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Research Article

Age-related hearing loss and dementia risk across the lifespan: mechanisms, equity, and prevention☆

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ABSTRACT

Age-related hearing loss (ARHL) has emerged as a significant and potentially modifiable risk factor for neurodegenerative disorders, including Alzheimer's disease. A growing body of evidence links ARHL to structural and functional changes in the brain, with implications for cognitive decline and dementia onset. However, both ARHL and dementia are multifactorial conditions shaped not only by biological mechanisms but also by broader social determinants of health. Inequities in access to hearing care, underrepresentation of marginalised populations in research, and variability in intervention outcomes emphasise the need for a more inclusive and integrated approach to prevention. This review synthesises current evidence on the neurocognitive consequences of ARHL, the potential mechanistic pathways linking hearing loss to dementia, and the role of inequity. It includes a novel perspective by highlighting the need for cross-cultural, population neuroscience approaches incorporating exposome and equity frameworks that capture phenotypic variability across diverse populations. This review outlines key priorities for advancing neurocognitive and translational research on hearing loss, neurodegeneration, and global health equity. Addressing these gaps is essential to developing equitable strategies for dementia prevention and optimising cognitive health across the lifespan.

Age-related hearing loss and dementia: a pressing global health concern

Age-related hearing loss (ARHL) is one of the most prevalent chronic conditions affecting older adults and a leading contributor to years lived with disability (GBD 2016 Disease and Injury Incidence and Prevalence Collaborators, 2017). ARHL has been associated with a range of adverse health outcomes, including elevated risks of depression, accidental falls, and stroke (Deal et al., 2019). Notably, it has also been identified as a risk factor for several forms of dementia, including the most common subtype, Alzheimer's disease (AD) (Lin et al., 2011b; Livingston et al., 2024; Loughrey et al., 2018). It has also been linked with other dementia subtypes, such as Lewy body dementia (LBD), and frontotemporal dementia (FTD) (Jiang et al., 2024; Katanga et al., 2023).

As the global population continues to age, the incidence of dementia is rising, prompting growing interest in ARHL as a modifiable risk factor for delaying or preventing the clinical onset of neurodegenerative diseases (Livingston et al., 2024). A *meta*-analysis of observational and trial studies suggests that hearing interventions may significantly attenuate cognitive decline (Yeo et al., 2023). As such, ARHL may contribute to dementia through potentially modifiable pathways.

Nevertheless, the precise mechanisms linking ARHL with increased risk of neurodegenerative disorders such as AD remain incompletely understood (Griffiths et al., 2020; Wayne & Johnsrude, 2015). Given projections that over two billion individuals will be affected by hearing loss by 2050 (WHO, 2025a), there is an urgent need to delineate these pathways. Doing so could facilitate the development of early diagnostic tools and enhance the efficacy of hearing rehabilitation strategies. This review identifies key priorities for future neurocognitive and translational research at the intersection of hearing loss, neurodegeneration, and global health equity.

Auditory measures and dementia phenotypes

Age-related hearing loss (ARHL) has long been viewed as an inevitable consequence of ageing. However, increasing evidence linking ARHL to a broad range of adverse health outcomes—including social isolation, depression, and increased healthcare costs—has reframed it as

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a critical and modifiable public health issue (Olusanya et al., 2014; WHO, 2025a). Despite this growing recognition, our understanding of the condition remains limited. As with Alzheimer's disease (AD), the aetiology of age-related hearing loss (ARHL) involves a complex interplay of biological mechanisms and environmental exposures (Bowl & Dawson, 2019; Förster et al., 2022). It is principally characterised by declines in both peripheral hearing sensitivity and central auditory processing, highlighting the complex interplay between auditory and cognitive systems, particularly in the domain of speech-in-noise perception (Gates & Mills, 2005; Slade et al., 2020). This complexity has contributed to the emergence of cognitive hearing science, an interdisciplinary field investigating the neural and cognitive mechanisms underpinning auditory perception (Rönnberg et al., 2013).

