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Normal Audiogram Does Not Confirm Intact Cochlear Amplification: Compressive Nonlinearity Reveals Incipient Cochlear Changes

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Data Availability Statement

The datasets analyzed for the current study are available upon request.

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Abstract

The cochlear amplifier, mediated by outer hair cell–driven nonlinear mechanics, enables the remarkable sensitivity and dynamic range of human hearing. Cochlear compression is a defining feature of this active process and is known to deteriorate with hearing loss. Among individuals with normal audiograms, cochlear compression is generally presumed to be invariant, an assumption that has not been systematically examined. If incipient cochlear changes precede measurable threshold shifts, variability in compressive nonlinearity may represent an early peripheral correlate of emerging dysfunction and a potential mechanistic contributor to auditory processing differences. Whether cochlear compression varies meaningfully among individuals with normal audiograms, and what such variability reveals about the earliest stages of cochlear dysfunction, remains unknown. Here, we quantified compressive nonlinearity using distortion-product otoacoustic emission (DPOAE) input/output functions with stringent signal-to-noise criteria, applying covariate-controlled mixed-effects models within a dimensional framework that treated compression as a continuous outcome to isolate structured individual differences (n=144 ears, male=46, female=98). Compression metrics demonstrated substantial interindividual variability despite normal hearing thresholds. Subtle differences in standard-frequency thresholds were associated with variations in cochlear gain. Extended high-frequency sensitivity, an index of basal cochlear integrity, showed additional associations with DPOAE input/output function, implicating basal vulnerability in shaping cochlear nonlinearity. Age-related effects were detectable even within a restricted young-adult cohort, indicating that cochlear operating characteristics may begin to diverge early in adulthood. These findings challenge the assumption of invariant compression in normal hearing and suggest that alterations in cochlear nonlinear processing may reflect incipient cochlear dysfunction that precedes overt hearing loss.

Keywords: Cochlear Compression; Early Aging; Extended High Frequency Hearing; Individual Differences; Otoacoustic Emissions; Subclinical Dysfunction

Significance Statement

Hearing has traditionally been defined by audiometric thresholds, with normal results interpreted as evidence of an intact auditory periphery. This study challenges that assumption by demonstrating meaningful physiological variation within the clinically normal-hearing range. Specifically, cochlear nonlinear amplification, the core mechanism regulating sensitivity and dynamic range, differs across individuals and is associated with subtle biological factors, including age-related variation detectable in early adulthood. These findings indicate that cochlear function reflects a continuum of underlying biological states, some of which may represent incipient dysfunction preceding measurable hearing loss.

Introduction

Normal hearing depends on the active mechanics of the cochlea, driven by outer hair cells (OHCs) that amplify basilar membrane motion and shape the response to sound (Yates, 1995). A central feature of cochlear amplification is compressive nonlinearity, which enables sound encoding across a wide intensity range by transitioning from linear growth at low levels to compressive growth as amplification saturates (Robles et al., 1986; Ruggero et al., 1997). When OHC function is compromised, this nonlinearity is reduced, leading to more linear responses and perceptual consequences such as loudness recruitment, degraded frequency resolution, and impaired temporal processing (Ruggero et al., 1997; Shera et al., 2002; Oxenham & Bacon, 2003).

In humans, cochlear compression cannot be measured directly and is instead inferred using indirect approaches, such as distortion-product otoacoustic emission (DPOAE) input/output (i/o) functions (Kummer et al., 1998). Despite extensive work in hearing-impaired ears, far less is known about how cochlear compression varies among individuals with normal audiograms. This gap is consequential because a normal audiogram reflects threshold sensitivity but does not assess nonlinear cochlear gain, leaving open the possibility that individuals with normal thresholds differ in cochlear amplifier function. Supporting this notion, variability in DPOAE i/o functions has been observed among normal-hearing individuals, suggesting that cochlear mechanics are not uniform even within clinically defined limits (Poling et al., 2014; Glavin et al., 2021). Such variability may reflect early, subclinical OHC dysfunction that precedes measurable threshold shifts. In parallel, substantial variability in suprathreshold auditory processing, such as differences in level coding, spectral resolution, and speech-in-noise perception (Bharadwaj et al., 2015; Pienkowski et al., 2017), has been observed in normal-hearing listeners and is often attributed to neural or central mechanisms. However, variability in cochlear compression itself could contribute to these perceptual differences by altering the transformation of sound level and spectral contrast at the cochlear stage, thereby influencing the neural representation available to higher auditory centers. Under this framework, interindividual variability in compression may provide a mechanistic substrate for auditory processing differences among normal-hearing listeners.

Several biological and environmental factors may contribute to variability in cochlear compression, though their relative influence remains unclear. Age-related changes, even in early adulthood, may reflect cumulative cellular stress and metabolic alterations affecting OHC function (López-Otín et al., 2013; Yamasoba et al., 2013). Reported reductions in DPOAE growth with age

are often confounded by subtle threshold differences across groups, complicating interpretation (Ortmann & Abdala, 2016; Abdala et al., 2021; Glavin et al., 2021). Lifetime noise exposure is another potential contributor. Animal studies show that noise damages OHCs and reduces cochlear compression (Ruggero et al., 1996), but human findings are mixed: some report reduced DPOAE levels with greater exposure (Bhatt et al., 2023), while others find weak associations (Stamper and Johnson, 2015; Le Prell et al., 2018; Poortere et al., 2023). Notably, the effects of noise on compression metrics themselves have not been systematically assessed in humans. A third factor is extended high-frequency (EHF) hearing, reflecting basal cochlear integrity. The basal region is particularly vulnerable to early age- or noise-related damage, and EHF deficits are common even in audiometrically normal individuals (Lee et al., 2012; Le Prell et al., 2013; Aryal et al., 2025; Mishra et al., 2025). EHF loss has been linked to altered OAEs and broader auditory filters at lower and mid frequencies, suggesting that basal dysfunction may influence cochlear processing beyond its tonotopic region (Badri et al., 2011; Mishra et al., 2022; Mishra et al., 2024; Aryal & Mishra, 2025b; Aryal et al., 2026).

Yet, these factors have rarely been examined together within a unified framework, and their contributions to nonlinear cochlear function remain poorly understood. The present study addresses this gap by systematically examining the contributions of age, lifetime noise exposure, and both standard- and EHF hearing sensitivity to variability in cochlear compressive nonlinearity. Using DPOAE i/o functions and a rigorous, covariate-controlled analytical framework, we test the hypothesis that subtle differences in cochlear integrity are associated with measurable differences in nonlinear cochlear gain, even in young adults with clinically normal audiograms.

We apply a dimensional framework that considers cochlear function as a continuous biological variable rather than a categorical distinction between normal and impaired hearing. Threshold-based classifications, while clinically useful, may obscure structured individual variation in nonlinear cochlear processing. Because OHC-mediated compression underlies sensitivity, dynamic range, and frequency selectivity, differences in compressive nonlinearity can reflect fundamental variation in cochlear operating state not captured by standard audiometry. A dimensional perspective enables systematic characterization of interindividual variability (McDermott et al., 2013; Whiteford & Oxenham, 2015; Borjigin & Bharadwaj, 2025). By focusing on individuals classified as normal by conventional criteria, this study seeks to uncover biologically meaningful variability in cochlear function, revealing whether early, subclinical differences are already measurable.

Methods

Participants

All experimental procedures were conducted in a double-walled, sound-attenuated booth and were approved by the institutional review board (#STUDY00006139). Written informed consent was obtained from all participants, who were compensated for their time. Seventy-two participants (n=144 ears, 18–34 years; M=21.53, SD=3.63; males=46 ears) were included in the study. Outer and middle ear status were evaluated using otoscopy and tympanometry with a Titan handheld tympanometer (Interacoustics). Tympanometry using a 226-Hz probe tone confirmed normal middle ear function, evidenced by type “A” tympanograms in all participants. Participants reported no active otologic or neurologic disorders that could have affected the experimental measures. Air-conduction thresholds were measured using a Maico MA42 audiometer with DD450 RadioEar circumaural headphones. Inclusion required normal hearing sensitivity, defined as thresholds ≤20 dB HL at octave frequencies from 250 to 8000 Hz. Thresholds at 10000, 12500, and 16000 Hz were also obtained to assess EHF hearing.

Middle-ear transmission was assessed using wideband energy absorbance (MEPA3, ER-10C, Mimosa Acoustics) with Thevenin source calibration performed before each session using a four-cavity calibration standard. Wideband absorbance was measured using a one-second chirp (60 dB SPL) across 210–6000 Hz, and band-averaged values were computed within frequency regions corresponding to DPOAE analysis bands.

