Contemporary research is increasingly focused on the topics of cognition and hidden hearing loss. The articles in this collection take us beyond audibility to look at these topics that affect perception and understanding of complex sounds.

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Hidden Injury in the Noise-Exposed and Aging Ear

Sharon G. Kujawa, Ph.D.

SUMMARY

Noise exposure and aging are two common causes of hearing loss in humans. Historically, the focus has been on hair cell damage and the threshold elevations that this causes. However, we now know that, for both noise and aging, inner hair cell synapses with cochlear afferent neurons are primary targets. Well before threshold elevations and hair cell damage compromise function by reducing the audibility of sound signals, synapse loss compromises function by permanently interrupting sensory-to-neural communication for affected neurons. In unexposed ears, loss of synapses is gradual and modest until advanced age. After noise, it is sudden; up to ~50 percent of synapses can be lost acutely with exposure, including many producing only temporary threshold shifts. In ears receiving even a single “synaptopathic” exposure, subsequent losses with age are accelerated and exaggerated. This loss is not revealed as changes in threshold sensitivity and is not detected by routine light microscopy; thus, it remained hidden from our view. It is nevertheless widespread, and the permanent interruption of information flow from hair cells to auditory nerve fibers that results must have important consequences for hearing function, whether thresholds are elevated or not. Here, the aim is to provide a brief review of this hidden injury in noise and aging and to place it into context relative to the threshold-elevating injuries that have been the focus of regulatory, diagnostic and treatment efforts to date.

Permanent Cochlear Injury After TTS-Producing Noise

Figure 1. Post-noise threshold shifts vs. suprathreshold amplitude declines. A two-hour noise exposure (8–16 kHz, 100dB SPL) to 16 week-old CBA/CaJ mice produced large, but temporary threshold shifts in DPOAEs [a] and wave 1 of the ABR [b]. Thresholds are expressed relative to age-, gender- and strain-matched, unexposed controls. Despite threshold recovery, suprathreshold amplitude of ABR wave 1, the far-field response of the cochlear nerve, recovered incompletely, to less than 50 percent of its pre-exposure value [d] in cochlear frequency regions with maximal TTS. In contrast, DPOAE amplitudes [c] returned to normal, suggesting functional recovery of the OHCs. Means ± SE shown; n=7–21/group. Gray bar in a, b denotes the noise exposure band. Modified from (Kujawa and Liberman, 2009).
Noise-induced hearing loss results when exposure to damaging levels of sound injures delicate inner ear structures and compromises vulnerable inner ear functions. The noise-induced threshold sensitivity loss captured by the audiogram can be temporary or permanent, and the underlying injury subtle or dramatic (Kujawa 2009). Permanent threshold losses after noise are associated with permanent cochlear injury, often hair cell loss or damage. In contrast, complete post-exposure recovery of thresholds has been assumed to indicate a recovered ear and a “safe” exposure, with no delayed consequences for hearing function as noise-exposed individuals age (Institute of Medicine, 2006; ACOEM, 2012). These assumptions form the basis for noise exposure regulations, they shape our assessments of noise-induced injury and they guide approaches to treatment and prevention. Our recent work in several mammalian models of noise and aging, however, provides strong evidence that they require significant modification.

Thresholds recover, but neural response amplitudes do not. To examine these assumptions, we undertook a series of experiments in which we studied acute and long-term consequences of noise exposure on the ear and hearing. Below, for example (Kujawa & Liberman 2009), we adjusted the level and duration of a single noise exposure to produce a temporary threshold shift (TTS) on the border of reversibility. We used two complementary techniques, outer hair cell-based distortion product otoacoustic emissions (DPOAEs) and neural-based auditory brainstem responses (ABRs), to assess function, much as we do in the clinic. In the same ears, we quantified hair cells, cochlear neurons and the synapses that allow them to communicate with each other. As shown in Figure 1, a two-hour exposure with an octave-band of noise centered in a region of good hearing sensitivity in our mouse model resulted in a ~30–40dB maximum threshold shift by both DPOAEs (Figure 1a) and ABR wave 1 (Figure 1b) when measured 24 hours after exposure. Threshold sensitivity recovered in the days following exposure and remained stable two and eight weeks later.

Although response thresholds returned to pre-exposure baselines, suprathreshold amplitudes of the DPOAE and ABR wave 1 responses showed a different pattern of recovery with post-exposure time. At 32 kHz, where the TTS was large (Figures 1a, b), the amplitudes of the neural responses displayed a permanent decline relative to the
pre-exposure baseline (Figure 1d). In contrast, the amplitude-versus-level functions for the DPOAEs (Figure 1c), which require only presynaptic processes for their generation, recovered to pre-exposure levels. This persistent reduction in the suprathreshold neural response, in combination with full recovery of response thresholds and DPOAE amplitudes, suggested that the OHCs had recovered function but that there was permanent injury involving IHCs, cochlear afferent neurons or their synaptic connections in basal (high-frequency) cochlear regions. To determine the underlying cause(s), we examined tissues from the same noise-exposed vs. unexposed ears, as is reviewed next.

Hair cells remain, but about half lose connections with cochlear neurons. When assessed in the initial hours, days and weeks following this noise exposure, there was no hair cell loss (OHC, IHC) and no loss of spiral ganglion cells relative to counts in unexposed age-, gender- and strain-matched ears. However, direct evidence for lingering injury after noise was evident in the structures that form the synaptic communications between the IHCs and afferent neurons. To briefly review, the IHC-type I afferent fiber synapse is a specialized conduit through which information about the acoustic environment is transmitted to the neuron and ultimately to the brain. In the normal ear, roughly 95 percent of all cochlear afferent nerve fibers make synaptic connections only with IHCs (Spoendlin, 1972). Each of these fibers has a cell body in the spiral ganglion, a peripheral axon that passes through the osseous spiral lamina and, in the organ of Corti, an unmyelinated terminal dendrite that forms a single synapse with a single IHC. The synapse is comprised of a synaptic ribbon with its surrounding halo of neurotransmitter-containing vesicles on the hair cell side of the synapse (Nouvian et al., 2006) and a post-synaptic glutamate receptor patch on the cochlear nerve terminal containing AMPA-type receptors for the released neurotransmitter (Matsubara et al., 1996; Ruel et al., 2007). The capabilities of these synapses push biological limits in their ability to rapidly and precisely convey graded temporal information about the stimulus and in maintaining this temporal coding fidelity over a large dynamic range (Moser et al., 2006).