A wide range of auditory measures—from peripheral thresholds to speech comprehension tasks—have demonstrated consistent associations between ARHL and various neurodegenerative conditions (Gates et al., 2011; Johnson et al., 2021; Lasica et al., 2025; Medel et al., 2024; Ruan et al., 2023). Pure-tone audiometry, which measures hearing thresholds in decibels hearing level (dB HL) across key speech frequencies (0.5, 1, 2, and 4 kHz), is the most widely used clinical tool and remains the gold standard for assessing peripheral hearing loss (Powell et al., 2021). Historically, mild hearing loss was defined as an average threshold of 25 dB HL across these frequencies (Humes, 2019). Numerous epidemiological studies have linked ARHL with cognitive decline, cognitive impairment and dementia using this metric (Loughrey et al., 2018). More recent guidelines have revised the diagnostic threshold to 20 dB HL, recognising the functional impact of even minimal hearing impairment (Humes, 2019; Olusanya et al., 2019). Importantly, several studies have demonstrated that even within the 'normal' range of hearing, subtle deficits are associated with poorer cognitive outcomes and elevated depressive symptoms (Chern et al., 2021; Golub et al., 2020; Golub et al., 2019; Irace et al., 2022).

Measures of speech-in-noise perception have shown particularly strong associations with cognitive impairment and dementia risk, supporting their use as sensitive indicators of central auditory dysfunction (Gates et al., 2011). Deficits in auditory processing are associated with large-scale cortical reorganisation involving attentional, visual, and motor networks—changes that may reflect both compensatory plasticity and neurodegenerative processes (Slade et al., 2020; Slade et al., 2022). However, the extent to which peripheral hearing loss directly contributes to cognitive decline—rather than through central auditory processing deficits, which may themselves result from reduced peripheral acuity, age-related changes, or underlying neuropathology—remains unclear.

Central auditory dysfunction is increasingly recognised as an early and distinguishing feature of multiple neurodegenerative diseases (Johnson et al., 2021). In AD, impairments in central auditory processing may precede global cognitive decline, occur independently of peripheral hearing thresholds, and are associated with structural atrophy in temporoparietal regions, particularly within the default mode network (Golden et al., 2015; Idrizbegovic et al., 2011; Johnson et al., 2021). Other forms of dementia, including Lewy body dementia (LBD) and frontotemporal dementia (FTD), display distinct auditory processing profiles, suggesting that auditory biomarkers may help differentiate between dementia phenotypes and provide insight into underlying pathophysiology (Johnson et al., 2021). Despite these advances, significant gaps remain in our understanding of the neurobiological mechanisms that disrupt the auditory–cognitive interface. Elucidating these pathways is essential for the development of early diagnostic tools and targeted interventions to mitigate cognitive decline in populations affected by hearing loss.

Potential mechanisms linking hearing loss and dementia

The pathophysiological relationship between age-related hearing loss (ARHL) and dementia remains incompletely understood, with multiple mechanisms proposed. These reflect the complex and multifactorial aetiology underlying both ARHL and neurodegenerative diseases (Lin et al., 2013; Panza et al., 2015; Wayne & Johnsrude, 2015). These mechanisms may co-occur and contribute either as a common aetiological cause of both ARHL and dementia (common cause hypothesis) or through the impact of ARHL on cognitive function.

One possibility is that ARHL and dementia arise from shared pathological processes—such as systemic frailty or common genetic factors—that simultaneously affect peripheral auditory function and cognitive decline (Alvarado et al., 2021; Förster et al., 2022; Lin et al., 2011b; Loughrey et al., 2018; Panza et al., 2015; Uchida et al., 2018; Wayne & Johnsrude, 2015). There may be a shared vascular pathophysiological mechanism affecting both cochlear health—via the strial microvascular structure of the inner ear—and the blood–brain barrier (Förster et al., 2022). Alternatively, ARHL may exert indirect effects by contributing to modifiable dementia risk factors, particularly social isolation, loneliness, or reduced cognitive stimulation—factors that may accelerate the clinical expression of underlying neuropathology (Lin et al., 2011b; Loughrey et al., 2020; Loughrey et al., 2018; Panza et al., 2015; Uchida et al., 2018; Wayne & Johnsrude, 2015).

