

A Field Guide to Interpreting and Discussing the Public Health Landscape on Hearing Loss and Cognition

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An explosion in public health literature over the past 15-20 years has linked hearing loss to numerous areas of aging, including cognitive decline, social isolation, health resource utilization, dementia, and physical activity. The result has been significant media coverage highlighting the importance of hearing loss and aging. This is especially true of the association between hearing loss with cognitive decline and dementia among older adults. Communication and interpretation of research into public policy, messaging, and clinical practice is difficult. Science is nuanced and progresses simultaneously (1) rapidly at the micro level with new studies constantly coming out, and at (2) a glacial pace at the macro level as each study fills narrow gaps to add to an overall big picture conclusion. Clinicians have the difficult task of evaluating the strengths and weaknesses of each individual study while forming a continually evolving, broad conclusion about the state of the science. It is easy to over- and understate the full picture. This narrative aims to provide a basic review of public health concepts and an interpretation of the research on hearing loss and cognitive decline within that framework, for hearing care providers to relay balanced messaging to patients.

Evolution in Public Health Interest in Aging, Hearing, and Cognition

Public health is a relatively young field in the broad context of health care. Formal documents and training in clinical health care have existed for over a millennium, with some medical schools in Europe reporting continuous operations for more than 800 years. Public health finds its

contemporary roots in the 19th century with the use of statistics, spatial mapping, and epidemiologic reasoning to determine origins and sources of communicable diseases. The field quickly evolved to communicable disease prevention at the population scale (e.g., immunization and sanitation), and by the mid-20th century, public health took the lead in studying population risk factors and public policy to prevent chronic diseases and mortality (e.g., smoking and lung cancer). By the 21st century, public health had entered broader research focused on risk factors, enabling activities, advocacy, and translation into policy for general health promotion across the lifespan.

The world's demographics have rapidly shifted toward older populations due to longer life expectancies and lower birth rates. Consequently, preventing age-related conditions like cognitive decline and dementia have taken a front seat in recent public health research. While often used interchangeably, cognition refers to domains and processes (e.g., memory, language, processing speed, or executive function) that are measurable via individual instruments, such as cognitive tests. The domains can be described in isolation or combined in a global summative score. Dementia is a clinical diagnosis in which an individual has experienced measurable decline in at least two cognitive domains that has led to significant interference with the ability to work or function.

In the context of aging, hearing loss has traditionally been portrayed as a bothersome, stigmatizing, yet relatively inconsequential aspect of aging. However, hearing loss is common, and its prevalence increases with age: from nearly half of all adults over 60 years to two-thirds of those over 70 years, to nearly all of those over 90 years. Sensory input is the only way our brain can make sense of the world around us. Social and physical activities all require some sensory input whether it be visual, auditory, kinetic or others. In the context of aging, the brain has developed and been shaped over a lifespan dependent on that sensory input. Changes in that sensory input (e.g., hearing loss) could alter how one functions.

Hearing Loss and Cognitive Function: Epidemiologic Concepts on Causality

Epidemiology, the study of disease distribution, frequency, and determinants in populations, is the foundational science of public health that describes risks (e.g., probability of an event occurring), rates (e.g., frequency of occurrence), and association (e.g., quantifying the relationship between two variables) of conditions in a population sample. Some epidemiologic work is purely descriptive surveillance to describe the prevalence of a condition. Conversely, other work is intended to lead to a conclusion, or inference about, the nature of the relationship between two factors. Inference work combines epidemiologic and biostatistical methods to determine a **significant association** or statistically probable link beyond correlation that considers **bias** (e.g., systematic errors in study design) and **confounding** (e.g., extraneous variables that distort an association because the effects of the exposure and confounder are mixed, making it difficult to understand what is the effect of the exposure and what is the effect of the confounder).

Inferential models are vital in differentiating correlations from potential causation. For example, hearing loss and cognitive decline are **correlated** because they both increase with age, but that does not necessarily mean they are causally related. Importantly, understanding **causality** is not possible within a single study. Establishing a causal relationship requires evaluating the strength of the individual studies and ensuring they are sufficiently free of bias and confounding in the context of the entire literature on a topic across basic, clinical, and population disciplines of science. There is no precise checklist for causality, but frameworks can help guide thinking.