To determine the effects of noise at the level of the synapse, we use immunostaining techniques to make these synaptic structures of interest visible and thus quantifiable. The presynaptic ribbon can be immunostained with antibodies to a protein called CtBP2, which is a prominent component of the ribbon (Khimiich et al., 2006). Post-synaptic elements can be immunostained with antibodies to the AMPA-type glutamate receptors (GluA2) found at this synapse (Matsubara et al., 1996) or with antibodies to neurofilament proteins or to a Na+-K+ATPase found in the membranes of the nerve fiber terminals (McLean et al., 2009). Staining with myosin VIIa allows us to better visualize the hair cells. Figure 2a shows a schematic of an IHC with two of its auditory nerve fibers colorized to identify primary structures of interest; Figure 2b shows the paired ribbons and receptor patches as displayed for quantification.

In young, normal ears of CBA/CaJ mice, synapses are most numerous in the mid-frequency region, with roughly 17–18 synapses/IHC (Figure 2c). Noise exposure causes an immediate and dramatic reduction in their number; losses are greatest in regions where the acute TTS is maximum, and in those regions, can reach ~50 percent (Kujawa & Liberman, 2009). Because one auditory nerve fiber makes contact with only one IHC via one synaptic connection, these synapse counts also provide an estimate of the maximum number of auditory nerve fibers that could be carrying information from the cochlea to the brain. ABR wave 1 amplitudes yield permanent and proportional reflections of this loss (Figure 1d; see also Figure 3d) and provide information about the functional integrity of those neurons that remain.

Are all noise exposures synaptopathic? Noise-induced synaptopathy has now been demonstrated for a wide range of both TTS- and PTS-producing exposures (Kujawa & Liberman, * Spectral iQ is a Starkey brand name.
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Not all exposures are acutely synaptopathic, however. By reducing exposure level (sequentially halving the energy by reducing level in 3dB steps) while keeping the frequency content and duration of the noise constant, we identified a TTS-producing exposure (8-16 kHz, 2 h, 91dB SPL) that yielded no persistent ABR wave 1 amplitude decline after threshold recovery (Fernandez et al., 2015). Histologic study of these ears also revealed no acute loss of synapses. We then asked whether there are differences in the long-term consequences of exposures that produce acute synapse loss versus those that do not, on structure and function in aging ears.

Aging vs. Aging after Noise

Delayed effects as ears age after noise. The question of whether ears and hearing age differently after noise has received significant experimental attention. In humans, where investigations have concentrated on changes in pure-tone thresholds, consensus opinions concerning possible delayed effects of noise have not yet emerged (Gates et al., 2000; Rosenhall, 2003; Lee et al., 2005; Cruickshanks et al., 2010). The challenges to such study in the human are significant, where a lifetime’s worth of noise-exposure events must be determined retrospectively, where confounding influences cannot be readily controlled and may not be known, and where large, genetic heterogeneity influences susceptibility to noise, to aging, and their interactions exists, all with the potential to introduce interindividual variability. Thus, although there is general agreement that threshold losses recorded at advanced age may include a contribution arising from prior noise (Gates & Mills, 2005), the question of whether noise can exert delayed effects on “hearing” remains an open one. Hearing function, of course, depends on more than threshold sensitivity/audibility. Recent laboratory experiments done in genetically-identical individuals receiving identical exposures and then aging in identical environments reveal dramatic and progressive synaptic and neural losses to which threshold measures are largely silent. These studies show that prior noise does, indeed, change the ways ears and hearing age, long after the noise has stopped.

Figure 2. Quantifying synapse loss. (a): Colorized schematic shows a single IHC and 2 of the ~20 Type I auditory nerve fibers (ANF) per IHC as they appear in tissue immunolabeled for pre-synaptic ribbons (CtBP2-red), post-synaptic glutamate receptor patches (GluA2-green), post-synaptic ANF terminals (neurofilament or Na-K-ATPase-green) and IHCs (Myosin VIIa, blue); the viewing angle for the x-y projections is noted. (b): To facilitate quantification of synapses, the synaptic region (white box in a) is analyzed using custom software that displays the paired synaptic ribbons and glutamate receptor patches or ANF terminals as x-y projections of the space within 1 µm of each ribbon. (c): Corresponding counts of synapses (means ± SE) in the IHC area from six cochlear locations are given for mice exposed at 16 weeks and assessed 24 hours or 8 weeks after exposure, compared to counts in age-matched, unexposed controls. Results show permanent synaptic loss throughout the basal half of the cochlea. Modified from (Kujawa and Liberman, 2009) and (Fernandez et al 2015).
In the experimental series shown for example here (Fernandez et al., 2015), mice were exposed at 16 weeks (when noise vulnerability has stabilized at adult levels) to an 8–16 kHz band of noise that was delivered at either 100dB (synaptopathic) or 91dB (non-synaptopathic) for 2 hr, as discussed above. They were then held, without additional exposure, for post-exposure times from 1 hour to roughly 2 years of age, along with controls held identically, except for the single noise exposure. Structure and function were assessed at various ages/post-exposure holding times, with results for age-only versus exposed, then aged animals discussed below.

Synapses are most vulnerable in aging ears. Threshold elevations and hair cell losses in unexposed CBA/CaJ mice are minimal until about 2 years of age (Fig. 3a). Thereafter, accelerating threshold deterioration is mirrored by accelerating loss of OHCs; very few IHCs are lost as a function of age in this strain (Sergeyenko et al., 2013).

