



QUANTITATIVE MODELLING OF ACOUSTIC EXPOSURE AND AUDITORY HEALTH: RISK PATTERNS, SYMPTOM ASSOCIATIONS, AND PREDICTIVE RELATIONSHIPS

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Abstract

Although acoustic exposure is a significant factor in auditory dysfunction, it can also cause changes beyond what has traditionally been considered "hearing threshold" and involve neural and functional auditory processing. Multiple indicators of auditory health were quantitatively correlated with lifetime noise exposure in this study with assessments of frequency-specific hearing loss, auditory brainstem response (ABR) measures, and speech-in-noise (SIN). Data on 74 participants were analysed for descriptive statistics, correlation analysis, comparison of groups and multivariable linear regression. Positive correlation of lifetime noise exposure was statistically significant for higher high-frequency hearing loss and lower amplitude of the auditory brainstem response Wave I. When the effects of demographic factors were accounted for, age was the most important factor associated with high- and extended-high-frequency hearing loss, while lifetime noise exposure was still independently associated with decreased amplitude of Wave I. The hearing loss at high frequency and attentional performance were significant contributors to speech-in-noise performance, but not lifetime acoustic exposure. The results suggest that the accumulated acoustic exposure can have specificity in neural auditory function in the periphery and early neural function, but not necessarily in all aspects of hearing. Therefore, a multidimensional approach using exposure history, hearing thresholds, electrophysiological responses and functional listening measures might be more comprehensive in identifying subtle noise-related auditory risk. More extensive longitudinal research is needed to establish cause-and-effect relationships and the predictive capacity.

Keywords: lifetime noise exposure; auditory health; high-frequency hearing loss; auditory brainstem response; speech-in-noise performance

1. Introduction

In addition to communication, occupational safety, social participation, and quality of life, hearing is an important part of life, and chronic acoustic exposure is an important, and potentially preventable, cause of auditory dysfunction. Worldwide data show that hearing loss is a significant and increasing burden of disease, both within and across age groups and geographical areas, and has significant implications for communication and healthy ageing (Haile et al., 2021). The evidence of persistent inequalities in the burden of hearing loss and the need to identify environmental factors that can be changed is also demonstrated by international trend analyses (Jiang et al., 2023). In addition to population ageing, the exposure to occupational, recreational, and amplified noise has led to increasing concerns about noise-induced progressive hearing loss and the early detection of dysfunctions with sensitive quantitative measures (Dong et al., 2025).

The mechanisms of noise-induced hearing loss are complex, involving mechanical, metabolic and neural processes. Repeated high-intensity noise exposure without regular hearing protection is a key risk factor in the workplace, particularly in workplaces where high-intensity sounds are present repeatedly (Chen et al., 2020). Evidence from India also shows that there is a significant burden of disease in the occupation and provides justification for intensified surveillance and preventive measures for exposed populations (Basu et al., 2022). At the cellular level, excessive acoustic stimulation causes oxidative stress, mitochondrial dysfunction, inflammatory response, and damages the cochlear structures, which renders a mechanism to explain progressive threshold elevation and auditory impairment (Kitama et al., 2025). But while normal audiometry can detect some types of noise-induced damage, others may not be detected, especially where there is no obvious threshold loss and neural degeneration is the cause.

This restriction put a new spotlight on cochlear synaptopathy and what is known as “hidden hearing loss.” Experimental and human studies indicate that even though audiometric thresholds are clinically normal, synaptic injury between inner hair cells and auditory-nerve fibres may compromise their coding fidelity (Aedo & Aguilar, 2020). These synaptopathic changes have been associated with changes in loudness perception and suprathreshold auditory problems (Çildir et al., 2022). Further reviews of the literature highlight the importance of combining behavioural, electrophysiologic and exposure-based measures when examining subtle auditory deficits (Colla et al., 2023). This is especially important with regard to extended-high frequency audiometry, which may show damage at frequencies higher than the standard frequencies that are conventionally used for hearing assessments, prior to the onset of clinically detectable loss at those frequencies (Hunter et al., 2020). In recent times, meta-analytical studies have suggested that noise exposure may be linked to impaired extended-HF thresholds even though traditional audiograms are normal (Aryal et al., 2025).