An important area of investigation in recent research has been the impact of ARHL on brain structure and function, particularly in relation to maladaptive neuroplasticity. Neural reorganisation in response to prolonged auditory deprivation has been posited as a key mechanism linking hearing loss with dementia risk (Armstrong et al., 2019; Belkhiria et al., 2019; Belkhiria et al., 2020; Campbell & Sharma, 2013, 2014, 2020; Ha et al., 2020; Husain et al., 2011; Lin et al., 2014; Park et al., 2016; Qian et al., 2017; Ren et al., 2018; Rudner et al., 2019; Slade et al., 2022; Wang et al., 2022; Xu et al., 2019). This may be driven by a maladaptive neuroplastic adjustment in response to distorted auditory input (sensory deprivation hypothesis) (Lin et al., 2011b; Wayne & Johnsrude, 2015). ARHL may cause altered neural connectivity which may be adaptive in the short term but may undermine the brain's resilience to neuropathologies and lower the threshold for the symptomatic expression of dementia (Armstrong et al., 2019; Lin et al., 2014; Slade et al., 2022). Animal models of induced hearing loss have demonstrated hippocampal atrophy, impaired spatial memory, altered synaptic function, and increased tau phosphorylation—providing biological plausibility for a causal relationship between auditory input loss and neuropathological changes characteristic of dementia (Dong et al., 2018; Jafari et al., 2023; Ko et al., 2024; Liu et al., 2016; Paciello et al., 2021; Park et al., 2018; Park et al., 2016; Yu et al., 2011).

The diversity of mechanisms likely contributes to interactive effects, giving rise to heterogeneous neurocognitive phenotypes of ARHL with important implications for public health interventions. Recent findings from the Aging and Cognitive Health Evaluation in Elders (ACHIEVE) trial, a multicentre randomised controlled study, illustrate this complexity. The intervention—hearing rehabilitation using hearing aids—was found to be effective in a pre-specified subgroup of older adults at increased risk of cognitive decline, but not in healthier community-dwelling participants (Lin et al., 2023). These results suggest that the benefits of intervention may depend on underlying vulnerability profiles.

Despite this heterogeneity, a robust body of evidence supports a consistent average effect and ARHL remains one of the largest modifiable risk factors for dementia at the population level (Livingston et al., 2024; Loughrey et al., 2018). Further, many neuroimaging studies with small sample sizes show group-level neural differences from the early stages of ARHL without stratifying for various audiometric or health profiles (Campbell & Sharma, 2013, 2014, 2020; Husain et al., 2011; Peelle et al., 2011; Qian et al., 2017; Tong et al., 2023). However, larger scale neuroimaging studies that can adequately stratify participants by audiometric severity, comorbidities, or other relevant health variables would be informative (Armstrong et al., 2019; Fitzhugh & Pa, 2023; van 't Hooft et al., 2023; Xu et al., 2019).

Advancing mechanistic research toward more precise phenotyping is

essential for identifying individuals most at risk and for informing targeted intervention strategies. While auditory dysfunction is a symptomatic feature of several canonical dementias (e.g., Alzheimer's disease, frontotemporal dementia, Lewy body dementia) (Johnson et al., 2021), it is critical to delineate the specific neurocognitive patterns that are attributable to ARHL and that may be most responsive to hearing-focused interventions (Lin et al., 2023).