As with noise injury, however, sensory-to-neural communication shows early compromise in aging ears. Synaptic losses reach roughly 25 to 30 percent in middle age (Fig. 3b), when loss of hair cells is no more than 5 percent and threshold losses are not greater than about 5dB (Fig. 3a). Losses ultimately reach ~40 to 50 percent in the oldest ears. ABR amplitude declines (here, focused on wave 1; Fig. 3c) also begin early and progress steadily, in proportion to the declining synapse counts in the same ears (Fig. 3d). Age-related loss of IHC synapses also is paralleled, with a delay by proportional spiral ganglion cell losses in the same cochlear regions (Fig. 3b). When we compare the ganglion cell losses recorded in these aging mice to those we obtained in an age-graded series of human temporal bones (0–100 years) with full complements of hair cells (Makary et al., 2011), we see that the rate of loss is remarkably similar. Together, these findings suggest that age-related loss of IHC-cochlear nerve synapses may be an early contributor to the performance declines of aging listeners. Are such difficulties exaggerated in aging ears with a history of noise exposure?
Synaptopathic exposure accelerates cochlear aging. Both exposures we used for these experiments produce significant TTS (~30-45dB at 24 hour) as reflected in the DPOAEs and ABR wave 1 responses; frequencies of involvement extended basally, and overall magnitude at 24 hour was greater, for the 100dB noise. For both exposures, thresholds returned to baseline, and as expected, there was no hair cell loss (Fernandez et al., 2015). Despite reversibility of threshold shift and intact sensory cells in these ears, noise-induced degeneration progressed from the IHC-nerve fiber synapse to the nerve cell body with post-exposure time, exaggerating changes otherwise seen with aging alone. Panels of Figures 4a and 4b contrast the gradual and generally mild, age-related loss of synapses with immediate [one hour] and delayed [to 20 months] loss after noise. In the region of maximum TTS (32 kHz; Fig. 4b), synapse loss of ~50 percent is immediately apparent for 100dB but not for 91dB exposure. As ears age after noise exposure, cochlear regions that originally appeared uninvolved in the noise injury [e.g., 11.3 kHz; Fig. 4a] begin to demonstrate synaptic losses in excess of those seen in never exposed animals. Acutely non-synaptopathic (91dB) exposure did not produce this exaggeration of age-related losses, at least to one year.

Subsequent loss of the cell bodies of these neurons within the spiral ganglion is slow (Figure 4c and 4d), progressing over one to two years; for the synaptopathic, 100dB exposure, it reaches dramatically exaggerated levels regarding age-alone controls. In terms of function, however, the communication failure occurred long before, with the loss of the synapse. Such diffuse neurodegeneration, which is not detectable by threshold metrics, raises challenges for evaluation and risk assessment, as discussed below.

Threshold-Based Assessments and Implications for Diagnosis and Management

Our standard, threshold-based tests are silent to the synaptopathy. The pure-tone threshold audiogram serves as a primary tool for quantifying the effects of noise and aging on hearing in clinical and occupational settings. Protocols for the measurement of threshold sensitivity are well established, standardized and validated. Threshold data captured from large-scale studies form the basis for population sensitivity norms to which individuals can be compared. Additionally, models of noise-induced hearing loss risk, which form the basis of every existing noise exposure standard, utilize audiometric threshold data.
Audiometric thresholds provide important functional information relative to signal levels required for detection and how this is impaired in aging and after noise. However, pure-tone audiograms provide imperfect reflections of underlying pathology (Schuknecht & Woellner, 1953; Halpin et al., 1994; Moore, 2004; Lobarinas et al., 2013) and do not capture performance declines that depend on functions in addition to detection. Although audiometric thresholds for pure tones are the current “gold standard” for quantifying noise-induced hearing loss in humans and OAEs can provide additional sensitivity to underlying OHC compromise, these metrics, along with neural response thresholds, can be quite insensitive to even dramatic synaptic and neural loss, as shown here.

High-threshold, low-SR neurons appear most vulnerable. How is it that thresholds can return to normal despite loss of ~50 percent of the nerve fibers connecting hair cells to the brain? It appears that the fibers most vulnerable to noise exposure (Furman et al., 2013) and to aging (Schmiedt et al., 1996) are those that originally had the highest thresholds. Auditory nerve fibers contacting IHCs display a range of sensitivities and, in the normal cochlea, thresholds for fibers with similar best frequencies of response can differ by as much as 60dB (Liberman, 1978). These fibers also differ systematically in their spontaneous rates (SR) of firing, and their sound-driven firing rates vary over different ranges to support a large dynamic range of neural response; high-SR fibers have low (good) thresholds, but their driven rates saturate at levels where high-threshold, low-SR fibers continue to code level with increased firing rate. These features of low-SR neurons make them more resistant to continuous noise masking; as noise level increases, low-threshold, high-SR fibers are the first to be masked, while high-threshold, low-SR fibers are masked last (Costalupes et al., 1984). Thus, although noise-induced and age-related loss of these high-threshold fibers has little impact on overall threshold sensitivity, it is tempting to speculate that such losses, which are well underway by middle age, may contribute to the fairly universal finding that speech-in-noise performance becomes progressively more difficult with age, even when thresholds remain well preserved (Grose et al., 2006; Snell & Frisina, 2000; Ruggles et al., 2012).

Summary

Noise exposure produces “hearing loss” [threshold elevations] and cochlear injury with effects that are largest at acute post-exposure times (Miller et al., 1963). Commonly, a period of rapid post-exposure recovery is followed by one of relative stability, which can be quite long lasting. This threshold recovery and subsequent stability, however, can mask persistent and progressive cochlear synaptic and neural loss. Accumulating evidence suggests that such loss is widespread, occurring as a primary event in at least two common causes of human hearing loss, noise and aging. Noise-induced synaptopathy has now been demonstrated in several mammalian models. There is no reason to suspect that the human will provide an exception to this general finding.