In fact, recreational noise is now considered to be an important part of lifetime acoustic exposure. Personal listening devices, concerts, clubs or other environments with sound amplification can create significant personal exposure for young adults and both exposure behaviour and attitudes towards hearing protection can affect hearing outcomes (Degeest et al., 2022). There are also systematic evidence associations between recreational noise exposure and hearing function changes in adolescents and young adults, but there exists heterogeneity in exposure measurement (Elmazoska et al., 2024). Public health programs to encourage safe listening at entertainment venues are then still relevant to hearing conservation (Eichwald, 2023).

In addition to threshold sensitivity, auditory health is comprised of functional listening and symptom burden. Suprathreshold testing can be useful for determining in the real world how well a person can hear words in noise, even when the person's conventional hearing is fairly normal. In addition to measurable and hidden hearing loss, subjective tinnitus is another clinically relevant manifestation of altered auditory processing that could coexist with the other two (Kang & Zheng, 2024). These factors justify a multidimensional approach to the analysis, where lifetime exposure, frequency-specific hearing sensitivity, neural auditory responses, and functional performance of listening are analyzed together.

The study uses quantitative modelling to quantify the acoustic-exposure patterns and identify the relationship between cumulative exposure and more than one auditory outcome. The study combines demographic covariates with measures of hearing, electrophysiology, and speech-in-noise, and aims to shed light on whether acoustic exposure is a risk factor for the auditory system independent of age effects. The integrated framework could enhance the interpretation of auditory changes from a variety of exposures and ages.

Objectives:

- ☐ To measure lifetime acoustic exposure and frequency-specific auditory sensitivity.
- ☐ To investigate the relationship between acoustic exposure, hearing thresholds, neural auditory responses, and speech-in-noise performance.
- ☐ To develop and model independent predictors of adverse auditory outcomes.

2. Materials and Methods

2.1 Study Design and Analytical Framework

The research design used for this study was a quantitative secondary data analytical design. The analytical framework used three key areas: acoustic-exposure patterns, relationships between lifetime exposure and audiology outcomes, and predictive relationships between exposure and hearing-related measures. Objective audiological measures were emphasized such as hearing sensitivity, auditory brainstem responses, and speech-in-noise performance.

2.2 Data Source

Data for “Effects of lifetime noise exposure on the middle-aged human auditory brainstem response and speech-in-noise intelligibility” were used for the analysis. The data set was created to examine the relationship between cumulative lifetime noise exposure and hearing sensitivity, auditory brainstem responses and speech-in-noise performance for middle aged adults. Variables directly related to acoustic exposure, auditory function, and demographic and cognitive performance

were included in the present study. The final dataset that was analyzed consisted of 74 respondents and 19 variables, with one row corresponding to one respondent (Valderrama et al., 2018).

2.3 Study Variables

Study variables were organized into exposure, auditory health, demographic and cognitive domains. The main independent variable was lifetime noise exposure (LNE), measured as $\log_{10}$ Pa²h, and analysed continuously, using tertile groups for descriptive comparisons of lower, intermediate and higher exposure groups. Auditory outcomes were low-frequency hearing loss, high-frequency hearing loss, extended-high-frequency hearing loss (all measured in dB HL), Waves I, III, V interpeak latency, amplitude of Waves I, III, V (measured in dB), and LiSN-HC speech-in-noise performance (measured in dB SNR). Demographic covariates included age and gender, and a cognitive covariate was the Test of Everyday Attention (TEA) score, especially in analysis of speech-in-noise performance.

2.4 Data Preparation and Quality Control

The data set was checked for duplicity, missing data, incorrect data and consistency of data variables prior to the analysis. All data were available for age, gender, lifetime noise exposure, hearing-loss measurements, TEA score, and LiSN-HC. The missing observations were primarily limited to a few variables from ABR, and were not imputed, but rather left as missing. Analyses were thus performed with available observations for these variables. Some continuous variables were also evaluated for extreme values and distributional properties prior to statistical testing.

2.5 Descriptive and Exposure-Pattern Analysis

Participant characteristics, acoustic exposures, and auditory outcomes were summarized using descriptive statistics. Continuous variables were presented as mean, standard deviation, median, interquartile range, minimum and maximum as suitable and categorical variables were presented as frequencies and percentages. Lifetime noise was analysed both as a continuous variable and, if appropriate, in tertiles. For each exposure category, auditory outcome was compared using one-way ANOVA if the parametric assumptions were met or the Kruskal-Wallis test if the parametric assumptions were not met.

