Trajectory of Metabolic and Sensory Components of Age-related Hearing Loss in 1.7 Million US Workers From the NIOSH Occupational Hearing Loss Surveillance Project

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Introduction

Age-related hearing loss (ARHL) results from cumulative exposures across the lifespan [1,2], including occupational noise.

Common cochlear pathologies involved in ARHL weaken active cochlear amplification with differential effects on hearing sensitivity:

- **Strial / metabolic**: lower endocochlear potential weakens OHC motility, producing a gently sloping pure-tone threshold pattern across low- and high-frequency thresholds.

- **Sensory:** damage to OHCs, especially near the cochlear base, results in increased high-frequency thresholds.

These pathologies often co-occur [1,2], however metabolic and sensory components of ARHL can be differentially estimated from audiograms [3].

Chronic noise exposure produces OHC and strial pathology in animals [4], while self-reported noise histories from older adults have linked noise exposure primarily to sensory hearing loss [1,3].

Our current goal was to characterize the effect of occupational noise on the progression of ARHL across the adult lifespan.

Methods: Retrospective Data Sets

Retrospective analyses were performed with a longitudinal data set that includes audiograms from US workers [5] collected for the NIOSH Occupational Hearing Loss Surveillance Project (1981-2010).

- The data selected for analysis included 1.73 million participants (77% men, 23% women; 1-6 audiograms collected at five-year intervals; 302 industry groups; six US geographical regions).

- Audiograms were excluded from the analysis if there were two or more missing pure-tone thresholds or their configurations were inconsistent with ARHL [3].

- All age groups (ages 18-75 years) were included to examine the effects of occupational noise on the progression of hearing loss across the adult lifespan.
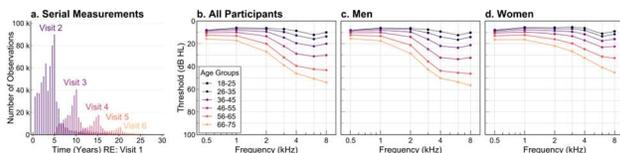

Above. Pure-tone thresholds were serially measured over a period up to 30 years (a). Average audiograms with standard error of the mean (SEM) error bars are shown for all participants (b), men (c), and women (d).

Method: Hearing Loss Components and Noise Levels

Audiograms were used to calculate ear-averaged metabolic and sensory estimates [3] (in dB) in addition to worse ear 4-frequency pure tone averages (0.5-4 kHz; PTA).

- Occupational noise levels were estimated for each participant by linking industry group codes to noise measurement data in the Job Exposure Matrix 2 [6-7].

- Regression analyses were performed on the metabolic estimates, sensory estimates, and PTA to evaluate associations with age, sex, and occupational noise exposure, while controlling for US region.

- Growth curve analyses (GCA) were performed to characterize hearing loss trajectories across serial visits.
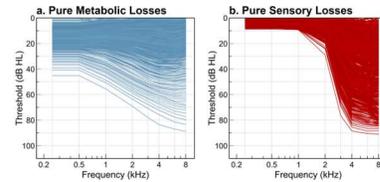
Left. Average audiograms show distinct patterns for "pure" metabolic (a) and "pure" sensory (b) hearing loss, defined by nonzero metabolic or sensory estimates while the other component was equal to zero. Audiograms were ordered by increasing loss prior to averaging.

Results: Participant Age and Sex Differences

Age Groups: All three hearing loss measures (metabolic, sensory, PTA) significantly increased from younger to older age groups [Z ≥ 30.78, p < 0.001].

Time: All three hearing loss measures significantly increased over time, after the baseline visit [Z ≥ 2.77, p < 0.01].

Time × Age Interaction: Metabolic and PTA progressed significantly faster for the older groups [Z ≥ 8.33, p < 0.001], while sensory progressed significantly faster for the younger groups [Z = -11.82, p < 0.001].

Sex Differences: Men had significantly higher sensory estimates, metabolic estimates, and PTA than women [Z > 10.42, p <= 0.001].