Neurobiological changes and neurocognitive impact associated with ARHL

A growing body of research has sought to characterise the neurocognitive consequences of age-related hearing loss (ARHL) and clarify its temporal dynamics. Over the past several decades, numerous epidemiological studies have established strong associations between ARHL and cognitive decline across multiple domains, including executive function, episodic memory, visuospatial abilities, and global cognition (Baltes & Lindenberger, 1997; Lin et al., 2011a; Lindenberger & Baltes, 1994; Stickel et al., 2024). A systematic review and *meta*-analysis of 40 cross-sectional and longitudinal studies confirmed these associations, demonstrating declines in all major cognitive domains (Loughrey et al., 2018). Furthermore, a dose–response relationship has been observed: more severe hearing loss is associated with a substantially higher risk of developing dementia (Lin et al., 2011b). Collectively, these findings suggest that the neurocognitive consequences of ARHL extend well beyond auditory-specific functions and may reflect broader compromise of brain health.

ARHL has been linked with multiple neurodegenerative dementias, including AD, FTD, and LBD, implicating diverse and potentially distinct neural mechanisms that may operate independently of classical dementia-related neuropathologies (Jiang et al., 2024; Parker et al., 2024; van 't Hooft et al., 2023). Biomarker studies have associated ARHL with elevated cerebrospinal fluid tau levels (Xu et al., 2019), increased β-amyloid burden (Golub et al., 2021; van 't Hooft et al., 2023; Zheng et al., 2022), and Lewy body pathology. However, these associations appear variable across studies, raising the possibility that ARHL may influence neurocognitive function independently of any one specific neuropathological pathway (Lasica et al., 2025; Morita et al., 2019; Parker et al., 2024; Slade et al., 2022; van 't Hooft et al., 2023). Genetic research supports this hypothesis: ARHL has been shown to be linked to cognitive decline independent of polygenic risk for either hearing loss or Alzheimer's disease (Brenowitz et al., 2020; Morita et al., 2019).

Neuroimaging studies indicate that functional brain changes may occur from the early stages of ARHL, potentially preceding overt structural atrophy and contributing to long-term cognitive vulnerability (Campbell & Sharma, 2013, 2014; Sakurai et al., 2025). Initial stages of hearing loss have been associated with increased connectivity and local grey matter volume in certain regions, followed by accelerated atrophy in areas critical for memory and cognition, such as the hippocampus and entorhinal cortex (Armstrong et al., 2019; Campbell & Sharma, 2013, 2014; Lin et al., 2014; Xu et al., 2019). Indeed, the rate of atrophy in temporal regions and whole brain volume among individuals with ARHL has been reported to approximate that seen in those developing mild cognitive impairment (Lin et al., 2014) and may mediate differences in cognitive function including memory and global cognition (Parker et al., 2024; van 't Hooft et al., 2023). Atrophy has been observed in the hippocampus and entorhinal cortex (Armstrong et al., 2019; Xu et al., 2019), which are also affected from the early stages of AD (Braak & Braak, 1991). A recent *meta*-analysis further identified consistent grey matter reductions in the temporal lobe associated with ARHL (Slade et al., 2022).

Behavioural and neuroimaging studies provide insight into the potential trajectory of cognitive decline associated with ARHL and may inform mechanistic understanding. Evidence suggests that following the onset of hearing loss, compensatory neural adaptations—such as cross-modal reorganisation and reallocation of cortical resources—emerge to maintain auditory processing (Campbell & Sharma, 2013, 2014, 2020). These changes may, however, come at a cognitive cost, potentially diverting resources from other domains such as episodic memory encoding (Campbell & Sharma, 2013; Rönnberg et al., 2013; Tun et al., 2009). In the longer term, this may lead to selective deterioration in long-term, multimodal memory systems, compared to short-term memory or working memory—suggesting a specific cognitive signature of ARHL distinct from global neuropathological causes (Rönnberg et al., 2021; Rönnberg et al., 2013).