Noise Exposure Structured Interview

Lifetime noise exposure was assessed across occupational, recreational, and environmental sources using the Noise Exposure Structured Interview (NESI; Guest et al., 2018). The NESI protocol segments participants’ lives into discrete time periods with stable noise exposure patterns to facilitate accurate recall. It was selected over open-ended questionnaires (e.g., Noise Exposure Questionnaire, Johnson et al., 2017) because its structured format systematically captures exposure duration, intensity, and hearing protection use, reducing underestimation of cumulative noise exposure. For each period, participants reported detailed information on noisy activities (e.g., occupational tasks, music performance, firearm use), including duration, frequency, and estimated sound level. Sound levels were estimated using standardized metrics based on vocal effort required for communication or typical volume settings. When vocal effort

could not be directly observed, such as for loud musical instruments, participants estimated the vocal effort needed by a conversational partner to be heard. Use of hearing protection, including type and consistency, was also recorded. For firearm-related exposure, participants specified firearm type, number of rounds fired, and hearing protection use. All exposure data were converted into standardized noise exposure units linearly related to total acoustic energy, with one unit defined as one working year (2080 hours) of exposure at 90 dB(A). NESI was administered by a trained audiologist/researcher at the end of the testing session, after completion of physiological and behavioral measurements.

Distortion Product Otoacoustic Emissions

DPOAEs were recorded using the ER-10C probe system from Etymotic Research, connected to an ARIEL DSP-161 digital signal processor (HearID's CUBeDIS™ software, Mimosa Acoustics). The ER-10C probe includes dual loudspeakers and a microphone coupled to the ear canal via a disposable foam tip. Each primary tone was generated through separate output channels and delivered independently to the corresponding loudspeaker within the probe assembly positioned in the ear canal. Before DPOAE measurements, in-the-ear sound pressure level (SPL) calibration was performed to confirm accurate stimulus presentation, proper probe placement, and system integrity. During calibration, each transducer emitted chirp stimuli separated by a one-second interval to assess the noise floor, and pressure responses recorded in the ear canal spanned the frequency range of interest (800 to 6600 Hz). Calibration waveforms from both transducer channels were visually confirmed for alignment; substantial overlap between channels indicated a secure probe seal and proper system function, whereas deviations indicated poor coupling, obstruction, or equipment malfunction. The "Repeat" metric, representing the proportion of valid data frames collected, was used to monitor response stability and ambient noise levels. Calibration was accepted only when the Repeat score exceeded 95%, ensuring consistent and reliable measurements.

SPL calibration was used rather than forward pressure level (FPL) calibration because the test frequencies were within the standard frequency range (1000–6000 Hz), where standing wave effects and SPL-FPL differences are minimal below ~4000 Hz (Scheperle et al., 2008; Keefe & Schairer, 2011; Souza et al., 2014). To verify that standing waves did not influence measurements at 6000 Hz, trial-to-trial variability was evaluated at low versus high frequencies (1000 vs. 6000 Hz). Equality of trial variances was assessed using the Pitman–Morgan test for DPOAE levels at L_2 = 65 dB SPL. No significant difference in trial variances was observed at 1000 Hz (r=0.16, t

(135) =1.84, p=0.07) or 6000 Hz (r= −0.05, t (142) = −0.57, p=0.57), indicating comparable variability between trials and suggesting that measurements at 6000 Hz were not substantially influenced by standing-wave artifacts. Consistent with this, trial-to-trial variability did not differ significantly between frequencies (t (278.8) = −1.08, p=0.28), and coefficients of repeatability were similar (1000 Hz=4.71 dB; 6000 Hz = 5.09 dB).

DPOAE i/o functions were recorded at participant-specific frequencies selected from fine-structure peak regions, with f_2 frequencies near 1000, 2000, 4000, and 6000 Hz. The peak-based method was selected based on findings from our previous study (Aryal & Mishra, 2025a), which demonstrated higher SNR and test–retest reliability compared to the conventional approach. The mean f_2 across individuals was 1015.85 Hz (SD = 41.49), 1991.86 Hz (SD = 66.98), 4086.91 Hz (SD = 185.60), and 5878.26 Hz (SD = 235.67), respectively. The f_2/f_1 ratio was maintained at 1.22. Stimulus levels for the primary tones (L_1 and L_2) were varied systematically using the scissor paradigm proposed by Kummer et al. (1998): $L_1 = 0.4 \times L_2 + 39$ dB. The L_2 intensity ranged from 70 dB SPL down to 20 dB SPL in 5 dB decrements. DPOAE recordings were obtained separately from both ears in two independent trials per ear. The probe was removed and reinserted between trials, and the calibration was repeated after each reinsertion to account for changes in ear canal acoustics (Aryal & Mishra, 2025a).

Modeling DPOAE i/o Functions

DPOAE measurements were obtained at four discrete frequencies corresponding to the fine-structure peak regions of f_2 (1000, 2000, 4000, and 6000 Hz), yielding a total of 1152 i/o functions (72 participants × 2 ears × 2 trials × 4 frequencies). Data points that did not meet the minimum SNR criterion (≥ 6 dB) were excluded from subsequent analyses. Across all L2 levels and trials, only a small proportion of data points (<2%) required interpolation. Specifically, 59 of 3168 datapoints (1.86%) at 1000 Hz, 31 of 3168 (0.98%) at 2000 Hz, 12 of 3168 (0.38%) at 4000 Hz, and 28 of 3168 (0.88%) at 6000 Hz were replaced using linear interpolation based on the mean of the adjacent valid datapoints. Interpolation was not performed for datapoints at the boundary stimulus levels (20- or 70-dB SPL) to avoid introducing artificial trends at the extremes of the i/o functions. Across each frequency, 3168 datapoints (144 ears × 2 trials × 11 stimulus levels) were available for analysis. Although the peak-based method yielded higher SNR than the conventional approach, a few measurements still had SNRs < 6 dB. After applying the two-trial averaging rule, the final proportions of missing data across all the L_2 levels (20-70 dB) were 11.4% (180/1584),

5.43% (86/1584), 1.89% (30/1584), and 6.76% (107/1584) at 1000, 2000, 4000, and 6000 Hz, respectively. Missing data were most common at the lowest stimulus level (L_2 = 20 dB SPL), where usable measurements were unavailable for 42.4%, 26.4%, 11.8%, and 36.8% of i/o function points at 1000, 2000, 4000, and 6000 Hz, respectively. The proportion of missing data decreased progressively with increasing stimulus level and was nearly zero at higher stimulus levels. A 6 dB SNR criterion was used to ensure robust measurements, representing a more conservative approach compared with the 3 dB SNR criterion used in some previous studies (e.g., Glavin et al., 2021).

For robust averaging, data from the two trials were combined using some prespecified rules. When both trials had SNR ≥ 6 dB and the absolute difference in DPOAE levels between the two trials was ≤ 5 dB, the responses were averaged. If the level difference exceeded 5 dB, the trial with the higher DPOAE level was retained for analysis. In cases where only one trial had SNR ≥ 6 dB, and the other had SNR < 6 dB, the trial with SNR ≥ 6 dB was selected. This approach was used because the difference of 5 dB within one test session is likely due to movement and/or probe-drift related measurement error, which could otherwise introduce artificial dips in the fitted input/output functions. To ensure robustness, only functions with at least six valid L_2–DPOAE pairs were included in the line-fitting analysis.

To characterize the nonlinear growth of DPOAE responses, a segmented linear regression model with one breakpoint was employed, implemented using the segmented package in R (Muggeo, 2003; Muggeo, 2008). The *segmented* package provides an extension of the classical linear model framework, allowing for estimation of regression relationships in which the predictor variable contains one or more unknown breakpoints treated as model parameters. These breakpoints are identified iteratively using a linearization procedure within the Gaussian linear regression framework, and the model then estimates separate linear slopes for the regions before and after each breakpoint, while enforcing continuity of the fitted function at the breakpoint. In the present study, only a single breakpoint was specified for each i/o function, corresponding to the hypothesized transition between the linear low-level region and the compressive mid-to-high-level region. The segmented regression method allows estimation of the breakpoint directly as a continuous model parameter, providing finer breakpoint resolution while yielding comparable slope estimates across segments.

For each i/o function, an initial base linear regression was fit to the observed DPOAE data, assuming normally distributed residuals, after which the segmented procedure was applied. The

breakpoint was initialized at the median of the observed L_2 intensity range to provide a stable starting estimate, with a maximum of 50 fitting iterations specified. The algorithm iteratively updated the breakpoint location until convergence was achieved. The fitted model delineated two distinct regions: the first segment (Slope 1), reflecting linear growth at low input levels, and the second segment (Slope 2), reflecting reduced growth consistent with cochlear compressive nonlinearity. Along with the breakpoint estimate (interpreted as the compression threshold), Slope 1 and Slope 2 were extracted, providing quantitative measures of growth before and after the breakpoint. Segmented regression was applied both to individual ear-level i/o functions and to the mean growth functions across frequencies to illustrate the overall population-level growth pattern.