Hill's Criteria of Causality, proposed by Austin Bradford Hill, are a set of principles used in epidemiology to assess whether an observed association between an exposure and an outcome is likely to be causal, based on the cumulative evidence. The criteria include **strength of association**, meaning stronger relationships are more suggestive of causation (because they are less likely to be due to bias or confounding), and **consistency**, where similar findings are observed across different studies and populations. **Specificity** refers to a cause leading to a single effect, though this is less

emphasized today. Moreover, specificity is less important in the context of chronic conditions with multiple risk factors as it was intended for infectious disease with a single cause. **Temporality**, the most essential criterion, requires that exposure precede the outcome. The **biological gradient**, or dose–response relationship, indicates that greater exposure is associated with greater risk.

Plausibility and **coherence** emphasize that the association should align with existing biological and scientific knowledge. **Experimental** evidence, such as results from interventions or randomized trials, can further support causality. Finally, **analogy** considers whether similar exposures are known to produce similar effects. Together, these criteria serve as flexible guidelines rather than strict rules, helping evaluate evidence and make informed judgments about the causal relationship between hearing loss and cognitive decline.

Causal and Non-causal Mechanisms Linking Hearing Loss and Cognitive Decline

A key step in causality is establishing a conceptual framework for how hearing loss and cognitive decline could plausibly be connected, while also acknowledging and addressing potential threats to validity (e.g., bias and confounding). Notably, we don't have definitive studies that demonstrate a link between hearing loss and cognitive decline, and a direct pathway is biologically implausible. However, researchers have proposed three major indirect mechanistic pathways connecting the two: cognitive load, direct structural brain changes, and social isolation.

Cognitive load refers to the idea that a degraded auditory signal requires the brain to put in more effort to decode and make sense of information. That constant extra load on the brain could impose another 'hit' that reduces cognitive reserve or limits the brain's resilience against other pathologies (e.g., vascular or amyloid). Second, recent imaging studies suggest that hearing loss is independently associated with poorer white-matter microstructural integrity and brain atrophy, especially in the temporal lobe. The decreased stimulation from the auditory signal could lead to reconfiguration and atrophy in brain areas, which, in turn, could contribute to further cognitive decline. Lastly, hearing loss can limit oral communication and participation in some social activities. A long line of research shows that social isolation, an objective measure of social connection (e.g., number of interactions), is associated with numerous health measures in older adults, including dementia. Importantly, none of these pathways has been proven, and many rely on smaller experimental studies.

While these represent potentially causal paths, epidemiologic studies must also consider non-causal paths when an association is observed. **Bias** refers to a systematic error in the design, conduct, or analysis of a study that leads to an incorrect estimate of the association between an exposure and an outcome. Unlike random error, bias consistently skews results in a particular direction (e.g., overestimating or underestimating the true effect). Numerous forms of bias exist and can arise at multiple stages of a study. **Selection bias** is a systematic error in how participants join the study, are selected for the analytic sample, or leave the sample over time. In hearing and cognition research, we may consider that those with severe hearing loss could be excluded from a study or that populations of hearing aid users are fundamentally different from those of non-users.

Information bias (or measurement bias) occurs when errors occur in how data are collected. For example, information bias would be present if people with hearing loss do poorly on cognitive assessments because they can't hear the prompts. **Confounding**, sometimes considered a type of bias, occurs when a third variable is related to both the exposure and outcome and distorts the observed relationship. For example, hearing loss and cognitive decline could each be caused by

another variable, like cardiovascular disease.

Measurement Matters in Determining Inference

It's not news to any audiologist, but hearing can be measured numerous ways in research. However, a problem arises when the distinct measures aren't considered in interpreting the findings. For example, some research has suggested a reverse pathway of dementia causing hearing loss. However, the models used a speech understanding in noise measure to define hearing loss without considering that speech understanding in noise is both auditory (e.g., peripheral access to sound) and cognitive processing in nature. The use of self-reported hearing to approximate audiometrically measured hearing loss can also be problematic in the context of studying hearing loss and cognitive decline as cognitive decline and dementia could affect how one responds to questions about hearing status.

Peripheral measures of hearing (e.g., pure-tone audiometry) fit the proposed causal framework (e.g., decreased access to sound leads to mechanisms that cause dementia). Moreover, peripheral measures are not affected by potential reverse causation pathways and are relatively free of information bias, as research suggests that older adults with dementia have valid and reliable pure-tone measures. To avoid incorrect study conclusions, research questions motivated by causal frameworks should use peripheral measures. This is not to say that speech and self-reported measures are less important than objective measures, but rather that, in this context, they do not fit the proposed framework.