Emerging findings also suggest that ARHL may initially affect lower-order automatic processes across cognitive domains such as attention, memory, and language which is asymmetric compared to higher order executive functioning (Loughrey et al., 2021; Loughrey et al., 2020b; Loughrey et al., 2019). This asymmetry may occur prior to more noticeable cognitive decline. Recent electroencephalography (EEG) research using a visual working memory task indicated that individuals with ARHL recruited higher-order cognitive control networks to support lower-order implicit processing, suggesting a shift in neural resource allocation (Loughrey et al., 2023). Another study using functional magnetic resonance imaging (fMRI) reported that cognitive decline with ARHL may be underpinned by disrupted information flow between primary sensory areas and higher-order association networks (Tong et al., 2023). Broadly, these findings suggest that even early-stage hearing loss may lead to altered neurocognitive function, neural connectivity and atrophy, and potentially lowering the threshold for the clinical manifestation of dementia.

Toward prevention and the role of inequity in ARHL and dementia

As outlined above, there is substantial evidence linking ARHL with an increased risk of dementia (Livingston et al., 2024; Loughrey et al., 2018). Given that ARHL affects a large proportion of the global population of older adults and is estimated to contribute to a significant fraction of dementia cases, it represents a promising target for prevention strategies aimed at reducing the global burden of dementia (Livingston et al., 2024). A recent *meta*-analysis combining observational and interventional studies reported that hearing aids and cochlear implants were associated with a reduced risk of cognitive decline (Yeo et al., 2023). However, findings from a recent randomised controlled trial (ACHIEVE) suggest that the protective effect of hearing aids may be limited to individuals at elevated risk for dementia, with no significant benefit observed in a broader cohort (Lin et al., 2023). These findings indicate that the efficacy of hearing interventions may vary across populations, shaped by underlying risk profiles.

A key challenge in identifying populations most likely to benefit from hearing intervention is the limited understanding of the mechanistic pathways linking ARHL and cognitive decline. Relatively few studies have simultaneously examined the multiple biological, psychosocial, and environmental factors that may serve as markers of either a shared causal pathway or a direct effect of ARHL on neurocognitive deterioration (Loughrey et al., 2018; Matthews et al., 2024; Zhang et al., 2025). Many datasets or studies include markers of only a limited number of mechanisms (e.g., frailty, social isolation), while omitting others such as cognitive load or neuroimaging data on cortical atrophy. Although emerging evidence supports a plausible neural mechanism linking hearing loss to dementia risk (Griffiths et al., 2020; Lasica et al., 2025; Loughrey et al., 2023; Slade et al., 2022), both ARHL and dementia are embedded within broader constellations of risk that include genetics, lifestyle, comorbidities, environmental exposures, and social determinants of health (Asakawa et al., 2024; Bach et al., 2024; Baez et al., 2024; Matthews et al., 2024; Panza et al., 2015). Clarifying these pathways is essential, as they may serve as actionable targets for both hearing rehabilitation and broader dementia prevention strategies (Zhang et al., 2025).

Advancing mechanistic understanding will require integrated,

multimodal approaches—particularly large-scale neuroimaging datasets capable of examining interactions across biological (e.g., genetics, epigenetics), psychological (e.g., depression, stress, loneliness), and environmental (e.g., noise exposure, healthcare access) domains (Birba et al., 2022; Jiang et al., 2024; Moguilner et al., 2024; Panza et al., 2015; Tarawneh et al., 2022; Zhang et al., 2025). Research on these mechanisms is crucial for identifying possible ARHL-dementia phenotypes and providing further robust evidence of the association (Zhang et al., 2025). It will be necessary to identify the key cognitive metrics which index an impact of ARHL from the earlier stages in order to understand the trajectory of cognitive decline and to refine clinical subtyping and accurately gauge how effective interventions truly are (Lin et al., 2023; Yeo et al., 2023). The shorter-term reallocation of executive resources may mask subtle neurocognitive effects to support auditory task performance, delaying detection until decline becomes more pronounced (Campbell & Sharma, 2013; Loughrey et al., 2023).