Model adequacy was assessed using the Bayesian information criterion (BIC). Only i/o functions containing at least six valid data points were included in the curve-fitting analyses. Segmented models exhibited substantially lower BIC values (mean = 3.90, range = −38.30 to 35.47) than linear models (mean = 21.19, range = −13.66 to 44.50), indicating superior fit after accounting for model complexity. Frequency-wise, paired comparisons confirmed that BIC values were significantly lower for segmented models than linear fits at all tested frequencies (1000, 2000, 4000, and 6000 Hz; all $p < 0.001$), which confirmed their appropriateness in describing the DPOAE i/o function. Finally, all i/o functions were visually inspected to confirm that the estimated breakpoint did not occur at the intensity boundaries, thereby preserving the interpretability of the segmented growth functions.

Statistical Analyses

Statistical analysis had two primary goals: (1) to evaluate the extent to which individual factors (e.g., age, sex, EHF sensitivity, noise exposure, and audiometric thresholds) predict variability in DPOAE levels across low-, mid-, and high-level stimuli for the tested f_2 frequencies; and (2) to determine whether these factors predict individual variability in cochlear compression estimates (Slope 1, Slope 2, and breakpoint) at the same frequency range. Repeated-measures analyses of variance (RM-ANOVAs) were conducted to evaluate the effect of frequency on cochlear compression metrics, including breakpoint, Slope 1, and Slope 2 across f_2 frequencies of 1000, 2000, 4000, and 6000 Hz. Frequency was treated as a within-ear repeated factor, and analyses were conducted at the ear level. When significant effects were observed, post hoc pairwise comparisons were conducted using estimated marginal means with Benjamini–Hochberg (BH) correction to control the false discovery rate across multiple frequency comparisons. Pearson correlation (two-tailed) analyses were also performed to examine the relationships among

compression metrics, including Slope 1, Slope 2, and breakpoint across frequencies with statistical significance set at $p < 0\ 05$.

To identify factors associated with interindividual differences in DPOAE levels and cochlear compression metrics, LMEM were fitted separately for each frequency to appropriately account for the hierarchical data structure and repeated measures from both ears within individuals. LMEMs were selected for their flexibility in handling correlated observations and unbalanced and missing data (Faraway, 2016). Subject ID was included as a random intercept to account for within-subject dependence across ears.

Each model for each frequency included fixed effects for NESI Score, EHF hearing sensitivity (i.e., 16000 Hz thresholds), age (log-scaled), ear (right vs. left), behavioral pure-tone threshold at the corresponding f_2 frequency (e.g., 4000 or 6000 Hz), and sex (male vs. female). To assess whether middle-ear transmission influenced DPOAE outcomes, frequency-specific wideband absorbance was correlated with DPOAE levels and compression metrics using two-tailed Pearson correlations. Absorbance was not significantly associated with any DPOAE metric across 1000–6000 Hz ($r = -0.18$ to 0.19; all adjusted $p \geq 0.25$). Therefore, absorbance was not included in the final LMEM models to avoid unnecessary model complexity. Age was log-transformed because age-related cochlear changes may not follow a strictly linear pattern, even within adulthood (Bom et al., 2001; Snir et al., 2019). Instead of using the pure tone average EHFs, the use of threshold at 16,000 Hz was decided *a priori* as the predictor variable, as it exhibited a wider range of hearing sensitivity across participants, allowing better evaluation of its effect. This choice was also supported by prior findings demonstrating the vulnerability of the basal region of the cochlea to damage from noise exposure (Mehrparvar et al., 2011; Le Prell et al., 2013; Aryal et al., 2025) and aging (Lee et al., 2012; Braza et al., 2022; Mishra et al., 2025). The dependent variables were analyzed separately and included DPOAE level at low, mid, and high stimulus levels, Slope 1, Slope 2, and the breakpoint at each frequency. LMEM adjusted plots were generated representing model-predicted relationships between the predictor and outcome after controlling for other covariates included in the LMEM.

Model assumptions were evaluated by visual inspection of residual-versus-fitted plots, which indicated homoscedasticity, and by assessing multicollinearity using variance inflation factors, with all values below the conservative threshold of 5 (Kutner et al., 2005). BH correction was applied to all models for controlling the false discovery rate across multiple frequency

comparisons (Benjamini & Hochberg, 1995). Effect sizes for fixed effects were quantified using partial omega squared (ω^2) and interpreted using established benchmarks (small: $\omega^2 \approx 0.01$; medium: $\omega^2 \approx 0.06$; large: $\omega^2 \geq 0.14$) (Olejnik & Algina, 2003). All statistical analyses were performed in R (version 4.4.2).

Results

EHF Hearing Loss Hiding Beneath Normal Audiogram

Figure 1 shows individual and mean hearing thresholds across frequencies from 250 to 16,000 Hz for all participants. EHF thresholds range from −20 to 55 dB HL across frequencies (10,000, 12500, and 16000 Hz). At EHFs, thresholds ranged from −15 to 25 dB HL at 10000 Hz, −15 to 40 dB HL at 12500 Hz, and −20 to 55 dB HL at 16000 Hz (n=144 ears). Greater elevation of thresholds was evident at 16,000 Hz compared to 12500 and 10000 Hz. Linear regression revealed that age was the primary factor associated with elevated EHF thresholds (β = 1.09 dB/year, $p < 0.001$), indicating that for each additional year of age, EHF thresholds increased by an average of 1.09 dB. In contrast, NESI score and sex were not significant predictors ($p > 0.05$). Only for audiogram visualization purposes, participants with hearing thresholds greater than 20 dB HL at any EHFs (10000, 12500 and 16000 Hz) in either ear were categorized into the EHF loss group (n=30 ears).

DPOAE Levels and Mean i/o Function Fits

DPOAE levels were selected as the primary outcome measure rather than SNR because DPOAE levels have been shown to demonstrate greater test–retest reliability and lower variability across repeated measurements (Portugal et al., 2024). Figure 2 shows DPOAE levels as a function of stimulus level for f_2 frequencies of 1000, 2000, 4000, and 6000 Hz. Across all frequencies, DPOAE levels increased with increasing stimulus level until a breakpoint, beyond which the growth rate decreased, consistent with expected cochlear compressive nonlinearity. The mean SNRs were 15.2 dB (SD=5.3; range: 6.0–33.4) at 1000 Hz, 20.2 dB (SD=8.1; range: 6.2–42.7) at 2000 Hz, 23.8 dB (SD=8.8; range: 6.8–42.7) at 4000 Hz, and 20.7 dB (SD=8.2; range: 6.0–41.0) at 6000 Hz across all stimulus L_2 levels. Segmented regression was applied to the mean growth functions and identified significant changes in slope at all frequencies (Davies test: $p \leq 0.001$). The models provided excellent fits to the mean data (all $R^2 \approx 0.99$). Overall, the segmented fits illustrate the expected transition from near-linear growth at lower stimulus levels to reduced, compressive growth at higher levels across frequencies. Frequency-wise comparisons further

showed a steeper compressive slope (Slope 2) at higher frequencies compared to lower frequencies (Fig. 2).

Individual Fitted DPOAE i/o Function Across Frequencies

Among the 144 ears analyzed, the segmented regression method could not be fitted for 4 ears at 1000 Hz due to an insufficient number of valid data points across stimulus levels, which precluded reliable estimation of breakpoints and segment-specific slopes. For 2000, 4000, and 6000 Hz, all ears had sufficient data density for model estimation, and no ears were excluded due to model non-convergence or insufficient points. In addition to these non-fitted cases at 1000 Hz, further ears were excluded due to poor i/o function quality: 13 ears at 1000 Hz, 4 ears at 2000 Hz, 5 ears at 4000 Hz, and 10 ears at 6000 Hz (See supplemental Fig.1 as an example of poor i/o function). After excluding both non-fitted and poor-quality i/o functions, the final number of ears included in the compression (Slope 2) analysis was 127 at 1000 Hz, 140 at 2000 Hz, 139 at 4000 Hz, and 134 at 6000 Hz. These exclusions did not show any clear pattern related to demographic, physiological, or environmental factors, including age, sex, EHF thresholds, noise-exposure history, and SNR. Representative segmented fits with identified breakpoints are illustrated in Figure 3, and examples from EHF loss ears are provided in Supplementary Figure 2.