What do we Know? Observational Literature Findings Linking Hearing Loss and Cognitive Decline

The concept of a link between hearing and cognition is not necessarily as new. Audiology and hearing science studies using laboratory- and clinic-based research designs have long examined the interplay between auditory and cognitive processes in the brain. Public health research hasn't had the same rich history. However, the first papers may be older than one realizes. In 1989, Uhlmann et al. published a paper in JAMA describing a case-control study suggesting that adults with dementia had poorer hearing than those without dementia. Despite being published in a high-profile academic journal, public health work on the topic was sparse until more recently.

Over the past 15-20 years, a steady stream of research has consistently reported associations between hearing loss and cognitive decline among older adults. A 2013 study of 1966 older adults over 6 years in the HealthABC epidemiologic study reported a 32% faster rate of cognitive decline among those with hearing loss than among those without. Notably, the study reported a 40% faster rate of cognitive decline when a measure of cognition that requires auditory input was used. The study is a strong example of information bias, yet it reported a significantly faster decline when using a measure that required no auditory input. In 2010, Lin et al. examined the incidence of dementia in a cohort of 639 older adults aged 65 years or older. In models adjusted for age, sex, race, education, diabetes, smoking, and hypertension. They reported significantly higher hazard ratios or risk for developing dementia as the severity of hearing loss increased, such that those with mild, moderate, and severe hearing loss had two, three, and five times the risk of developing dementia in the timeframe relative to those without hearing loss. Deal et al. (2017) followed this work with an analysis of 1889 older adults over 9 years that found no association between mild hearing loss and incident dementia. Still, moderate or greater hearing loss was associated with a 1.5-fold increased risk. Notably, the study reported a significant association between increasing

pure-tone average (PTA) and the risk of incident dementia, such that every 10dB increase in PTA was associated with a 1.14 increase in risk. Both studies offer a glimpse into a gradient response, whereby increasing hearing loss is associated with a higher risk.

The Lancet Commission on Dementia Prevention, Intervention and Care has conducted two rigorous meta-analyses of the association between hearing and dementia. The most recent report, hearing loss increased the estimated dementia risk by 32%, contributing to 7% of global cases. Nonetheless, limitations include the use of screening measures with limited ability to quantify hearing loss, the use of the same cohort in some studies, the inclusion of studies assessing other associations (e.g., multisensory), and expanded age ranges that include young adults who may not be at risk of developing dementia. The prior Lancet meta-analysis included only three studies but addressed some of these limitations and estimated a 55% increased risk of dementia. Results of other meta-analyses agree, suggesting the true magnitude of the hearing loss-dementia association may be 30–50%. Within the context of Hill’s Criteria for Causality, the literature has been consistent. However, overall, it is still in its nascency and lacks racial, socioeconomic, and geographic representation on a global scale.

Notably, the Lancet Commission report is often interpreted as stating that hearing loss is the largest, strongest, or most important risk factor, given its higher attributable risk fraction of 7%. However, the interpretation of this number is that if all hearing loss cases were eliminated, then 7% of dementia cases would be prevented. Recall just how prevalent hearing loss is among older adults, and now consider that other modifiable risk factors include things like smoking, which is less common, at 4%. The prevalence of exposure matters in interpreting the number, and caution should be applied when discussing individual risk. In fact, a common fallacy in public health research is applying population risk, which represents an average across a sampled group, to individual risk, which varies with other variables that increase or mitigate an individual’s risk. For example, sugary drinks may be associated with a higher risk of diabetes, but that risk is very different depending on an individual’s consumption habits, physical activity, genetics, and other broader health variables.

Intervention Research: Does Intervention Modify the Association

After reviewing the research on hearing loss, cognitive decline, and dementia, many have jumped to champion hearing aids as a major way to prevent cognitive decline and dementia. In fact, popular media is awash with this messaging. However, can we be sure that is the case? The observational study literature generally suggests that hearing aid use is associated with reduced cognitive decline. However, individuals with hearing loss who use hearing aids are typically different from those without, with respect to educational attainment, average income, and health care access and utilization. Given that these factors protect against dementia, definitive answers about the effect of hearing aid on delaying dementia and cognitive decline likely needs to come from randomized trials. **Randomized controlled trials** are the gold standard study design for establishing causal relationships in epidemiologic research. This is because, compared to observational studies, randomization protects against bias and confounding, increasing comparability of treatment groups with respect to factors that influence outcome risk. In an ideal scenario, any observed difference in the outcome comparing treatment to control may therefore be ascribed solely to treatment.