2.6 Bivariate Association Analysis

Correlation analysis was used to examine the associations between lifetime noise exposure and auditory-health measures. Pearson's correlation coefficient was applied for continuous variables that were normally distributed and linearly related, while Spearman's rank correlation was applied when there were variables that were not normally distributed and/or not linearly related. The associations between LNE and hearing-loss measures as well as between ABR parameters and LiSN-HC were explored, as were associations between hearing sensitivity and neural auditory responses and speech-in-noise performance.

2.7 Multivariable Predictive Modelling

The auditory-health outcomes were assessed with respect to lifetime noise exposure using multiple linear regression, after controlling for relevant covariates. Separate models were developed for the extended high-frequency hearing loss, selected ABR parameters, LiSN-HC, and high-frequency hearing loss. The main predictor was lifetime noise exposure and age and gender were added as covariates in hearing models. In addition to hearing sensitivity, models of speech-in-noise performance included the TEA score. The number of predictors was deliberately reduced to avoid overfitting and model instability to have a manageable sample of 74 participants.

2.8 Model Diagnostics and Statistical Assumptions

The models were evaluated for regression assumptions prior to model interpretation. Linearity was checked with the scatter plots, normality was checked with the distribution of the residuals, and homoscedasticity was checked with the residual-versus-fitted plots. To check for multicollinearity, tolerance statistics and variance inflation factors were used. Standardized residuals, leverage statistics and Cook's distance were used to examine influential observations. Model performance was reported with regression coefficients, standard error, 95% confidence interval, R^2 , adjusted R^2 and p value.

2.9 Sensitivity Analysis

Robustness of the key exposure-outcome relationships was assessed by conducting sensitivity analyses. Exposure-group comparisons were secondary analyses; continuous LNE were primary analyses. The magnitude of associations was compared between adjusted and unadjusted models to examine if differences in age and gender significantly changed the observed associations. Variations in the number of samples reported indicate that some electrophysiological measurements were not recorded for ABR analyses.

2.10 Statistical Software and Significance Criteria

All statistical tests were two-tailed, with a significance level of $p < 0.05$. Effect estimates, 95% confidence intervals, and measures of explained variance were interpreted alongside p-values to provide a comprehensive assessment of statistical relationships. Graphical analyses were additionally used to support the interpretation of acoustic-exposure patterns, auditory outcomes, and regression diagnostics.

2.11 Ethical Considerations

The research in this study was a secondary analysis of an existing data set, with no need for new recruitment of participants or collection of personal data. Anonymized study codes were used as participant identifiers, and only these were used for distinguishing observations. Only those variables relevant to acoustic exposure and auditory health were analysed in the present study. Ethical approval, informed consent and participant protection procedures were consistently traceable to the original study and its documentation of the data sets. The secondary analysis was carried out following the conditions set for the data use and principles of responsible research-data management.

3. Results

3.1 Participant Characteristics, Acoustic Exposure, and Auditory Profile

A total of 74 participants (37 males and 37 females) were included in the analytical sample. The average age was 43.36 ± 6.94 years, ranging from 29 to 55 years. There was also a significant variation in lifetime noise exposure, with a mean of $3.58 \pm 0.66 \log_{10} \text{Pa}^2\text{h}$ and a range from 1.43 to $4.93 \log_{10} \text{Pa}^2\text{h}$. As shown in Table 1, mean hearing loss was significantly higher with increasing frequency bands, with an increase from $5.91 \pm 4.35 \text{ dB HL}$ at low frequencies to $9.41 \pm 6.75 \text{ dB HL}$ at high frequencies and $20.67 \pm 16.72 \text{ dB HL}$ at extended-high frequencies. The extended-high frequency hearing loss range was quite broad (40.50 dB HL) and included some individuals with very high sensitivity (71.50 dB HL) and others with very low sensitivity (-4.00 dB HL), suggesting that there was considerable heterogeneity of hearing sensitivity within the sample.

The mean LiSN-HC score was $-20.14 \pm 2.92 \text{ dB SNR}$, while the mean TEA score was 8.03 ± 2.01 . The mean amplitude of ABR Wave I was $0.22 \pm 0.10 \mu\text{V}$, and the mean amplitude of ABR Wave V was $0.34 \pm 0.13 \mu\text{V}$. The latencies of mean Wave I and Wave V were 1.89 and 6.13 ms, respectively. The distribution of cumulative lifetime noise exposure is shown in Figure 1, which has a wide range of exposures appropriate for exposure-response modelling.

