

# HEARING LOSS WITH AGE IS NOT NORMAL PHYSIOLOGICAL AGING: IT'S NOISE-INDUCED HEARING LOSS

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## INTRODUCTION

The World Health Organization defines hearing loss as a 25-40 dB decrease in hearing.<sup>1</sup> Hearing loss is widely regarded as an inevitable result of aging, exemplified by the terms *presbycusis* and *age-related hearing loss* (ARHL). A careful review of the literature, however, strongly suggests that this is not normal physiological aging, but is largely caused by cumulative excessive lifetime noise exposure:

The biological bases of aging are still not precisely known. Regardless of the mechanisms, the effects of aging are readily apparent even to the most casual observer:

- As humans reach old age, usually defined as 60 or 65, men and women gain weight.
- Hair thins and turns gray.
- Skin sags and wrinkles and age spots appear.
- Performance times in running events increase.
- Hearing gets worse.

## Is ARHL really noise-induced hearing loss (NIHL) in older persons?

That is the question this critical analysis attempts to answer. Why does this matter? If a clinical condition common in old age is the result of normal physiological aging, e.g., thinning gray hair, nothing can be done to avoid it. If the condition can be prevented, behavior changes may delay the onset or prevent development of the condition entirely. Reducing lifetime noise exposure might prevent ARHL

## NORMAL VS. ABNORMAL AGING

Many changes prevalent in older humans, all once thought to be part of normal aging, are now known to be the result of abnormal or pathological aging. Among these are obesity, muscle weakness, diabetes, hypertension, heart disease, and dementia. Absent specific genetic syndromes, abnormal aging can be ascribed to 4 basic causes: 1) exposures, 2) dietary intake, both quantity and quality, 3) disuse atrophy, and 4) suboptimal medical care. Suboptimal care refers not to poor quality care, but rather to accepted standard care that is later shown to have been inadequate. The successive lowering of blood pressure target goals for treatment, with better clinical outcomes in subsequent years, is perhaps the best example of this.

## SIX AREAS OF RESEARCH SUPPORT THE HYPOTHESIS THAT ARHL IS NIHL IN ELDERLY INDIVIDUALS

1. **Studies of hearing in isolated populations not exposed to noise** done in the 1960s found hearing to be well-preserved into old age.<sup>2</sup> These studies can be criticized for the testing protocols, equipment used, uncertainty about the actual ages of the subjects, and unknown history or ear or other infections, but they offer the only insights available about what happens to human hearing without excessive noise exposure. [See Figure 1.]
2. **Occupational studies** correlate occupational noise exposure with hearing loss.<sup>3</sup> Exposure to greater amounts of noise for longer periods of time causes more hearing loss, not just in workers
3. **Females have better hearing than males** beginning in the second decade of life.<sup>4</sup> Hearing is the same in both genders at birth, but boys and men engage in recreational and then occupational activities exposing them to more noise. There is a protective effect of estrogen on hearing<sup>5</sup>, but it does not prevent NIHL in women working in textile mills.<sup>6</sup>

4. **Variations in the prevalence of hearing loss** in different population groups must be explained. If hearing loss was due to normal physiological aging, one would expect to find similar hearing loss prevalence in all populations, just as almost all older individuals anywhere in the world have gray hair. The differences in hearing loss prevalence could be due to genetics, ear or other infections, or exposure to ototoxic drugs or chemicals, but published evidence does not support any of these putative causes. The most likely cause is different amounts of noise exposure in different population groups..

5. **If hearing loss was part of normal aging**, one might expect it be distributed across all frequencies bilaterally. Instead, NIHL occurs in the frequencies to which the ear is exposed, with occupational studies showing asymmetric hearing loss greater in the ear closest to the noise source.<sup>7</sup> For example, dentists have unilateral high-frequency hearing loss in the ear nearest the high-speed turbine powering the drill.

6. **Basic science research** shows that noise causes biochemical genetic, and structural changes changes in cells and subcellular structures in the auditory system leading to organelle and hair cell damage and death.<sup>8</sup> Animal studies confirm human studies based only on audiograms, showing that noise exposure at specific frequencies damages the cochlear hair cells that receive these frequencies.