This work has sobering implications for public and occupational health, as opportunities for TTS-producing noise exposures are common in work and recreational activities and as federal guidelines for allowable noise exposure rely on PTS-based assessments of noise injury. It is now quite clear that these assessments fail to detect what is likely the more common consequence of noise exposure; thus, noise is much more dangerous than we previously thought.

Acknowledgments

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References


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Auditory Deprivation: Use It or Lose It

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With regards to hearing loss, have you ever wondered if there’s any truth to the “use it or lose it” theory? Have you ever questioned if hearing impairment means your ability to understand speech or decipher sounds will decrease over time? Researchers have been asking those same questions for more than 40 years. In searching for answers, they examined the phenomenon of “adult onset auditory deprivation,” or the systematic decrease in auditory performance over time as associated with the reduced availability of acoustic information (Arlinger et al., 1996, p. 87S). Or to put it in layman’s terms, the process in which humans struggle to recognize speech due to lack of auditory stimulation (hearing loss).

Despite the large body of literature surrounding adult onset auditory deprivation, there is still a lack of understanding regarding the causes and effects of this phenomenon. This article will discuss the concept of auditory deprivation with a focus on adults with bilateral hearing impairment.

One method of assessing this effect has been through the testing of individuals with bilateral hearing loss who use only one hearing aid. The expectation is that the unaided ear would be deprived of some auditory input as compared to the ear with the hearing aid. Previous studies used already established data to examine the effects of auditory deprivation. However, Palmer and colleagues believe that using retrospective data to measure auditory deprivation is problematic due to a historic lack of documentation on the severity of hearing loss, hearing aid use, and overuse of “outcome measures that are dependent on mid-to-low frequency sounds that the individuals have probably been hearing all along as opposed to outcome measures that tap the newly restored high-frequency hearing” (1998, p. 1709).

In a hallmark study, Silman and colleagues (1984) experimented with amplifying only one ear in adults with bilateral sensorineural hearing impairment. The researchers did not find any significant changes to the audiometric thresholds over time; however, their results did show a significant decline in suprathreshold speech recognition performance in the unaided ears of the patients even when age and hearing threshold effects were removed. The researchers did not see the same decline in the binaurally aided patients. They concluded that there was an auditory deprivation effect and it manifested as a decline in supra-threshold word recognition performance, not as poorer audiometric thresholds.

Gelland and colleagues, in 1987, showed similar declines in speech recognition performance from 86 monaurally aided patients with bilateral sensorineural hearing loss. The results agreed with the findings of Silman and colleagues in that there were no significant threshold shifts between the initial test and the follow-up test; the patients wearing one aid had significantly poorer word discrimination in the unaided ear when compared to their binaurally aided counterparts.

In 1999, Raymond Hurley conducted a prospective study to investigate the prevalence of the unaided ear effect to determine if the amount of hearing loss is a factor in auditory deprivation. This study consisted of 142 participants with bilateral sensorineural hearing loss, 77 were fit with one hearing aid and 65 were fit binaurally. The study
followed these individuals one, three and five years post-fitting. The author observed significant declines in word recognition scores for 25 percent of the monaural subjects compared to only six percent of the binaural subjects experiencing a decline in word recognition performance. The author also concluded those with more severe hearing loss tend to have more auditory deprivation.

Additional studies have failed to show evidence of auditory deprivation. Dalzell and colleagues attempted to replicate the 1984 Silman and colleagues study with 95 monaurally or binaurally fit individuals with moderate sensorineural hearing losses (1992). The researchers examined word recognition performance over time and found no greater decline in word recognition performance for the unaided versus aided ears of the monaurally fit participants. In a study performed by the University of Finland Department of Audiology, 500 participants with symmetrical, sensorineural hearing impairment were recruited and examined before and after monaural hearing aid use, and the results did not reveal significant changes in suprathreshold speech discrimination (Jauhiainen, 2001).

The research on auditory deprivation to date lacks a clear consensus and does not reflect investigations on effects other than measures of word recognition and audiometric thresholds. Clearly, additional research is needed to better understand the effects of auditory deprivation. However, we cannot lose sight of the fact that there are several studies that show an auditory deprivation effect when comparing suprathreshold word recognition performance of aided and unaided ears in monaurally fitted individuals. This evidence suggests that as clinicians we should consider each patient’s case individually and monitor for significant changes in suprathreshold measures of performance of monaurally aided patients. Word recognition measures in quiet may not be sensitive to auditory deprivation, and more challenging baseline and periodic measures may be appropriate. If there are significant declines in suprathreshold measures unilaterally, an appropriate treatment plan should include amplification in both ears.

References


The Relationship Between Hearing Ability & Cognition/Dementia

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It is well known that older individuals, with or without hearing loss, have difficulty understanding speech in challenging listening environments. Cognitive abilities explain why, in some listeners and in some listening situations, speech is easily and effortlessly understood, while in other situations, understanding speech takes significant effort and consumes significant cognitive resources (Akeroyd, 2008; Rönnberg et al., 2008; Tun et al., 2012).

Cognitive abilities are the mental processes involved in crystallized and fluid intelligence. Crystallized intelligence encompasses world knowledge, vocabulary, and long-term memory. Fluid intelligence involves the cognitive constructs of working memory, speed of processing, attention/inhibition, and central executive function (Edwards, 2002). While some decline in cognitive function is normal with aging, when decrements in memory, reasoning, planning and/or behavior become severe enough to reduce a person’s ability to perform everyday activities, the diagnosis is dementia (Santacruz & Swagerty, 2001).

Similar to age-related hearing loss, dementia is often progressive, starting out as a mild cognitive impairment (MCI) which can gradually become worse (Petersen, 2004). While over 20 percent of adults over the age of 70 are estimated to have an MCI, 60–80 percent of them are likely to progress into a dementia, including the most prevalent type—Alzheimer’s disease (Petersen et al., 2009). Of particular concern to hearing healthcare professionals are the research findings that indicate that older adults with hearing loss are significantly more likely than adults with normal hearing to develop dementia (Lin et al., 2011).