The Aging and Cognitive Health Evaluation in Elders (ACHIEVE) Study was a large, multicenter randomized controlled trial designed to test whether treating hearing loss can slow cognitive

decline in older adults. The study enrolled 977 community-dwelling adults aged 70–84 years in the United States with untreated mild-to-moderate hearing loss. Participants were randomized to either a hearing intervention group, which received hearing aids, counseling, and ongoing audiologic support, or a control group that received a health education program focused on healthy aging. **Overall, the trial found no significant difference in cognitive decline between groups when analyzing the full study population over about 3 years, resulting in a null finding.** However, a key prespecified subgroup analysis of participants recruited from a long-standing cardiovascular cohort revealed an important nuance. Participants at higher risk for cognitive decline (e.g., poorer cognition at baseline and a greater number of risk factors for cognitive decline, such as lower education level, older age, and higher prevalence of cardiovascular disease) showed a hearing intervention significantly slowed cognitive decline by approximately 48% compared to controls. In contrast, lower-risk participants (generally healthier volunteers) showed little measurable benefit, possibly because their baseline rates of decline were slower. This subgroup finding is consistent with the healthy volunteer effect observed in many trials with older adults (i.e., the same people who are able and healthy enough to join a trial at older ages are overly healthy compared to the general population). We don't know whether the subgroup finding is a strong signal or spurious, with questions remaining about the same group that showed benefit also appears to use the hearing aids the least (e.g., fewer daily hours and more likely to stop altogether). The mixed overall results indicate that effects may depend on baseline risk, duration of follow-up, and timing of intervention, and further long-term studies are needed to clarify the magnitude and generalizability of benefit.

Putting it All Together: Causation?

After a dive into public health principles and brief review of the literature on hearing loss and cognitive decline, does the association between hearing loss and cognition and dementia meet Hill's Criteria of Causality? Perhaps as one might expect, the answer requires some nuance. There is substantial, though not definitive, support for a causal relationship. Evidence for temporality is strong, as longitudinal cohort studies consistently show that hearing loss precedes cognitive decline and incident dementia. The association is also highly consistent across analytic populations and study designs, with multiple investigations reporting a 1.5- to 2-fold increased risk of cognitive impairment among individuals with hearing loss. A clear biological gradient further strengthens causal inference, as greater severity of hearing loss appears to be associated with progressively higher risk of dementia. Plausible mechanisms have been proposed, including increased cognitive load due to degraded auditory input, social isolation leading to reduced cognitive stimulation, and shared neurodegenerative or vascular pathways affecting both auditory and cognitive systems. These mechanisms are coherent with the current understanding of brain aging and sensory deprivation. However, some criteria are less well satisfied. Experimental evidence is emerging; while recent intervention studies suggest that hearing aid treatment may slow cognitive decline, randomized controlled trial data remain limited and inconclusive. Additionally, residual confounding, particularly from age-related and vascular factors, cannot be entirely excluded. Overall, the evidence fulfills several key criteria, supporting a relationship that is moderately suggestive of causality but not definitively established.

Conclusion and Messaging to Patients

Should audiologists and hearing care providers tell patients that hearing aids will prevent cognitive decline? Again, as you might expect, this requires nuance. We've established that there is substantial literature that leans towards a causal relationship between hearing loss, cognitive

decline, and dementia. However, there is not enough evidence to say hearing care prevents this association – even in high-risk individuals, it is unclear whether the subgroup finding reported in ACHIEVE is true signal for a future study or spurious. Patients must understand that population-level risk does not equate to individual risk and that hearing care is one of many factors that determine an individual’s cognitive trajectory. Relaying positive messaging and avoiding overpromising a single intervention, such as hearing care, to prevent cognitive decline is important. For example, messaging could focus on how it cannot harm them and has many, many benefits, including reducing social isolation, improving communication, enhancing quality of life, and reducing fatigue, which, taken together, may help support cognitive health in late life.

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