Effect of Frequency on Cochlear Compression Estimates

Figure 4 shows the segment-specific slopes (Slope 1 and Slope 2) and breakpoint estimates across frequencies. Slope 1 showed a nonmonotonic frequency pattern, increasing from 0.62 at 1000 Hz to 0.87 at 6000 Hz, with variability across the intermediate frequencies. In contrast, Slope 2 showed a shallower slope and increased monotonically with frequency, shifting from −0.05 at 1000 Hz to 0.27 at 6000 Hz, indicating reduced compression at higher frequencies. RM-ANOVA revealed a significant main effect of slope type [$F(1, 113) = 2241.78$, $p<0.001$, $\omega^2=0.87$], indicating that Slope 1 was consistently larger than Slope 2. There was also a significant main effect of frequency [$F(2.63, 297.52) = 47.41$, $p<0.001$, $\omega^2=0.22$], as well as a significant slope × frequency interaction [$F(2.78, 314.01) = 15.24$, $p<0.001$, $\omega^2=0.07$]. Post hoc pairwise comparisons revealed that Slope 1 was significantly higher than Slope 2 at all frequencies (1000 Hz, 2000 Hz, 4000 Hz, and 6000 Hz; all adj $p<0.001$). Frequency comparisons within each slope revealed different patterns. For Slope 1, the slope at 2000 Hz was significantly higher than at 1000 Hz (adj $p=0.002$), and the slope at 6000 Hz was significantly higher than at 1000 Hz, 2000 Hz, and 4000 Hz (all adj $p<0.01$). No significant difference was observed between 1000 Hz and 4000 Hz ($p = 0.33$). For

Slope 2, slopes increased progressively with frequency, and all pairwise comparisons between frequencies were significant after BH correction (all adj p<0.001), indicating progressively steeper slopes from 1000 Hz to 6000 Hz.

Breakpoint estimates occurred at lower stimulus levels with increasing frequency, decreasing from approximately 51 dB SPL at 1000 Hz to 43 dB SPL at 4000–6000 Hz. RM-ANOVA revealed a significant main effect of frequency [F (2.90, 327.94) = 34.97, p<0.001, ω^2=0.18]. Post hoc pairwise comparisons showed that the breakpoint at 1000 Hz was significantly higher than at 2000 Hz, 4000 Hz, and 6000 Hz (all adj p<0.001). Similarly, the breakpoint at 2000 Hz was significantly higher than at 4000 Hz and 6000 Hz (both adj p<0.001). However, no significant difference was observed between 4000 Hz and 6000 Hz (adj p=0.46). Overall, the breakpoint decreased with increasing frequency from 1000 Hz to 4000 Hz, with similar breakpoint values observed at 4000 Hz and 6000 Hz. Together, these results demonstrate systematic frequency-dependent differences in DPOAE growth behavior, characterized by steeper low-level growth, reduced compression, and earlier transition points at higher frequencies.

Correlation among slopes and breakpoint across frequencies

Relationships among Slope 1, Slope 2, and breakpoint were evaluated across frequencies. At 1000 Hz, both Slope 1 (r= −0.59, adj p<0.001, n=127) and Slope 2 (r= −0.58, adj p<0.001, n=127) were strongly and negatively correlated with breakpoint, and Slope 1 and Slope 2 were positively correlated (r= 0.31, adj p<0.001). At 2000 Hz, Slope 1 (r= −0.466, adj p<0.001, n=140) and Slope 2 (r= −0.55, adj p<0.001, n=140) showed significant negative correlations with breakpoint, while Slope 1 and Slope 2 were strongly positively correlated (r=0.56, adj p<0.001). At 4000 Hz, Slope 1 showed a modest negative correlation with breakpoint (r = −0.3, adj p <0.001, n=139), whereas the association between Slope 2 and breakpoint was not significant (r= −0.12, adj p=0.15). Slope 1 and Slope 2 remained strongly positively correlated (r=0.54, adj p <0.001). At 6000 Hz, both Slope 1 (r=−0.43, adj p <0.001, n=134) and Slope 2 (r= −0.23, adj p = 0.006, n=134) were significantly negatively correlated with breakpoint, and Slope 1 and Slope 2 were moderately positively correlated (r= 0.30, adj p < .001). Overall, both Slope 1 and Slope 2 showed negative relationships with breakpoint across frequencies, indicating that ears with steeper slopes tended to exhibit lower breakpoints (earlier onset of compression), and Slope 1 and Slope 2 were positively correlated across frequencies, indicating that ears with steeper Slope 1 also tended to show steeper Slope 2. Cross-frequency correlations of Slope 1, Slope 2, and breakpoint were

generally weak and nonsignificant (all adjusted p > 0.05), except for a modest correlation in Slope 2 between 2000 and 4000 Hz (r = 0.25, adjusted p = 0.03).

Effect of EHF Hearing Sensitivity, Age and Noise Exposure on DPOAE Level

Table 1 shows the LMEM outcomes for DPOAE levels at 4000 and 6000 Hz across high, mid, and low L_2 levels. For f_2=6000 Hz**,** at 70 dB**,** poorer threshold at 16000 Hz was significantly associated with reduced DPOAE levels (p adj=0.01; ω^2=0.10). The 6000 Hz threshold was also significantly associated with reduced DPOAE levels (p adj = 0.009; ω^2 = 0.06). At 50 dB, both the threshold at 16000 Hz (p adj=0.05; ω^2 =0.04) and the 6000 Hz threshold (p adj=0.0003; ω^2=0.13) were significantly associated with reduced DPOAE levels. Age was not significant at either high or mid stimulus levels (p>0.05). At 20 dB**,** none of the predictors, including 16000 Hz threshold, 6000 Hz threshold, or age**,** were significantly associated with DPOAE levels (*all* p>0.05). Overall, the effects of 16000 Hz threshold and standard-frequency thresholds were evident primarily at high and mid stimulus levels, but not at the low level for f_2=6000 Hz (Table 1).

Similarly, for f_2=4000 Hz**,** at 70 dB**,** poorer threshold at 16000 Hz was significantly associated with reduced DPOAE levels (adj p=0.04; ω^2=0.03). The 4000 Hz threshold was also significantly associated with reduced DPOAE levels (adj p=0.004; ω^2=0.07). At 50 dB**,** both the 16000 Hz threshold (adj p=0.05; ω^2=0.03) and the 4000 Hz threshold (adj p=0.0003; ω^2=0.09) were significantly associated with reduced DPOAE levels. Age was not significant at either high or mid stimulus levels (p > 0.05). At 20 dB, only the 4000 Hz threshold remained significantly associated with reduced DPOAE levels (adj p=0.0008; ω^2=0.10). The 16000 Hz threshold and age were not significant at this level (*all* p>0.05). The detailed LMEM results for 4000 Hz are shown in Table 1**.** Overall, the effects of the threshold at 16000 Hz were evident primarily at high and mid L_2 levels, but not at the low level**.** However, the effect of the threshold at f_2 was evident at all stimulus levels.

For f_2=2000 Hz, threshold at 16000 Hz was a significant predictor of DPOAE levels measured with L_2=70- and 50-dB SPL (both adj p=0.04) with a small effect size (ω^2=0.03, Supplemental Table 1). At f_2=1000 Hz, threshold was a significant predictor of DPOAE levels at stimulus L_2=70 dB SPL (adj p=0.008). No other predictors reached statistical significance at any stimulus level for either 1000 Hz or 2000 Hz (all adj p > 0.05). These results suggest an effect of 16000 Hz threshold at moderate-to-high stimulus levels across mid-to-high f_2 frequencies (2000–6000 Hz), with a more robust effect observed at 6000 Hz (Table 1, Supplemental Table 1).

Effect of EHF Hearing Sensitivity, Age and Noise Exposure on Cochlear Compression Estimates

Table 2 shows the LMEM outcomes on cochlear compressive metrics (Slope 1, Slope 2 and Breakpoint) at 4000 and 6000Hz. For f_2=4000 Hz, the LMEM showed that the 4000 Hz behavioral threshold was significantly associated with Slope 1 (adj p=0.05, ω^2=0.03). None of the other predictors, including age, sex, threshold at 16000 Hz, ear, or noise exposure, were significantly associated with Slope 1 (all adj p > 0.05). For Slope 2, none of the predictors were statistically significant (all adj p > 0.05). For breakpoint, age showed a significant negative association with moderate effect size at 4000 Hz (adj p=0.04, ω^2=0.08), indicating a lower breakpoint with increasing age (refer to Fig.5). None of the other predictors were significantly associated with breakpoint after correction for multiple comparisons.

For f_2=6000 Hz, the LMEM showed that poorer threshold at 16000 Hz was significantly associated with Slope 2 (adj p=0.03, ω^2=0.08), indicating shallower/altered slope with elevated 16000 Hz thresholds (Fig. 6). None of the other predictors were significantly associated with Slope 1, Slope 2, or breakpoint (all adj p>0.05). The detailed LMEM results for Slope 1, Slope 2, and breakpoint across frequencies are presented in Table 2. LMEMs with the same predictors were also run for 1000 Hz and 2000 Hz; however, none of the predictors were significant for any of the compression metrics at those frequencies (refer to Supplemental Figures 3, 4, and Supplemental Table 2).