Indeed, in the United States, where age-related hearing loss is the third most common chronic disability, it is estimated that 5.2 million adults aged 65 years and older have Alzheimer’s disease or a related dementia. By 2025 this number is expected to increase by 40 percent to 7.1 million (Thies & Bleier, 2013). Given these statistics, there is a critical need to better understand the hearing-cognition relationship and its implications for practice.

Current literature supports the relationship between cognition and hearing (Bush et al., 2015), but why this occurs is unknown. One possibility is that the widespread neural degeneration in the brain that causes cognitive impairment also affects hearing function (Anstey et al. 2001). Another possibility is that as hearing loss occurs, greater resources are allocated to auditory processing, leaving fewer resources available for other cognitive
The Relationship Between Hearing Ability & Cognition/Dementia

processes [Schneider & Pichora-Fuller, 2000]. It may also be that hearing-related communication difficulties result in social isolation, which concomitantly impacts negatively on brain function (Mick et al., 2014). Finally, it is possible that these factors combine to result in the common complaint of older individuals — “I can hear you, but I can’t understand.”

Whatever the cause, the link between hearing and cognition may offer a starting point for intervention, with perhaps the simplest approach being amplification. While the use of hearing aids may not be a “cure” for dementia, it has been hypothesized that their use may slow the rate of cognitive decline (Lin et al., 2013) and possibly reduce daily mental fatigue [Hornsby, 2013; Sarampalis et al., 2009]. While it is also important to consider how cognitive function may impact the selection of hearing aid parameters [Lunner & Sundewall-Thorén, 2007] and the success of intervention [Pichora-Fuller & Singh, 2006], hearing aid use in adults with dementia reduces caregiver stress [Palmer et al., 1999]. In sum, hearing healthcare providers need to consider that their interventions may help preserve cognitive abilities and also the impact of cognitive decline on intervention options.

**Sources:**


**By 2025 the number of adults aged 65 and older with Alzheimer’s disease or a related dementia is expected to increase by 40%.**

**Adults with mild loss**
- 2x more likely to develop dementia

**Adults with moderate loss**
- 3x more likely to develop dementia

**Adults with severe loss**
- 5x more likely to develop dementia

**Those with untreated hearing loss experience a 30-40% greater decline in thinking abilities compared to those without hearing loss.**
References


Infographic Sources


Today, it is very common to pick up a journal or attend a continuing education session where the topic of dementia and its association with hearing loss is discussed. Harvey Abrams, Ph.D. recently interviewed Kathy Pichora-Fuller, a leading authority on the subject, to see how clinicians may make use of concepts in diagnosis and treatment of patients with hearing difficulty.

**IN (Innovations):** Why do you think there has been so much recent attention devoted to the relationship between auditory and cognitive aging?

**KPF (Kathy Pichora-Fuller):** The population is aging, so audiologists will be seeing older patients and more of them, than ever. By 70 years of age (typical age of first-time hearing aid users), about 1 in 20 will have mild cognitive impairment, and 1 in 10, dementia. People with hearing loss are at even higher risk of cognitive decline (Albers et al., 2015).

**IN:** You often refer to “successful aging” in your writings and presentations. Can you explain that with respect to the role of hearing loss and hearing rehabilitation?

**KPF:** A basic idea about aging is that there are both losses and gains. Successful aging happens when there is a positive balance of gains versus losses. Effective hearing rehabilitation to counteract age-related hearing loss is an excellent way to promote successful aging (Pichora-Fuller, 2014). By helping older adults to maintain good communication and remain socially active, audiologists can help them to stay healthier for longer.

**IN:** You’ve written about the Selective Optimization with Compensation model. How does that model relate to hearing loss?

**KPF:** The Selective Optimization with Compensation (SOC) model (Baltes & Baltes, 1990) provides a framework for successful aging that is highly compatible with hearing rehabilitation. First, individuals select the goals they wish to prioritize. For example, audiologists can help patients identify listening goals by using tools such as the Client-Oriented Scale of Improvement (COSI) (Dillon, Birtles, & Lovegrove, 1999). Then, individual patients optimize by practicing skills to achieve their selected goals, [e.g., they might use a hearing aid, do auditory training or learn conversational strategies]. They compensate by drawing on other relatively intact abilities to offset declines so that selected goals can be achieved, [e.g., vision could be used to lip-read or read captions]. The SOC model provides a way to frame hearing rehabilitation in the context of successful aging.

**IN:** What mechanisms might explain the relationship between auditory and cognitive impairment, and could hearing rehabilitation make a difference?

**KPF:** The mechanisms are not yet known; they might be social, cognitive and/or biological (Pichora-Fuller, Mick, & Reed, 2015). Insofar as cognitive declines are associated with social isolation and reduced social participation, hearing rehabilitation could help by maintaining social participation and preventing social isolation. Hearing rehabilitation could also reduce some of the long-term negative consequences of cognitive
overload by making it easier for older adults to process auditory information. If the mechanisms are biological, then it seems unlikely that hearing rehabilitation could alter the course of dementia; however, it could still be advantageous to take the greater risk of dementia in those with hearing loss into consideration (Dupuis et al., 2015).

**IN:** Why is it important that audiologists and other hearing healthcare providers assess the cognitive status of their patients?

**KPF:** For patients who have normal cognition and similar audiograms, cognitive measures (e.g., working memory span) could provide new insights into why some listeners experience more difficulties than others. Such information about individual differences in cognitive processing that affect everyday listening performance could guide recommendations about rehabilitation options. Cognitive measures also offer promise as new outcome measures that could enable audiologists to evaluate if interventions have increased a patient’s ease of listening (e.g., by showing pre- versus post-intervention improvements in a patient’s ability to understand and/or remember more of the words he or she heard). Of course, when planning hearing rehabilitation programs, it is also important to consider the possible influences of many nonauditory factors, including comorbid health conditions such as impairments in vision, dexterity and cognition.