There is also a substantial issue of equity (Fig. 1) as most people suffering from hearing loss and dementia reside in low- and middle-income countries (LMICs) (Mukadam et al., 2019). According to the World Health Organization (WHO), approximately 80 % of those with disabling hearing loss and over 60 % of those living with dementia are in LMICs (WHO, 2025a, 2025b). Both ARHL and dementia are linked with multiple risk factors that are tied to the socioeconomic status of a population. Some populations, including in western countries (e.g., lower socio-economic groups, ethnic minorities), are underrepresented in neuroimaging datasets or epidemiological studies with measures of dementia biomarkers which might mean that estimates of the ARHL-dementia association must be applied cautiously or may be

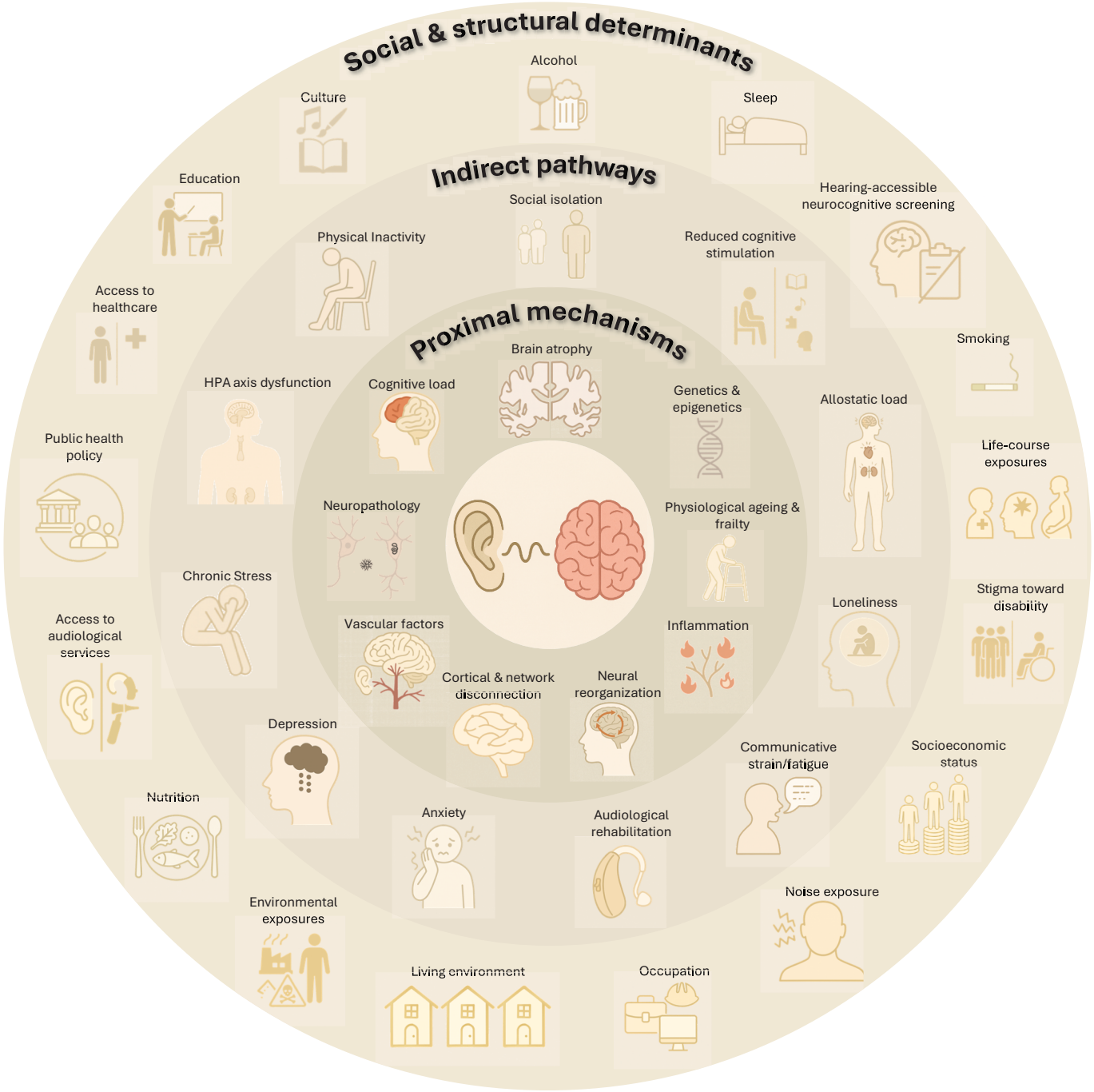

Fig. 1. The role of equity in age-related hearing loss to dementia risk.

underestimated in certain populations (Conceição Santos de Oliveira et al., 2023; Ishak et al., 2025; Lasica et al., 2025; Ordóñez et al., 2025; Samelli et al., 2025).

ARHL and dementia likely present with varying phenotypes and interaction effects across populations. Factors such as cardiovascular status, psychological stress, and socioeconomic conditions may co-vary with hearing loss and differentially influence dementia risk (Loughrey et al., 2018; Ordóñez et al., 2025). These interactions could explain differential rates of cognitive decline and variable responses to intervention, even when a robust average effect is observed (Loughrey et al., 2018; Yeo et al., 2023). To support equitable prevention, neurocognitive assessment tools must also be accessible to those with hearing loss as well as scalable and cost-effective. Tools that can capture the cognitive impact of hearing loss sensitively – particularly in low-resource settings – will be essential (Mukadam et al., 2019; Mushtaq & Ibáñez, 2025). Incorporating underrepresented groups into epidemiological neuroimaging studies that use affordable measures such as computerised tasks or EEG offers enormous translational potential for developing clinical tools that promote equitable access to dementia screening and healthcare (Loughrey et al., 2023). Such tools could potentially help index the efficacy of hearing interventions by providing a more valid metric of neurocognitive rehabilitation (Giroud et al., 2017).