Exploratory Analysis of DPOAE i/o Function in Middle-Aged Participants

To interpret the findings related to the breakpoint in the young adult sample (18–35 years), four additional participants (n=8 ears) aged between 38 and 52 years (mean age=42.8 years, SD = 5.27, male=8 ears) were further recruited and analyzed. The same segmented-fitting procedure used for the young-adult sample was applied. One ear could not be fitted for the slope due to the unavailability of adequate data with SNR ≥ 6 dB for line fitting and was therefore excluded from the comparison. Breakpoint analysis of the remaining seven ears showed a mean breakpoint of 44.26 dB SPL (SD=7.56) at 4000 Hz. This value is comparable to the mean breakpoint obtained from the young adult sample at 4000Hz (mean=42.02 dB SPL, SD=6.39, n=138 ears, mean age=22years). Despite the nearly doubled mean age in the present middle-aged sample, breakpoint estimates remained within a similar range. The mean data from these eight ears at 4000 Hz were also fitted, and the segmented fitting yielded a breakpoint of 47.6 dB SPL (Fig. 7), which is nearly equivalent to the mean fitted breakpoint obtained for 4000 Hz in the main analysis

of young adult participants (See Fig. 4, mean breakpoint=46.5). Given that age was significantly associated with the breakpoint in the young sample, these findings suggest that detectable alterations in compression may already be present in young adults. However, these interpretations should be considered preliminary for the middle-aged group and require further investigation with larger samples.

Discussion

Principal findings

The present study systematically examined determinants of interindividual variability in cochlear compressive nonlinearity in normal-hearing young adults. Using DPOAE i/o functions recorded from 144 ears and a peak-based approach with strict SNR criteria (≥6 dB), compression metrics were estimated with high stability and statistical rigor. By incorporating mixed-effects modeling and adjusting for age, sex, noise exposure, and both standard- and EHF thresholds, this study provides a comprehensive characterization of cochlear compression variability.

Three principal findings emerge. First, cochlear compression varied systematically with frequency. Second, subtle variations in hearing sensitivity, particularly at EHFs, were stronger predictors of compression metrics and DPOAE levels than self-reported lifetime noise exposure. Third, even within a restricted young-adult age range, age was associated with reduced breakpoint at 4000 Hz, suggesting early and frequency-specific changes in cochlear operating characteristics. Noise exposure was not associated with compression metrics or DPOAE levels, and no other relationships were observed at 1000 or 2000 Hz.

Frequency-Dependent Cochlear Compression

Compression metrics showed distinct frequency-dependent patterns (Fig. 4). Slope 1 showed modest, nonmonotonic variation across frequency, whereas Slope 2 increased monotonically with frequency, indicating steeper high-level DPOAE growth at higher frequencies. Breakpoint estimates decreased with increasing frequency, indicating an earlier transition to the compressive growth segment at higher frequencies. This pattern reflects known tonotopic differences in cochlear mechanics, where the basal cochlea is mechanically stiffer, more sharply tuned, and exhibits spatially restricted nonlinear motion compared to apical regions (Békésy, 1960; Cooper & Rhode, 1995; Robles & Ruggero, 2001).

Although absolute slope values fall within previously reported ranges from OAE studies (e.g., Kummer et al., 1998; Dorn et al., 2001; Ortmann & Abdala, 2016) and psychophysical studies (Oxenham & Plack, 1997; Nelson et al., 2001), the present study uniquely demonstrates a statistically significant monotonic frequency effect for compressive slope using a stringent data-quality threshold and mixed-effects modeling. Differences across studies likely reflect methodological variability, including slope estimation strategies, SNR criteria, and the number of data points used for fitting. The stringent data-quality criteria and the use of a peak-based approach (Aryal et al., 2025) used in the present study may have contributed to more consistent compression estimates.

Hearing Sensitivity and Nonlinear Cochlear Function

Thresholds at f_2 frequencies, even within clinically normal limits (≤20 dB HL), significantly predicted DPOAE levels across mid and high stimulus levels (1000–6000 Hz) (Table 1, Table S1). Similarly, thresholds at 16000 Hz were negatively associated with DPOAE levels (Table 1) and compressive slope at 6000 Hz (Slope 2, Table 2). These findings extend prior evidence that elevated behavioral thresholds are associated with reduced DPOAE levels (Moulin et al., 1994; Gorga et al., 1997) and demonstrate that subtle threshold variability within the *"normal"* range is functionally meaningful. The basal cochlea is particularly vulnerable to early metabolic stress and noise-related injury (Aryal et al., 2025; Mishra et al., 2025). Elevated EHF thresholds may therefore serve as a proxy for basal OHC integrity. The relationship of EHF thresholds with DPOAE levels and Slope 2 at 6000 Hz are consistent with previous findings suggesting that basal cochlear dysfunction may be accompanied by incipient changes at lower frequencies, potentially reflecting more widespread subclinical cochlear involvement along the cochlear length (Badri et al., 2011; Mishra et al., 2022; Mishra et al., 2024; Aryal & Mishra, 2025b; Aryal et al., 2026a; Aryal et al., 2026b).

The level-specific pattern is mechanistically informative. At moderate and high stimulus levels, cochlear excitation spreads beyond the characteristic frequency region, potentially engaging basal regions (Table 1). In contrast, at low stimulus levels, excitation is spatially restricted near the f_2 place, which may explain the absence of EHF and DPOAE associations at low L_2 and, consequently, for Slope 1. Importantly, no significant relationships were observed between EHF thresholds and low-level linear growth (Slope 1), reinforcing the level-dependent nature of these effects. Previous studies have shown that human DPOAEs can be influenced by an interference tone presented at a frequency higher than f_2, suggesting a basal contribution to emission

generation (Dhar & Shaffer, 2004; Martin et al., 2010; Martin et al., 2011). However, in those studies, the interference tone was approximately one-third of an octave above the f_2, indicating a relatively local interaction. To date, no human studies have demonstrated contributions from basal regions located as far as two octaves above the f_2 (e.g., effect of 16000 Hz tone on DPOAEs measured with f_2 =4000 Hz). Therefore, evidence for such distant basal sources contributing to DPOAEs remains limited.

Age-Related Variation Within Early Adulthood

Although age-related compression weakening is well documented in middle-aged and older adults (Ortmann & Abdala, 2016; Abdala et al., 2021), the present study demonstrates that measurable age associations can exist even within early adulthood. In this young cohort (mean ≈ 22 years), age was not associated with overall DPOAE levels. However, age was significantly associated with reduced breakpoint at 4000 Hz, suggesting earlier transition into the compressive operating range. The negative association between slope and breakpoint further indicates coordinated changes in nonlinear cochlear dynamics.

Notably, breakpoint values were similar between young and exploratory middle-aged samples despite substantial age differences, suggesting that detectable alterations in cochlear nonlinear dynamics may begin earlier than previously assumed. These findings suggest that age-related cochlear changes may initially manifest as subtle shifts in level-dependent gain mechanisms rather than overt sensitivity loss (Mills et al., 2006; Schmiedt, 2009; Yamasoba et al., 2013), consistent with evidence that age-related changes often emerge first in the EHF region (Lee et al., 2012; Mishra et al., 2025). In considering cochlear gain regulation, a small contribution from baseline or probe-elicited medial olivocochlear activity cannot be fully excluded (Guinan et al., 2003). However, because DPOAE i/o functions were measured without an efferent elicitor, they are expected to primarily reflect OHC-mediated cochlear mechanics under the baseline efferent state. Collectively, these findings suggest that early age-related cochlear changes may manifest as subtle alterations in cochlear compression at higher frequencies and sensitivity at EHFs before deficits appear in the standard audiogram.

No Evidence for Lifetime Noise Exposure Effects

Lifetime noise exposure was not associated with DPOAE levels or compression metrics. Although a trend was observed at 6000 Hz, it did not survive FDR correction or covariate adjustment. Noise exposure levels in this cohort were relatively low (NESI mean=2.53 units), which may have limited

detectability of effects or actual damage to OHCs. These findings align with prior reports of weak or inconsistent associations between self-reported noise exposure and OAE outcomes (Le Prell et al., 2018; Poortere et al., 2023). While structured instruments such as the NESI provide standardized estimates (Guest et al., 2018), self-report methods remain vulnerable to recall bias and measurement imprecision (Jiang et al., 2016). Studies using experimentally controlled or objectively measured noise exposure have demonstrated clearer physiological effects (Engdahl & Kemp, 1996), highlighting the limitations of retrospective exposure assessment.