**IN:** What tools should the audiologist be using to assess cognition in a busy practice?

**KPF:** To date, a number of tests assessing working memory have been used in much of the research investigating the connections between auditory and cognitive processing. Tests suitable for clinical use to evaluate working memory in patients with hearing loss are being developed, but more research is needed before these experimental measures will become available as standardized tests. Information about comorbid health issues, including information about dementia, could be provided in referral documentation, obtained from the patient or family members during history-taking, observed by the audiologist during appointments, or possibly discovered by conducting one of the widely used tests that have been developed to screen for dementia.

**IN:** Should there be specific training requirements for audiologists and other hearing professionals in order to properly assess cognitive function for the purpose of developing an appropriate treatment plan?

**KPF:** Let’s assume that testers would learn and follow standardized methods for conducting cognitive tests, including carefully controlling noise in the test environment and the audibility of test materials because the accuracy of cognitive
assessment can depend on how well the patient is able to hear during testing. More importantly, audiologists and other hearing professionals would need to learn how to use the results to plan treatments. Specifically, awareness of the patient’s cognitive status would need to be considered in relation to the goals of the patient and the potential of the patient to optimize and compensate to achieve those goals. In particular, rehabilitation for those who have both hearing and cognitive losses will need to be customized to meet the needs of the individuals and their caregivers (e.g., patients may need simpler technologies or assistive devices, more support from family or other caregivers, and more follow-up appointments to ensure success).

**IN:** How do patients react to cognitive testing? Could frustration during testing affect their attitude toward the clinician and treatment?

**KPF:** Audiologists often conduct difficult tests (e.g., speech-in-noise threshold testing), so they should be able to extend this expertise to other potentially difficult testing situations. Patients need to know why and how test results will be used to help them be more successful. Relating testing back to the patient’s problems should help to explain the relevance of cognitive testing. Patients often report that they can “hear” but cannot understand easily, experience difficulty following rapid speech, have trouble keeping track of multiple talkers during group conversations or have difficulty listening while multitasking. Patients who have these complaints are already on their way to realizing that the brain is working hard to understand, focus attention and remember what was said when they are struggling to hear.
Cognitive Decline and the Role of Hearing Aids

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Brief History of Cognitive and Auditory Research

Research scholars began investigating the interaction between hearing and cognition more than three decades ago (Duquesnhy, 1983; Bronkhorst & Plomp, 1988). In fact, groundbreaking research that studied cognition and hearing was conducted in the 1950s by Broadbent, Cherry and others in Cambridge (Broadbent, 1958). Following World War II, scientists embarked on studying the connection between cognitive and auditory processing; however, it was not until the 1990s that the study of cognition and hearing started to gain momentum and the number of publications linking hearing and cognition started to remarkably grow (Arlinger et al., 2009).

Because listening and understanding speech is a complex task that involves a wide variety of sensory and cognitive processes, researchers have always sought to understand how the brain builds meaningful descriptors of the auditory world. Further, the critical implications and impact of cognition-audition interactions on the diagnosis and treatment of hearing loss have lead researchers to combine their efforts in studying auditory and cognitive functions. As reported by Arlinger and colleagues, 2009, there are numerous factors that have contributed toward the convergence of auditory and cognitive research—some of them are listed in Table 1.

Table 1

<table>
<thead>
<tr>
<th>Contributing Factor</th>
<th>Reference</th>
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<tbody>
<tr>
<td>The need to understand the listener’s performance in real-world environments</td>
<td>Bregman, 1990; McAdams &amp; Bigand, 1993; Neuhoff, 2004</td>
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<tr>
<td>The need to understand how aging and impairments change performance</td>
<td>Schneider &amp; Pichora-Fuller, 2000; Wahlin, MacDonald, de Frias, Nilsson &amp; Dixon, 2006</td>
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<td>How to design new communication technologies using advanced signal processing</td>
<td>Edwards, 2007</td>
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<td>How to design educational and rehabilitation programs that improve performance</td>
<td>Kraus, McGee, Carrell, King, Tremblay &amp; Nicol, 1995; Tremblay, 2007</td>
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<tr>
<td>The emergence of new research tools, such as eye-movement tracking devices and advanced physiological methods</td>
<td>Durlach &amp; Mavor, 1995; Allopenna et al., 1998; Belin et al., 1999; 2000</td>
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<tr>
<td>The proposal of new models for cognition-audition interaction</td>
<td>Holt &amp; Lotto, 2008; Stenfelt &amp; Ronnberg, 2009</td>
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Connection Between Hearing Loss and Cognitive Decline

A large and growing body of literature exists on the connection between hearing loss and cognitive decline. Hearing loss has been associated with greater declines in cognitive function in older adults than in their counterparts without hearing loss (Lin et al., 2013; Lin et al., 2004; Lindenberger & Baltes, 1994; Ulhmann et al., 1989; Tay et al., 2006; Baltes & Lindenberger, 1997). For example, audiometric hearing thresholds have been linked to cognitive performance on test-specific tasks involving memory and executive and global functions in older adults (Lin et al. 2011a, b).
More specifically, hearing loss has been linked to dementia. Dementia was found to be more prevalent in older adults with hearing loss than those without (Uhlman et al., 1986; 1989). Pure-tone thresholds (Lin et al. 2011a, b; 2013) and scores on tests of auditory central processing (Gates et al., 2003; 2010; 2011) were found to be correlated with incident dementia. The study by Lin and colleagues (2011a) found that older adults with hearing loss are two to five times at risk of developing dementia compared to their counterparts with normal hearing. They also found that with every 10dB increase in hearing loss, there was a 20 percent increased risk of developing dementia (Lin et al., 2011b). Further, Gurgel (2014) reported that the mean time to develop dementia was found to be 10.3 years in individuals with hearing loss as opposed to 11.9 years for those without.