Table 1. Descriptive characteristics of acoustic exposure and auditory-health variables

Variable	n	Mean	SD	Minimum	Maximum
Age, years	74	43.36	6.94	29.00	55.00
Lifetime noise exposure, $\log_{10} \text{Pa}^2\text{h}$	74	3.58	0.66	1.43	4.93
Low-frequency hearing loss, dB HL	74	5.91	4.35	-2.50	19.00
High-frequency hearing loss, dB HL	74	9.41	6.75	-1.33	28.67
Extended-high-frequency hearing loss, dB HL	74	20.67	16.72	-4.00	71.50
TEA score	74	8.03	2.01	1.00	10.00
LiSN-HC, dB SNR	74	-20.14	2.92	-28.00	-13.00
ABR Wave I TIP amplitude, μV	70	0.22	0.10	0.02	0.56
ABR Wave V MAS amplitude, μV	67	0.34	0.13	0.09	0.68
ABR Wave I TIP latency, ms	70	1.89	0.17	1.53	2.36
ABR Wave V MAS latency, ms	71	6.13	0.26	5.46	6.87
ABR I-V interpeak latency, ms	68	4.27	0.24	3.59	4.97

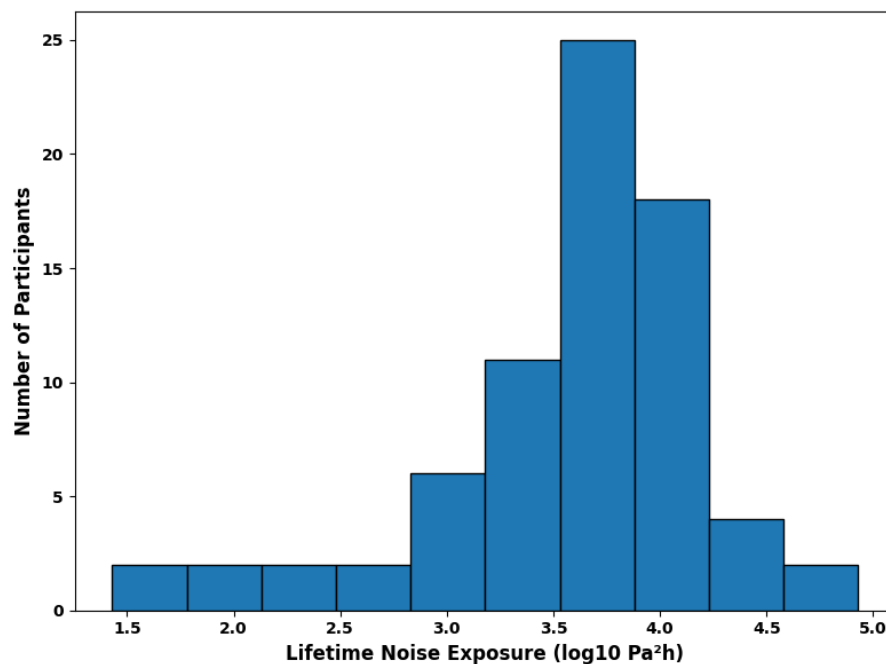


Figure 1. Distribution of lifetime noise exposure among the 74 participants, showing variability in cumulative acoustic exposure expressed as $\log_{10} \text{Pa}^2\text{h}$

3.2 Auditory Outcomes Across Lifetime Noise-Exposure Groups

Lower, intermediate, and higher exposure groups were defined by three tertiles, with 25, 25, and 24 individuals in each, respectively, to describe the patterns of exposure-related risk in a descriptive manner. The mean lifetime noise exposure was $2.89 \pm 0.62 \log_{10} \text{ Pa}^2\text{h}$ for the low group, $3.71 \pm 0.10 \log_{10} \text{ Pa}^2\text{h}$ for the middle group, and $4.18 \pm 0.25 \log_{10} \text{ Pa}^2\text{h}$ for the high group.

Table 2 shows that the average hearing level (HL) at low exposure was 4.96 dB, intermediate exposure 6.64 dB, and high exposure 6.15 dB. These differences were not significant ($F = 0.98$, $p = 0.379$). High frequency hearing loss was significantly higher in the intermediate group ($11.25 \pm 6.65 \text{ dB HL}$) compared to the lower-exposure group ($6.93 \pm 4.85 \text{ dB HL}$) and to the higher-exposure group ($10.06 \pm 7.94 \text{ dB HL}$). The overall difference was close to conventional statistical significance, $F = 2.87$, $p = 0.063$.

Higher exposure group and lower exposure group had an increased number of people with extended-high-frequency hearing loss, but the difference was not statistically significant. In the same way, there were no significant differences in LiSN-HC performance between the three exposure groups. Figure 2 shows the distribution of hearing loss at high frequencies by exposure category.