Translational Implications

Cochlear compression is fundamental to level-dependent gain control, loudness growth, frequency selectivity, and the neural encoding of sounds in complex acoustic environments. Because OHC–mediated amplification shapes the input to inner hair cells and auditory nerve fibers, variability in compression can influence the fidelity of neural coding across the dynamic range, particularly for suprathreshold sounds that depend on nonlinear cochlear gain. This has important implications for interpreting neural markers of peripheral dysfunction, including approaches used to diagnose cochlear synaptopathy, which rely on assumptions about stable cochlear gain.

Emerging evidence indicates that peripheral dysfunction can exist despite normal audiograms and may contribute to listening difficulties in some individuals (Noda et al., 2026). The present findings align with this view by demonstrating that cochlear nonlinear amplification varies among adults with clinically normal hearing, indicating that standard threshold testing does not capture the full range of cochlear functional diversity. The association between EHF thresholds and compression further supports the hypothesis that basal cochlear integrity contributes to nonlinear cochlear processing at lower frequencies. Such subtle alterations may represent early physiological changes preceding overt hearing loss. Together, these results support a mechanistic framework linking cochlear nonlinear function to neural encoding and early auditory vulnerability, even in the absence of standard audiometric deficits.

Potential Limitations

Strengths of this study include a relatively large sample, stringent SNR criteria, peak-based DPOAE measurement, and mixed-effects modeling with covariate adjustment. Compression parameters were derived using a modified segmented regression approach designed to improve breakpoint stability. Only i/o functions with at least six valid data points were included in the fitting

procedure, although most functions typically contained 10 data points. Segmented models consistently demonstrated good fit, as reflected by lower BIC values. However, because the number of valid data points varied across individuals and frequencies, some uncertainty in breakpoint estimation may remain. In addition, the cross-sectional design potentially limits direct causal inference. Noise exposure was self-reported and relatively low in magnitude. Longitudinal studies are needed to determine whether early changes in DPOAE i/o function predict future threshold shifts or perceptual consequences.

Conclusions

The present study demonstrates that cochlear compressive nonlinearity varies systematically among adults with clinically normal audiograms and is significantly associated with subtle differences in hearing sensitivity and age. Age-related variation, even within a restricted young-adult range, contributes to this variability, suggesting that early biological changes associated with aging may already influence OHC-mediated amplification. The results further suggest that cochlear dysfunction can alter nonlinear amplification before affecting hearing sensitivity, supporting the view that normal audiograms do not necessarily reflect intact cochlear processing. This reframes early auditory decline as a disruption in the cochlear gain mechanism, influenced by age-related changes, basal cochlear vulnerability, and subtle variations in hearing sensitivity despite a normal audiogram. More broadly, these findings support a shift toward mechanism-based assessment of hearing, enabling earlier detection and more precise monitoring of cochlear function before overt hearing loss.

Figures Legend

Figure 1: Individual (thin lines) and mean (thick lines) hearing thresholds as a function of frequency. The shaded areas represent 95% confidence intervals of the mean for extended high frequency (EHF) loss (n=30) and EHF normal (n=114) ears. The EHF-normal and EHF-loss groups were categorized solely for visualization.

Figure 2: Mean distortion product otoacoustic emissions levels plotted as a function of L_2 level for 1000, 2000, 4000, and 6000 Hz. The shaded area represents ±95% confidence intervals computed across ears at each level following trial averaging for both DPOAE level and noise floor. The dashed vertical red line indicates the estimated breakpoint separating the near-linear (pink fits) and compressive growth (green fits) regions, and the horizontal black line represents the 95% confidence interval of the breakpoint estimate.

Figure 3: Representative examples of distortion product otoacoustic emission (DPOAE) input/output functions fitted using a segmented regression method at various f_2 frequencies ranging from 1000 to 6000 Hz. The blue line represents the DPOAE levels, the grey line represents the noise floor, the pink fitted line represents the low-level linear slope, the green fitted line represents the compressive slope, and the red vertical line represents the breakpoint. Note that the breakpoint decreases as frequency increases.

Figure 4: Box-dot plots illustrating cochlear compression estimates (Slope 1, breakpoint, and Slope 2) for various f_2 frequencies. Each box represents the interquartile range (IQR: 25th–75th percentiles), with the mean indicated by the black broken line; the horizontal bar within the box represents the median, and whiskers are 1.5 times the IQR. Individual data points are shown as dots, providing a visualization of the distribution of values.

Figure 5: LMEM adjusted compression metrics (Slope 1, 2, and breakpoint) at 4000 Hz as a function of noise exposure score, 16000 Hz threshold, and age. Trendlines represent linear mixed-effects model fits adjusted for covariates. The 95% confidence interval is shown only for statistically significant relationships (p <0.05). A significant negative association was observed between age and breakpoint, indicating altered compression with increasing age [$Breakpoint_{4000Hz}$ $=72.36-22.97(Age_{log})+2.08(Sex_{Male})+0.001(Threshold_{16000})-0.001(Threshold_{4000})$ $-0.79(Ear_{Right})+0.43(NESI_{log})$]. No other predictors showed a significant association with compression parameters at 4000 Hz.

Figure 6: LMEM adjusted compression metrics (Slope 1, Slope 2, and Breakpoint) at 6000Hz as a function of noise exposure, 16000 Hz threshold, and age. Trendlines represent linear mixed-effects model fits adjusted for covariates (such as age, sex, and ear). The 95% confidence interval is shown only for statistically significant relationships (p < 0.05). A significant negative association was observed between 16000 Hz threshold and Slope 2 (adj p=0.03), indicating altered compression with elevated 16000 Hz thresholds [$Slope2_{6000}=0.10+0.16(Age)-0.03(Sex_{Male})-0.002(Threshold_{16000})+0.004(Threshold_{6000})-0.05(Ear_{Right})+0.04(NESI)$].

Figure 7: Mean distortion product otoacoustic emission (DPOAE) levels plotted as a function of L_2 level at 4000 Hz for middle-aged participants (mean age ~43 years; range: 38–52 years; n=8 ears). The shaded regions represent ±95% confidence intervals computed across ears at each level following trial averaging for both the DPOAE levels (blue) and the noise floor (grey). The dashed vertical red line indicates the estimated breakpoint separating the near-linear (pink fit) and compressive growth (green fit) region.

References

Abdala C, Ortmann AJ, Guardia YC (2021) Weakened cochlear nonlinearity during human aging and perceptual correlates. Ear and hearing 42:832-845.

Arnold DJ, Lonsbury-Martin BL, Martin GK (1999) High-frequency hearing influences lower-frequency distortion-product otoacoustic emissions. Archives of Otolaryngology–Head & Neck Surgery 125:215-222.

Aryal S, Mishra SK (2025a) Probing cochlear compressive nonlinearity using signal-to-noise ratio–optimized distortion product otoacoustic emission input/output functions. The Journal of the Acoustical Society of America 158:4335-4347.

Aryal S, Mishra SK (2025b) On the Sharpness of Auditory Filters: Considering Subclinical Deficits Reveals Sharper Otoacoustic Emission Estimates of Frequency Selectivity in Humans. Journal of the Association for Research in Otolaryngology 26:699-714.

Aryal S, Trevino M, Rodrigo H, Mishra S (2025) Is Noise Exposure Associated With Impaired Extended High Frequency Hearing Despite a Normal Audiogram? A Systematic Review and Meta-Analysis. Trends in Hearing 29:23312165251343757.

Aryal S, Cheng F-Y, Mishra SK, Smith S (2026a) Brainstem encoding of speech in the extended high frequencies and its behavioral correlates. Journal of Neurophysiology 135:190–201.

Aryal S, Flores S, Smith SB, Boothalingam S, Mishra SK (2026b) Associations Between Extended High-Frequency Thresholds and Middle Ear Muscle Reflex Growth Functions in Adults With Normal Audiograms. Ear & Hearing.

Attias J, Horovitz G, El-Hatib N, Nageris B (2001) Detection and clinical diagnosis of noise-induced hearing loss by otoacoustic emissions. Noise and Health 3:19-31.

Avan P, Elbez M, Bonfils P (1997) Click-evoked otoacoustic emissions and the influence of high-frequency hearing losses in humans. The Journal of the Acoustical Society of America 101:2771-2777.

Badri R, Siegel JH, Wright BA (2011) Auditory filter shapes and high-frequency hearing in adults who have impaired speech in noise performance despite clinically normal audiogramsa). The Journal of the Acoustical Society of America 129:852-863.

Benjamini Y, Hochberg Y (1995) Controlling the false discovery rate: a practical and powerful approach to multiple testing. Journal of the Royal statistical society: series B (Methodological) 57:289-300.

Bharadwaj HM, Masud S, Mehraei G, Verhulst S, Shinn-Cunningham BG (2015) Individual differences reveal correlates of hidden hearing deficits. Journal of Neuroscience 35:2161–2172.