This link between hearing impairment, cognitive performance and incident dementia has lead epidemiologic researchers to suggest that hearing loss may be a risk factor for cognitive decline (Lin et al. 2011a, b; 2013). The results of these studies demonstrate that indeed there is a link, but it is secondary to the predictive effects of hearing loss. There is no clear proof that hearing loss is the cause of the reduced cognitive function, but indirect evidence from many studies supports this hypothesis.

Table 2 lists some of the studies demonstrating the link between hearing loss and cognitive decline.

<table>
<thead>
<tr>
<th>STUDY</th>
<th>N</th>
<th>COGNITIVE MEASURE</th>
<th>COGNITIVE ABILITY TESTED</th>
<th>RESULTS</th>
<th>OUTCOME</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lin, 2011</td>
<td>605</td>
<td>Digital Symbol Substitution Test (DSST)</td>
<td>Executive function and psychomotor processing</td>
<td>Greater hearing loss was associated with lower scores on the DSST, hearing loss was positively associated with cognitive functioning.</td>
<td>Hearing loss is independently associated with lower scores on the DSST.</td>
</tr>
<tr>
<td>Lin et al., 2013</td>
<td>1984</td>
<td>3MS and DSST</td>
<td>Global and executive functions</td>
<td>Rates of cognitive decline and the risk for incident cognitive impairment were linearly associated with the severity of an individual’s baseline hearing loss.</td>
<td>Hearing loss is independently associated with accelerated cognitive decline and incident cognitive impairment in older adults.</td>
</tr>
<tr>
<td>Lindenberger &amp; Baltes, 1994</td>
<td>156</td>
<td>Digit Letter Test, Figural Analogies, Practical Knowledge, Activity Recall and Letter S</td>
<td>Speed, reasoning, knowledge, memory and fluency</td>
<td>Auditory acuity explained 34.6 percent of the reliable total variance in intellectual functioning.</td>
<td>Sensory functioning is a strong late-life predictor of individual differences in intellectual functioning.</td>
</tr>
<tr>
<td>Uhlmann et al., 1989</td>
<td>100</td>
<td>Mini-Mental State Examination (MMSE)</td>
<td>MMSE score used as an indicator of the severity of dementia</td>
<td>The prevalence of a hearing loss of 30dB or greater was significantly higher in cases than in controls. Greater hearing loss was associated with a higher adjusted relative odds of having dementia.</td>
<td>Hearing loss was also significantly and independently correlated with the severity of cognitive dysfunction.</td>
</tr>
<tr>
<td>Baltes &amp; Lindenberger, 1997</td>
<td>516</td>
<td>Digit Symbol Substitution, Figural Analogies, Paired Associates, Practical Knowledge and Letter S</td>
<td>Reasoning, memory, perceptual speed, knowledge and fluency</td>
<td>All five intellectual abilities were strongly correlated to sensory (including hearing) functioning than to socio-biographical factor.</td>
<td>Results suggest that aging-induced biological factors are a prominent source of individual differences in intelligence in old and very old age.</td>
</tr>
</tbody>
</table>

Table 2
Explanatory Hypotheses

Two main explanatory hypotheses have been proposed to explain the mechanisms underlying the association between hearing loss and cognitive decline. The first hypothesis suggests that hearing loss and cognitive decline share a common neuropathologic origin, such as age-related neurodegenerative changes caused by microvascular diseases and inflammation (Lindenberger, & Baltes, 1994; Baltes & Lindenberger, 1994). In other words, this model proposes that hearing loss and cognitive impairment in older adults may share the same underlying pathology.

The second hypothesis, known as the “cascade” hypothesis, argues for a causal relationship between hearing loss and cognitive decline, and suggests that hearing loss exists in conjunction with or interacts with other risk factors to accelerate cognitive loss (Lin et al., 2013).

According to this hypothesis, hearing loss may impact cognition in three main ways:

1. Long-term auditory deprivation may result in reduced cognitive function (Birren, 1964; Wahl & Heyl, 2003).

2. One possibility is that the link between hearing loss and cognitive decline is mediated by lifestyle factors. Hearing loss may result in reduced participation in social leisure activities and in withdrawal from social interactions. In fact, hearing loss is independently associated with social isolation and depression (Gates & Mills, 2005). There is also a connection between social isolation and depression and cognitive decline (Plassman et al., 2007, Steffens et al., 2006). The cascade hypothesis suggests that social isolation can lead to depression and other psychological consequences that may affect cognitive function.

3. Hearing loss may result in increased compensatory cognitive effort exerted to fill in the gaps caused by missing speech information, which may result in a shortage of cognitive resources dedicated for encoding speech into memory in an already reduced working memory in older adults (Schneider et al., 2010; Tun et al., 2012; Pichora-Fuller & Singh, 2006).

A third hypothesis suggests that the reduced cognitive function seen in older adults can be confounded by hearing loss. Individuals with hearing loss are at a disadvantage if cognitive tests are to be administered using auditory stimuli (Gussekloo et al. 2005). This hypothesis seems unlikely because the link between hearing loss and cognitive decline remains unaltered whether cognitive tests are administered through the auditory or visual modality (Tay et al., 2006). In order to account for hearing loss when

Cascade Model

Common Cause Model
administering cognitive tests, an effort should be made to ensure that the auditory signal is presented at an adequate level. In addition, visual stimuli can be used as an alternative to auditory stimuli when testing cognitive performance in patients with hearing loss.

**Hearing Aids and Cognition (Review of Studies)**

A small number of studies have examined the effects of hearing aid use on cognitive performance in older listeners. The findings of these studies were inconclusive. The inconclusive results may be attributed to differences in methodology. For example, some of the cognitive tests used in these studies employed auditory stimuli; therefore, improvements in cognitive function may be due to improved audibility with hearing aids. Also, there was a lack of information on whether amplification was well fitted and whether the hearing loss had been appropriately compensated for.

Up to date, there is a lack of strong evidence on the long-term protective effects of hearing aid use against cognitive decline (Kalluri & Humes, 2012). Further research is needed as to whether hearing aid use could prevent, reduce, arrest or reverse cognitive decline in older adults.