The global burden of dementia is projected to triple by 2050, reaching an estimated 150 million cases (GBD 2019 Dementia Forecasting Collaborators., 2022). A rapidly expanding body of research points towards ARHL being an important potentially modifiable risk factor for dementia (Livingston et al., 2024). However, these are both complex, multifactorial conditions with many potential mechanistic pathways linking them. For example, different audiometric profiles might be linked with different patterns of cognitive decline or dementia subtypes (Johnson et al., 2021). Longitudinal, large-scale, and cross-cultural studies with diverse cohorts are needed to explore hearing-cognition links and clarify the causal relationships. An inclusive approach will improve characterisation of the exposome shaping both conditions and help disentangle underlying mechanisms—potentially identifying high-risk phenotypes more amenable to targeted intervention. This is crucial as there may be variation across populations by which pathways connect these two conditions due to differences in environmental exposures and access to supports (Lin et al., 2023). Importantly, many of the populations most affected by hearing loss and dementia remain underrepresented in neurocognitive and biomarker research. Without their inclusion, intervention strategies and diagnostic tools risk being less effective, less generalisable, or less accessible. The WHO estimates that over 80 % of the people who would benefit from hearing aids do not use them (WHO, 2021), a disparity that reflects broader structural barriers to care and inequity in global brain health. Inclusive research that integrates diverse populations and considers social, biological, and environmental determinants is essential to establishing robust evidence for hearing interventions as a means of dementia prevention. Such efforts will not only advance our understanding of the hearing–cognition relationship but also contribute to more equitable approaches in promoting healthy cognitive ageing worldwide.

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