Bhatt IS, Lichtenhan J, Tyler R, Goodman S (2023) Influence of tinnitus, lifetime noise exposure, and firearm use on hearing thresholds, distortion product otoacoustic emissions, and their relative metric. The Journal of the Acoustical Society of America 154:418-432.

Bom SJH, De Leenheer EMR, Lemaire FX, et al. (2001) Speech recognition scores related to age and degree of hearing impairment in DFNA2/KCNQ4 and DFNA9/COCH. Archives of Otolaryngology–Head & Neck Surgery 127:1045–104

Borjigin A, Bharadwaj HM (2025) Individual differences elucidate the perceptual benefits associated with robust temporal fine-structure processing. Proceedings of the National Academy of Sciences 122:e2317152121.

Braza MD, Corbin NE, Buss E, Monson BB (2022) Effect of Masker Head Orientation, Listener Age, and Extended High-Frequency Sensitivity on Speech Recognition in Spatially Separated Speech. Ear and Hearing 43:90-100.

Cooper NP, Rhode WS (1995) Nonlinear mechanics at the apex of the guinea-pig cochlea. Hearing research 82:225-243.

Dhar S, Shaffer LA (2004) Effects of a suppressor tone on distortion product otoacoustic emissions fine structure: Why a universal suppressor level is not a practical solution to obtaining single-generator DP-grams. Ear and hearing 25:573-585.

Dorn PA, Konrad-Martin D, Neely ST, Keefe DH, Cyr E, Gorga MP (2001) Distortion product otoacoustic emission input/output functions in normal-hearing and hearing-impaired human ears. The Journal of the Acoustical Society of America 110:3119-3131.

Engdahl B, Kemp DT (1996) The effect of noise exposure on the details of distortion product otoacoustic emissions in humans. The Journal of the Acoustical Society of America 99:1573-1587.

Faraway JJ (2016) Extending the linear model with R: generalized linear, mixed effects and nonparametric regression models: Chapman and Hall/CRC.

Glavin CC, Siegel J, Dhar S (2021) Distortion product otoacoustic emission (DPOAE) growth in aging ears with clinically normal behavioral thresholds. Journal of the Association for Research in Otolaryngology 22:659-680.

Gorga MP, Neely ST, Ohlrich B, Hoover B, Redner J, Peters J (1997) From laboratory to clinic: a large scale study of distortion product otoacoustic emissions in ears with normal hearing and ears with hearing loss. Ear Hear 18:440-455.

Green P, MacLeod CJ (2016) SIMR: an R package for power analysis of generalized linear mixed models by simulation. Methods in Ecology and Evolution 7:493-498.

Guest H, Dewey RS, Plack CJ, Couth S, Prendergast G, Bakay W, Hall DA (2018) The Noise Exposure Structured Interview (NESI): An instrument for the comprehensive estimation of lifetime noise exposure. Trends in hearing 22:2331216518803213.

Guinan JJ, Jr., Backus BC, Lilaonitkul W, Aharonson V (2003) Medial olivocochlear efferent reflex in humans: otoacoustic emission (OAE) measurement issues and the advantages of stimulus frequency OAEs. J Assoc Res Otolaryngol 4:521–540.

Jiang W, Zhao F, Guderley N, Manchaiah V (2016) Daily music exposure dose and hearing problems using personal listening devices in adolescents and young adults: A systematic review. International Journal of Audiology 55:197-205.

Johnson T, Cooper S, Stamper G, Chertoff M (2017) Noise Exposure Questionnaire (NEQ): A Tool for Quantifying Annual Noise Exposure. Journal of the American Academy of Audiology 28:14-35.

Keefe DH, Schairer KS (2011) Specification of absorbed-sound power in the ear canal: application to suppression of stimulus frequency otoacoustic emissions. The Journal of the Acoustical Society of America 129:779-791.

Kemp DT (1978) Stimulated acoustic emissions from within the human auditory system. The Journal of the Acoustical Society of America 64:1386-1391.

Kummer P, Janssen T, Arnold W (1998) The level and growth behavior of the 2 f1− f2 distortion product otoacoustic emission and its relationship to auditory sensitivity in normal hearing and cochlear hearing loss. The Journal of the Acoustical Society of America 103:3431-3444.

Kutner M, Christopher, Nachtsheim JC, Neter J, Li W (2005) In Applied Linear Statistical Models.

Le Prell CG, Spankovich C, Lobarinas E, Griffiths SK (2013) Extended high-frequency thresholds in college students: effects of music player use and other recreational noise. Journal of the American Academy of Audiology 24:725-739.

Le Prell CG, Siburt HW, Lobarinas E, Griffiths SK, Spankovich C (2018) No reliable association between recreational noise exposure and threshold sensitivity, distortion product

otoacoustic emission amplitude, or word-in-noise performance in a college student population. Ear and hearing 39:1057-1074.

Lee J, Dhar S, Abel R, Banakis R, Grolley E, Lee J, Zecker S, Siegel J (2012) Behavioral hearing thresholds between 0.125 and 20 kHz using depth-compensated ear simulator calibration. Ear Hear 33:315-329.

López-Otín C, Blasco MA, Partridge L, Serrano M, Kroemer G (2013) The hallmarks of aging. Cell 153:1194-1217.

Martin GK, Stagner BB, Lonsbury-Martin BL (2010) Evidence for basal distortion-product otoacoustic emission components. The Journal of the Acoustical Society of America 127:2955-2972.

Martin GK, Stagner BB, Chung YS, Lonsbury-Martin BL (2011) Characterizing distortion-product otoacoustic emission components across four species. The Journal of the Acoustical Society of America 129:3090-3103.

McDermott JH, Schemitsch M, Simoncelli EP (2013) Summary statistics in auditory perception. Nature neuroscience 16:493-498.

Mehrparvar AH, Mirmohammadi SJ, Ghoreyshi A, Mollasadeghi A, Loukzadeh Z (2011) High-frequency audiometry: a means for early diagnosis of noise-induced hearing loss. Noise and Health 13:402-406.

Mills JH, Schmiedt RA, Schulte BA, Dubno JR (2006) Mechanisms of Age-Related Hearing Loss. Perspectives on Hearing and Hearing Disorders: Research and Diagnostics 10:2-9.

Mishra SK, Saxena U, Rodrigo H (2022) Hearing impairment in the extended high frequencies in children despite clinically normal hearing. Ear and hearing 43:1653-1660.

Mishra SK, Rodrigo H, Balan JR (2024) Exploring the Influence of Extended High-Frequency Hearing on Cochlear Functioning at Lower Frequencies. Journal of Speech, Language, and Hearing Research:1-10.

Mishra SK, Saxena U, Rodrigo H (2025) Early signs of auditory aging: Hearing declines faster in individuals with extended high frequency hearing loss. Hearing Research 456:109171.

Mishra SK, Fu Q-j, Galvin JJ, III, Galindo A (2023) Suprathreshold auditory processes in listeners with normal audiograms but extended high-frequency hearing lossa). The Journal of the Acoustical Society of America 153:2745-2750.

Moulin A, Bera J-C, Collet L (1994) Distortion product otoacoustic emissions and sensorineural hearing loss. Audiology 33:305-326.

Muggeo VM (2008) Segmented: an R package to fit regression models with broken-line relationships. R news 8:20-25.

Muggeo VMR (2003) Estimating regression models with unknown break-points. Statistics in Medicine 22:3055-3071.

Nelson DA, Schroder AC, Wojtczak M (2001) A new procedure for measuring peripheral compression in normal-hearing and hearing-impaired listeners. The Journal of the Acoustical Society of America 110:2045-2064.

Noda T, Nishida K, Chretien B, Fukui K, Tabuki T, Kubota H, Matsunaga T, Higashino Y, Ishikawa K, Komune N, Matsumoto N, Nakagawa T (2026) Subtle Peripheral Auditory Dysfunction in Patients With Listening Difficulties. Ear Hear 47:343-351.

Olejnik S, Algina J (2003) Generalized eta and omega squared statistics: measures of effect size for some common research designs. Psychological methods 8:434.

Ortmann AJ, Abdala C (2016) Changes in the compressive nonlinearity of the cochlea during early aging: Estimates from distortion OAE input/output functions. Ear and hearing 37:603-614.

Oxenham AJ, Plack CJ (1997) A behavioral measure of basilar-membrane nonlinearity in listeners with normal and impaired hearing. The Journal of the Acoustical Society of America 101:3666-3675.

Oxenham AJ, Bacon SP (2003) Cochlear compression: perceptual measures and implications for normal and impaired hearing. Ear and hearing 24:352-366.

Pienkowski M. (2017). On the Etiology of Listening Difficulties in Noise Despite Clinically Normal Audiograms. Ear and hearing 38:135–148

Poling GL, Siegel JH, Lee J, Lee J, Dhar S (2014) Characteristics of the 2f1-f2 distortion product otoacoustic emission in a normal hearing population. The Journal of the Acoustical Society of America 135:287-299.

