long-term cognitive impairment. Neuroplasticity refers to the brain's ability to reorganize and form new neural connections to accommodate changes in sensory input. When this sensory input is reduced or degraded (i.e., in hearing or vision loss), the brain compensates by reorganizing neural circuits, often recruiting alternative brain regions to process the missing sensory information.

Central to this hypothesis is the idea that the brain recalibrates its processing mechanisms in response to degraded sensory signals. This recalibration often relies on prior knowledge, experience, and contextual cues to interpret incomplete or unclear sensory input.[25] Over time, however, the brain's recalibrated baseline for sensory and cognitive processing becomes less accurate, which can increase perceptual and cognitive errors.

For example, Campbell and Sharma[6] found a significant negative correlation between cortical auditory evoked potential (CAEP) latencies in frontal brain regions and speech-in-noise performance in adults with untreated age-related hearing loss (ARHL). Specifically, earlier frontal latencies were associated with poorer auditory speech perception, suggesting that compensatory neural activity in frontal regions may be inefficient or maladaptive. Speech perception in noise is an inherently cognitive task, requiring the integration of auditory input with top-down processes such as attention, working memory, and linguistic knowledge. In challenging listening environments, the brain must actively reconstruct meaning, suppress irrelevant input, and sustain focus—placing considerable demands on cognitive resources. Over time, persistent sensory degradation may drive over-reliance on these compensatory mechanisms, increasing listening effort, cognitive load, and emotional strain. This sustained cognitive demand can contribute to broader psychological effects, including heightened risk for anxiety. Indeed, both hearing and vision loss have been linked to increased incidence of anxiety disorders, likely reflecting the emotional toll of navigating the world with impaired sensory input.[26,27]

The effects of sensory deprivation on cognitive functioning may be more pronounced in individuals with pre-existing cognitive or psychosocial vulnerabilities, such as those with pre-existing mild cognitive impairment (MCI) or depression. Individuals with reduced cognitive spare capacity are already more prone to more cognitive errors, and the maladaptive neuroplastic changes driven by sensory loss can compound these deficits. For example, individuals with MCI may already experience deficits in attention, working memory, executive function, and processing speed which can be exacerbated by the increased cognitive load required to process degraded sensory input.[28-31] In these individuals, the brain's neuroplastic reorganization mechanisms may struggle to compensate effectively, resulting in greater cognitive fatigue, frustration, and heightened psychological distress. Furthermore, these individuals may experience more significant impact on auditory communication, making it more difficult to recalibrate and compensate for the reduced and degraded sensory (i.e., auditory) input.

Clinical implications

The Cognitive Maladaptation Hypothesis presents a theoretical framework for understanding the potential long-term effects of untreated sensory (i.e., hearing and vision) loss. It suggests that sensory deprivation induces neuroplastic changes in the brain that, rather than compensating for auditory deficits, may contribute to cognitive deficits over time. While empirical studies specifically designed to test this hypothesis are lacking, it is consistent with observed patterns of compensatory cortical resource allocation in hearing loss.

Recent findings from the ACHIEVE randomized controlled trial also offer important clinical insights into the ways in which treatment of hearing loss may support cognitive health.[32] This study observed 48% reduction in global cognitive change scores over a three-year period in a cohort of adults with hearing loss receiving hearing aids with more baseline risk factors for cognitive decline, including poorer baseline cognitive functioning on average. These results suggest that among those adults at higher risk for cognitive decline, early intervention may yield more substantial cognitive benefit. From a clinical perspective, these findings highlight the importance of early identification and intervention for hearing loss in older adults, particularly those with additional risk factors for cognitive decline. The ACHIEVE trial's results support the idea that timely treatment, such as use of hearing aids, could support cognitive functioning in high-risk clinical sub-populations. Although the trial did not observe significant cognitive benefits in the cohort of adults with hearing loss with less risk factors in the same timeframe, it is possible that longer-term benefits may emerge in a longer follow-up period. Studies like Glick & Sharma[16] further demonstrate how "re-wiring" of auditory cortex and reversal in compensatory changes in cortical resource allocation (i.e., recruitment of pre-frontal and frontal cortices during for sensory processing) are reversible with hearing aid treatment and restored audibility prescribed according to audiology best practices.

These studies highlight the critical need for early screening, identification, and intervention of both sensory loss and cognitive impairment in aging adults. By identifying and addressing sensory

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loss early, healthcare providers can reduce the cognitive strain caused by sensory deprivation and support cognitive health. Importantly, the strong association between hearing loss and cognitive decline highlights the necessity of a multidisciplinary approach to care. Hearing care professionals should collaborate with psychologists, neuropsychologists, physicians, speech-language pathologists, geriatric specialists, family doctors and other healthcare providers to address both auditory and cognitive concerns. Including cognitive screening as part of routine healthcare can help identify individuals at higher risk for cognitive decline. Coordinating care between specialists ensures timely and appropriate interventions are in place to support cognitive health, potentially slowing down or preventing the progression of cognitive decline in at-risk individuals.

Conclusion

The correlational relationship between hearing loss and cognitive decline is well established, but the underlying mechanisms remain incompletely understood. Existing theories, such as the Harbinger, Cognitive Load, Cascade, and Common Cause hypotheses, provide valuable insights into this connection, yet they may not fully account for the complexities of how sensory deprivation impacts the brain over time. The Cognitive Maladaptation Hypothesis offers a new framework, suggesting that the brain's neuroplastic responses to degraded auditory input can, over time, lead to maladaptive neuroplasticity and cognitive compensation, potentially contributing to cognitive decline.

For clinicians, the hypothesis highlights the importance of early, comprehensive audiometric evaluations and intervention in reducing not only communicative impairments but also the potential for long-term cognitive disruption. This approach aligns with recent guidelines from the American Academy of Otolaryngology–Head and Neck Surgery (AAO-HNS), which advocate for proactive identification and timely treatment of age-related hearing loss as a means to preserve both functional communication and broader cognitive health. Early audiologic evaluation is increasingly recognized not just as a rehabilitative measure, but as a preventive strategy within the larger framework of healthy brain aging.

While more research is needed to evaluate the role of maladaptive neuroplasticity in cognitive decline, this framework offers a compelling case for early intervention in hearing loss as a strategy to protect cognitive health. Looking ahead, future studies should aim to clarify the nature, timing, and reversibility of these proposed neural changes in adults with and without additional health comorbidities which may place them at increased risk for dementia. Long-term longitudinal research, imaging, and cognitive testing will be essential to fully understand the impact of hearing loss on cognitive function and to guide evidence-based recommendations for hearing care in older adults. In the meantime, the Cognitive Maladaptation Hypothesis provides a valuable direction for future research, reinforcing the interconnectedness between sensory health and cognitive function. Early identification through comprehensive audiometric evaluations—such as speech-in-noise testing—followed by timely intervention, may play a crucial role in shaping the trajectory of cognitive health in older adults.

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Conflicts of interest

Dr Beck is Senior Director of Audiology at Essilor Luxottica Inc, manufacturer of Nuance Audio Glasses and other hearing and vision products.

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