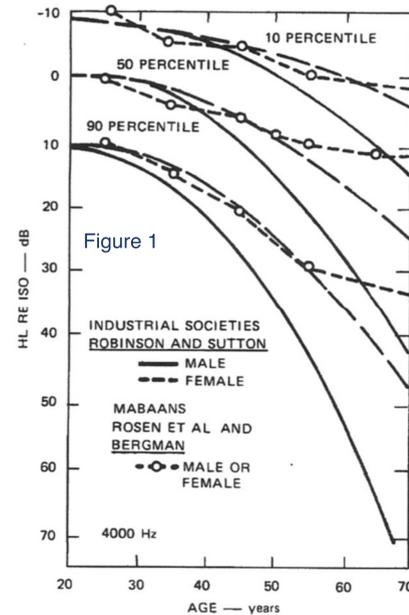


FIG. 11. HLs for young people from the studies of Mabaans and industrialized societies, and the differences in HLs for the older age groups. Test frequency, 4000 Hz. The raw data points for the Mabaans are plotted 10 dB higher (re: 15-25-year-olds) than reported on assumption of audiometer calibration error [see text].

Figure 1. Reproduced from Reference 4, Kryter KD. Presbycusis, sociocusis and nosocusis. *J. Acoust. Soc. Am.* 1 June 1983; 73 (6): 1897-1917. <https://doi.org/10.1121/1.389580> with permission of the Acoustical Society of America. Copyright 1983 Acoustical Society of America.

## WHAT COULD CAUSE ARHL?

Those using the terms presbycusis or ARHL state that hearing loss in old age is multifactorial, representing “the resulting phenotype that results from the cumulative effects of all these factors that exert themselves over the lifetime.”<sup>9</sup> They often cite nutritional, vascular, toxic, genetic, toxic, genetic, and “other” factors, but rarely mention.

and appear to downplay the importance of noise exposure. Dietary intakes vary widely, but it is unlikely that any specific vitamin or trace element deficiencies cause ARHL. Atherosclerosis is widespread, but the impact of vascular disease on hearing isn't clear. Could ototoxic drugs play a role? The use of potentially ototoxic drugs is widespread, but the prevalence of hearing loss from ototoxic drugs has not been determined. Many genes are associated with hearing loss, but genome-wide association studies have not yet yielded evidence of the prevalence of genetically caused hearing loss in the general population. The only other likely factor causing ARHL is cumulative lifetime noise exposure.

Decades of research have attempted to establish a correlation between anatomic studies of human hearing loss, limited to post-mortem examination of temporal bone samples, and research done on animal models of hearing loss. A recent study, however, suggests that there are important differences between the animal models and humans, with the animal studies not being directly relevant to ARHL in humans. As Wu et al. stated,

“Our analysis of over 120 autopsy specimens shows that inner-ear sensory cell loss can largely explain the audiometric patterns in aging, with minimal contributions from the stria vascularis, the “battery” that powers the inner ear, previously viewed as the major locus of age-related hearing dysfunction.... Our data also show that hair cell degeneration in aging humans is dramatically worse than that in aging animals, suggesting that the high-frequency hearing losses that define human presbycusis reflects avoidable contributions of chronic ear abuse to which aging animals are not exposed.”<sup>10</sup>

Additional evidence that hearing loss is not part of aging comes from a longitudinal study of hearing in Charleston, South Carolina.<sup>11</sup> Details of the subject ages in each category are not available, but the mean age of all subjects was 69.9 years with 94.4% of subjects >60. The Older-Normal hearing threshold levels up to 4 kHz in the upper left audiogram in Figure 2 are remarkably similar to those cited by Kryter for the Mabaans in Figure 1. This would appear to confirm the hypothesis that a slight decrease in hearing with age, not meeting the WHO criterion for hearing loss<sup>1</sup>, is normal, and that as shown in the other audiograms in Figure 2, anything >25 dB hearing loss is the result of pathological aging.