Poortere ND, Verhulst S, Degeest S, Keshishzadeh S, Dhooge I, Keppler H (2023) Evaluation of Lifetime Noise Exposure History Reporting. Journal of Speech, Language, and Hearing Research 66:5129-5151.

Portugal N, Poling GL, Dreisbach L (2024) Rethinking the clinical utility of distortion-product otoacoustic emission (DPOAE) signal-to-noise ratio. International journal of audiology 63:491-499.

Robles L, Ruggero MA (2001) Mechanics of the mammalian cochlea. Physiological reviews 81:1305-1352.

Robles L, Ruggero MA, Rich NC (1986) Basilar membrane mechanics at the base of the chinchilla cochlea. I. Input–output functions, tuning curves, and response phases. The Journal of the Acoustical Society of America 80:1364-1374.

Ruggero M, Rich N, Recio A (1996) The effect of intense acoustic stimulation on basilar-membrane vibrations. Auditory Neuroscience 2:329-345.

Ruggero MA, Rich NC, Recio A, Narayan SS, Robles L (1997) Basilar-membrane responses to tones at the base of the chinchilla cochlea. The Journal of the Acoustical Society of America 101:2151-2163.

Ruggles D, Bharadwaj H, Shinn-Cunningham BG (2011) Normal hearing is not enough to guarantee robust encoding of suprathreshold features important in everyday communication. Proc Natl Acad Sci U S A 108:15516-15521.

Scheperle RA, Neely ST, Kopun JG, Gorga MP (2008) Influence of in situ, sound-level calibration on distortion-product otoacoustic emission variability. The Journal of the Acoustical Society of America 124:288-300.

Schmiedt RA (2009) The physiology of cochlear presbycusis. In: The aging auditory system, pp 9-38: Springer.

Shera CA, Guinan JJ, Oxenham AJ (2002) Revised estimates of human cochlear tuning from otoacoustic and behavioral measurements. Proceedings of the National Academy of Sciences 99:3318-3323.

Snir S, Farrell C, Pellegrini M (2019) Human epigenetic ageing is logarithmic with time across the entire lifespan. Epigenetics 14:912–926.

Souza NN, Dhar S, Neely ST, Siegel JH (2014) Comparison of nine methods to estimate ear-canal stimulus levels. The Journal of the Acoustical Society of America 136:1768-1787.

Stamper GC, Johnson TA (2015) Auditory function in normal-hearing, noise-exposed human ears. Ear Hear 36:172-184.

Von Békésy G (1960) Experiments in hearing.

Whiteford KL, Oxenham AJ (2015) Using individual differences to test the role of temporal and place cues in coding frequency modulation. The Journal of the Acoustical Society of America 138:3093-3104.

Yamasoba T, Lin FR, Someya S, Kashio A, Sakamoto T, Kondo K (2013) Current concepts in age-related hearing loss: Epidemiology and mechanistic pathways. Hearing Research 303:30-38.

Yates GK (1995) Cochlear structure and function. Hearing 2:41-74.

Table 1. Summary of linear mixed-effect model outcomes for distortion product otoacoustic emissions levels

	4000 Hz					6000 Hz			
L_2 Level	Predictor	Estimate (SE)	*p* value	**adj *p***	**Partial ω^2**	Estimate (SE)	*p* value	**adj *p***	**Partial ω^2**
70	Age	5.30 (9.29)	0.57	-	-	3.78 (9.56)	0.69	-	-
	Sex_{Male}	–0.64 (1.29)	0.62	-	-	–2.12 (1.55)	0.18	-	-
	16000 Hz threshold	–0.06 (0.03)	**0.03**	**0.04**	0.03	–0.1(0.03)	**0.004**	**0.01**	0.10
	Threshold at f_2	–0.29 (0.09)	**0.0009**	**0.004**	0.07	–0.26 (0.10)	**0.007**	**0.009**	0.06
	Ear_{Right}	0.22 (0.73)	0.77	-	-	–1.51 (0.86)	0.08	-	-
	NESI Score	–0.38 (0.86)	0.66	-	-	–1.14 (1.04)	0.28	-	-
50	Age	12.09 (8.97)	0.18	-	-	6.13 (9.92)	0.53	-	-
	Sex_{Male}	–1.09 (1.25)	0.38	-	-	–0.84 (1.61)	0.61	-	-
	16000 Hz threshold	–0.06(0.03)	**0.03**	0.05	0.03	–0.07 (0.03)	**0.04**	0.05	0.04
	Threshold at f_2	–0.33 (0.09)	**0.0001**	**0.0003**	0.09	–0.39 (0.10)	**0.0001**	**0.0003**	0.13
	Ear_{Right}	0.68 (0.74)	0.36	-	-	0.32 (0.91)	0.72	-	-
	NESI Score	–0.41 (0.83)	0.62	-	-	–2.32 (1.08)	**0.04**	0.15	0.07
20	Age	14.73 (12.11)	0.23	-	-	13.58 (11.80)	0.25	-	-
	Sex_{Male}	–3.27 (1.69)	0.06	-	-	–4.36 (1.92)	**0.03**	0.10	0.06
	16000 Hz threshold	–0.05 (0.04)	0.26	-	-	–0.04 (0.05)	0.36	-	-
	Threshold at f_2	–0.45 (0.12)	**0.0002**	**0.0008**	0.10	0.05 (0.15)	0.76	-	-
	Ear	1.46 (1.02)	0.15	-	-	1.06 (1.62)	0.51	-	-
	NESI Score	0.31 (1.11)	0.78	-	-	–1.62 (1.30)	0.22	-	-

Note: Threshold at f_2=Pure-tone threshold at 4000 Hz or 6000 Hz; NESI= Noise exposure structured interview; SE=Standard error; ω^2 = omega-squared effect size. All significant values are in bold.

914 **Table 2**: Summary of linear mixed effect model outcomes for cochlear compression metrics
915 (Slope 1, Slope 2, and Breakpoint)

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		Slope 1				**Slope 2**				**Breakpoint**			
Frequency (Hz)	Predictor	Estimate (SE)	*p* value	**adj *p***	**Partial ω^2**	Estimate (SE)	*p* value	**adj *p***	**Partial ω^2**	Estimate (SE)	*p* value	**adj *p***	**Partial ω^2**
4000	Age	–0.46 (0.42)	0.28	-	-	–0.26 (0.15)	0.08	-	-	–22.97 (9.19)	**0.01**	**0.04**	**0.08**
	Sex_{Male}	0.05 (0.05)	0.41	-	-	0.02 (0.02)	0.32	-	-	2.08 (1.23)	0.10	-	-
	16000 Hz threshold	–0.0005 (0.001)	0.72	-	-	0.0003 (0.0005)	0.52	-	-	0.001 (0.03)	0.96	-	-
	Threshold at f_2	0.009 (0.005)	**0.04**	0.05	0.03	0.0008 (0.0014)	0.61	-	-	–0.001 (0.10)	0.99	-	-
	Ear_{Right}	–0.04 (0.04)	0.41	-	-	–0.02 (0.01)	0.18	-	-	–0.79 (1.06)	0.46	-	-
	NESI Score	–0.03 (0.04)	0.40	-	-	–0.003 (0.01)	0.81	-	-	0.43 (0.81)	0.59	-	-
6000	Age	–0.28 (0.52)	0.59	-	-	0.16 (0.20)	0.42	-	-	–23.95 (13.19)	0.07	-	-
	Sex_{Male}	0.02 (0.08)	0.85	-	-	–0.03 (0.03)	0.36	-	-	–0.84 (2.00)	0.68	-	-
	16000 Hz threshold	0.0002 (0.002)	0.91	-	-	–0.002 (0.0008)	**0.009**	**0.03**	**0.08**	0.09 (0.05)	0.09	-	-
	Threshold at f_2	–0.002 (0.006)	0.73	-	-	0.003 (0.002)	0.09	-	-	0.18 (0.15)	0.25	-	-
	Ear_{Right}	–0.07(0.07)	0.33	-	-	–0.05 (0.02)	0.02	0.07	0.09	–0.35 (1.68)	0.83	-	-
	NESI Score	0.02 (0.05)	0.720	-	-	0.04 (0.02)	0.07	-	-	2.92 (1.39)	**0.04**	0.16	0.06

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918 **Note**: Threshold at f_2=Pure tone threshold at 4000 Hz or 6000 Hz; 16000 Hz threshold=Pure-
919 tone threshold at 16000 Hz; SE= Standard error; Slope 1=Low level linear slope; Slope
920 2=Compressive slope; Breakpoint =Estimated compression threshold; ω²= Omega squared
921 effect size. All significant values are in bold.

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