Sex × Time Interaction: Sensory estimates and PTA increased faster for men than women [Z ≥ 51.90, p < 0.001]; no significant interaction for metabolic losses.
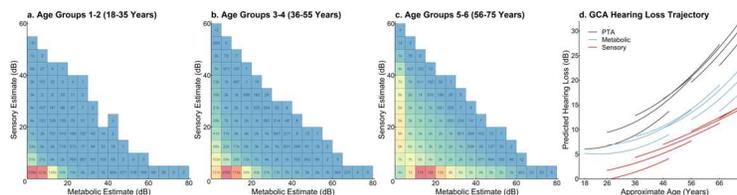

Above. Metabolic and sensory estimates were increasingly variable for older compared to younger age groups (a–c). Participants were counted in 5 dB bins for the metabolic and sensory estimates; warmer colors represent higher counts. Results from the GCA trajectory models (d) showed that each hearing loss measure significantly increased over time [steeper slopes: Z ≥ 180.44, p < 0.001] and across older age groups [steeper slopes: Z ≥ 42.11, p < 0.001]. The GCA fitted trajectories are shown in relation to *approximate age*, which was derived from baseline age groups and years since the initial visit.

Results: Occupational Noise

Noise Effect: All three hearing loss measures (metabolic, sensory, PTA) significantly increased with higher noise levels [Z ≥ 6.42, p < 0.001].

Noise × Age Interaction: All three hearing loss measures increased with noise faster for younger age groups [Z ≤ -2.78, p < 0.006].
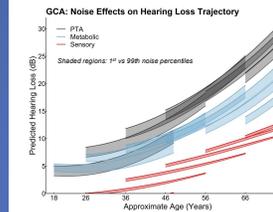
Left. Higher occupational noise levels were associated with a significantly faster progression of metabolic and PTA [GCA, steeper slopes: Z ≥ 6.42, p < 0.001], but not sensory. Each noise effect is depicted with shaded space between GCA-fitted hearing losses, predicted for the 1st and 99th industry group noise percentiles. The GCA-fitted trajectories are shown as a function of approximate age.

US Regional Differences

Above. Each US region was compared to the national average. The Southwest, South, and Mid-Atlantic had significantly higher than average metabolic and the Southwest, Western, and Midwest had significantly higher than average sensory (* p < 0.05, ** 0.01, *** 0.001).

Conclusions

Hearing loss accumulated across the adult lifespan, with metabolic losses accelerating and sensory losses slowing at older ages.

Higher occupational noise levels were associated with greater metabolic and sensory losses, despite slower progression at the oldest ages.

Consistent with animal models showing chronic noise effects on strial and OHC physiology [3], our results suggest that age-related metabolic losses, like sensory, can be worsened by occupational noise.

These findings underscore the importance of hearing protection and occupational noise control policies.

References: [1] Dubno, Eckert, Lee, Matthews, Schmiedt (2013). Classifying human audiometric phenotypes of age-related hearing loss from animal models. JARO, 14(5), 687–701. [2] Schmiedt (2010). The physiology of cochlear presbycusis. In Gordon-Salant, Frisina, Popper, Fay (Eds), The Aging Auditory System, 9–38. [3] Vaden, Eckert, Matthews, Schmiedt, Dubno (2022). Metabolic and sensory components of age-related hearing loss. JARO, 23(2), 253-272. [4] Fuentes-Santamaría et al. (2022) Age-related inflammation and oxidative stress in the cochlea are exacerbated by long-term, short-duration noise stimulation. Front Aging Neurosci 14:853320. [5] Masterson, Deddens, Themann, Bertke, Calvert (2015). Trends in worker hearing loss by industry sector, 1981-2010. Am J Ind Med, 58(4): 392-401. [6] Cheng, Roberts, Mukherjee, Neitzel. (2018) Meta-analysis of job-exposure matrix data from multiple sources. J Expo Sci Epidemiol, 28(3):259-274. [7] 2025 update: https://noisejem.sph.umich.edu

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