## WHY DOES HEARING LOSS MATTER?

Why does hearing loss matter? Hearing has been called the social sense, important for humans to connect with one another. The impacts of ARHL are underestimated for several reasons. First, the economic impact is limited because it becomes prevalent only in the seventh decade of life, when people are largely retired. Second, ARHL is invisible. Unlike those with vision impairments, people with ARHL are able to walk, drive, and complete their activities of daily living without apparent difficulty. Third, the onset of ARHL is usually gradual. People engage in a variety of compensatory strategies as their hearing worsens over time, cupping a hand over the ear, trying to find a quiet corner of a restaurant, eating early or late when the restaurant is quieter, asking someone to repeat what was said, pretending to understand by nodding or saying, “Uh, huh,” and finally, when communication becomes too difficult, limiting social interaction. Fourth, it is commonly believed that unlike cancer, “Nobody dies from hearing loss.” Unfortunately, the common wisdom is wrong. Hearing loss is associated, most likely causally, with falls, accidents, social isolation, dementia, and, increased mortality.<sup>12</sup>

Prevention of disease is better and almost always less expensive for the individual and society.<sup>13</sup> This is certainly true for ARHL, which is sensorineural hearing loss. The most common treatment is

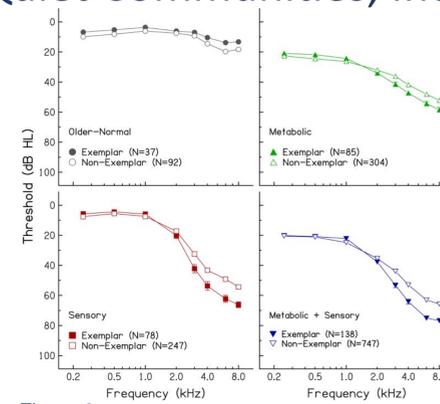


Figure 2

Mean audiograms and standard errors of exemplars (filled symbols) and non-exemplars (open symbols) in four audiometric phenotypes. Reproduced with permission from Dubno JR, Eckert MA, Lee FS, Matthews LJ, Schmiedt RA. Classifying human audiometric phenotypes of age-related hearing loss from animal models. *J Assoc Res Otolaryngol.* 2013 Oct;14(5):687-701.

amplification, i.e., hearing aids, with cochlear implantation reserved for the profoundly hearing-impaired. Hearing aids are costly, and in most countries, including the United States, only a minority of people who might benefit from hearing aids are able to afford them.

Perhaps more importantly, hearing aids do not restore normal hearing.<sup>14</sup> Unlike lenses used to treat myopia or presbyopia, which provide a visual correction, hearing aids do not provide an auditory correction. For those with sensorineural hearing loss, hearing aids provide much less benefit than desired in noisy environments, e.g., restaurants or parties, where people with hearing loss really want to be able to understand speech. Even in countries where the cost of hearing aids is largely covered by the national health system, many people don't want them.<sup>15</sup> In addition, there is a stigma attached to hearing loss, and to wearing hearing aids.<sup>16</sup> For those who have acquired them, there is significant non-usage.<sup>17</sup> Glasses work well. No one likes wearing them or contact lenses, but in industrialized countries almost everyone who needs them acquires and uses them. Unfortunately, hearing aids still do not work well in ambient noise.

## CONCLUSION

The combination of observational and epidemiologic studies showing a clear dose-response relationship between noise exposure and hearing loss, with basic science studies explaining how noise damages auditory cells, strongly suggests that hearing loss with age is not caused by normal physiological aging but is rather the cumulative effect of excessive lifetime noise exposure on the ear. The proof of this hypothesis would require well-done longitudinal studies measuring noise exposure, ideally continuously, correlated with tests of hearing conducted over decades. These studies will never be done.

Based on the available evidence, it appears that hearing loss with age is not normal physiological aging but is largely the result of noise exposure, i.e., ARHL is really NIHL. Terminology matters because words influence how people think. Livingstone-Banks noted that, “how we define diseases has an impact on how (and how effectively) we research and treat them.”<sup>18</sup> The words, “and how we prevent them” should be added.

To accurately reflect causality, ARHL should be renamed *sociocusis* or *noise induced hearing loss in elderly individuals*. The terms *presbycusis* and *age-related hearing loss* should be relegated to the proverbial dustbin of history.

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