Table 3 lists some of the studies that looked into the effects of hearing aid use on cognitive function in older adults.

<table>
<thead>
<tr>
<th>STUDY</th>
<th>COGNITIVE MEASURE</th>
<th>HEARING AID USE</th>
<th>SHOWED IMPROVEMENT IN COGNITIVE FUNCTION?</th>
<th>MODE OF ADMINISTRATION</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mulrow et al., 1990</td>
<td>Short Portable Mental Status Questionnaire (SPMSQ)</td>
<td>4 months</td>
<td>Yes</td>
<td>Verbally</td>
<td>The SPMSQ is commonly used as a simple screening tool that is not designed to detect changes in cognitive function.</td>
</tr>
<tr>
<td>Acar et al., 2011</td>
<td>MMSE</td>
<td>3 months</td>
<td>Yes</td>
<td>Verbally</td>
<td>Cognitive test was administered verbally</td>
</tr>
<tr>
<td>Lin, 2011</td>
<td>DSST</td>
<td>At least once a day over the preceding year</td>
<td>Yes; HA use was positively associated with cognitive function</td>
<td>DSST is a nonverbal test.</td>
<td>Results must be interpreted with caution because of the small number (n = 13) of participants using hearing aids.</td>
</tr>
<tr>
<td>Choi et al. 2011</td>
<td>Visual Verbal Learning Test (VVLT)</td>
<td>6 months</td>
<td>Yes, significant changes in the total score</td>
<td>Visual</td>
<td>Due to the small sample size, the findings of this study should be interpreted with some caution (n = 18)</td>
</tr>
<tr>
<td>Tesch-Römer, 1997</td>
<td>Executive function and memory</td>
<td>6 months</td>
<td>No</td>
<td>Verbal and nonverbal</td>
<td>Since the subjects were not randomly assigned, six months of HA use is too short to cause any significant change.</td>
</tr>
<tr>
<td>Van Hooren et al., 2005</td>
<td>Processing speed, reasoning, memory, knowledge and verbal fluency (broad range of cognitive tests)</td>
<td>12 months</td>
<td>No improvement was observed.</td>
<td>Verbal and nonverbal</td>
<td>Older adults did not pursue intervention for eight to 12 years after the first notice of a hearing impairment</td>
</tr>
<tr>
<td>Wong et al. 2014</td>
<td>MMSE</td>
<td>6.9 (4.3) years</td>
<td>No. Out of the eight domains measured on the MMSE, auditory factors [i.e. duration of hearing aid use, aided noise composite SRTs, and aided sound-field thresholds] predicted the scores on five MMSE domains that required understanding of the verbal instructions</td>
<td>Auditory and visual</td>
<td>Monaural hearing aid fittings</td>
</tr>
</tbody>
</table>
Implications on Clinical Practice

It is crucial for clinicians to learn whether earlier or better hearing healthcare could arrest, reverse or slow down the progression of cognitive decline in older adults (Pichora-Fuller, 2010). Further, older adults with hearing loss tend to wait a long time before they wear hearing aids. Therefore, it is important for hearing healthcare professionals to know whether or not to make evidence-based recommendations to try hearing aids early on.

In order to address these clinical questions and many more, a carefully planned longitudinal study is still required to examine the casual relationship between the long-term use of appropriately fitted hearing aids and the progression of cognitive function.

References


Pichora-Fuller, M. K. (2010). Using the brain when the ears are challenged helps healthy older listeners compensate and preserve communication function (pp. 53-63). In L. Hickson (Ed.). Hearing care for adults. Phonak: Stäfa, Switzerland.


The relationship between hearing loss and cognitive ability has been the focus of research across multiple disciplines, causing a convergence among psychology, gerontology, epidemiology and audiology. This work has resulted in meaningful observations, allowing us to determine that people who suffer from untreated hearing loss exhibit greater cognitive impairment than their normal hearing peers or their peers with appropriately treated hearing loss. Many questions remain, including the impact hearing aids have on cognitive ability.

Today, we understand that patients with differing cognitive abilities will demonstrate different hearing aid outcomes. As an example, older adults with reduced cognitive ability are more susceptible to distortions in sound quality from hearing-aid processing (Arehart et al., 2013). These same patients also show mild benefits of slower compression, when compared to faster compression time constants (Souza & Sirow, 2014). In both cases, providing a hearing aid that is less prone to distorting the speech signal when amplifying was beneficial to the cognitively impaired patient.

Amplitude compression in hearing aids isn’t the only signal processing technology that can interact with cognitive ability. Features like directional microphones and digital noise reduction allow patients to perform difficult listening tasks while also efficiently completing secondary tasks. Even in cases where individuals scored 100 percent on a speech-in-noise test, performance of a secondary task might improve when these features are activated (Wilson, 2011; Desjardins & Doherty, 2014).

As with almost any perceptual ability, the details of these interactions are far more complicated than could be addressed in this article. Take the case in which the cognitive demands of listening change with hearing aid experience.

Faculty at the University of Linköping in Sweden have recently completed a study in which cognitive abilities were compared to hearing aid outcomes at the time of hearing aid fitting and incrementally over the following six months. Data collection began four months prior to being fitted with hearing aids, at which time patients completed tests of cognition and speech recognition; the latter were administered again at the hearing aid fitting appointment and at three month intervals afterward. After six months of hearing aid use, speech recognition had improved, but correlations between cognitive measures and speech recognition had declined. In other words, at the time of hearing aid fitting, cognitive ability held a stronger relationship to speech recognition. These observations suggest that the cognitive requires of listening decreased as patients adapted to hearing aid use.

As clinicians we should be prepared to inform patients that our most recent research has begun to confirm that hearing aid use attenuates some negative effects of cognitive decline. Many details of these benefits remain unclear; we can speak of them in generalities but should not take the leap to suggest that hearing aid use affects onset of cognitively-related disorders such as dementia or Alzheimer’s disease.
References