Table 2. Auditory outcomes according to lifetime noise-exposure tertile

Outcome	Lower exposure n = 25 Mean $\pm$ SD	Intermediate exposure n = 25 Mean $\pm$ SD	Higher exposure n = 24 Mean $\pm$ SD	F	p-value
Lifetime noise exposure, $\log_{10} \text{ Pa}^2\text{h}$	2.89 ± 0.62	3.71 ± 0.10	4.18 ± 0.25	68.37	<0.001
Low-frequency hearing loss, dB HL	4.96 ± 4.25	6.64 ± 4.62	6.15 ± 4.16	0.98	0.379
High-frequency hearing loss, dB HL	6.93 ± 4.85	11.25 ± 6.65	10.06 ± 7.94	2.87	0.063
Extended-high-frequency hearing loss, dB HL	18.73 ± 15.14	20.59 ± 12.83	22.77 ± 21.62	0.35	0.704
LiSN-HC, dB SNR	-20.32 ± 3.05	-19.85 ± 3.26	-20.25 ± 2.48	0.18	0.833

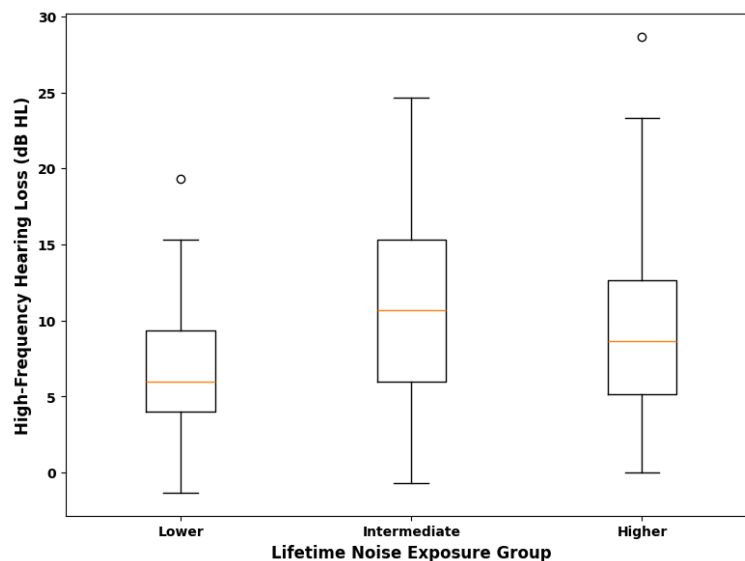


Figure 2. Distribution of high-frequency hearing loss across lower, intermediate, and higher lifetime noise-exposure groups

3.3 Associations Between Lifetime Noise Exposure and Auditory Outcomes

Selective associations between cumulative acoustic exposure and measures of auditory health were found using bivariate correlation analysis. High frequencies hearing loss showed significant positive relationship with lifetime noise exposure ($r = 0.251$; $p = 0.031$) as indicated in Table 3. This means that increased lifetime acoustic exposure correlated with increased hearing thresholds in the high-frequency range. The exposure-response relationship is displayed in Figure 3. The relationship between lifetime noise exposure and low-frequency hearing loss was positive but not statistically significant ($r = 0.200$ and $p = 0.088$), and with extended-high-frequency hearing loss was also positive but not statistically significant ($r = 0.167$ and $p = 0.156$).

The lifetime noise exposure and ABR Wave I TIP amplitude were significantly inversely associated ($r = -0.284$, $p = 0.017$). The Wave I amplitude was thus lower in participants who had a higher cumulative noise exposure. This relationship is depicted in Figure 4. Inverse (but not statistically significant) trends were also noted for the amplitude of Wave V and the I–V interpeak latency. There was no significant latency or LiSN-HC speech-in-noise association with lifetime exposure.

Table 3. Pearson correlations between lifetime noise exposure and auditory outcomes

Auditory outcome	n	Pearson r	p-value
Low-frequency hearing loss	74	0.200	0.088
High-frequency hearing loss	74	0.251	0.031
Extended-high-frequency hearing loss	74	0.167	0.156
ABR Wave I TIP amplitude	70	-0.284	0.017
ABR Wave V MAS amplitude	67	-0.225	0.067
ABR Wave I TIP latency	70	0.159	0.188
ABR Wave V MAS latency	71	0.033	0.786
ABR I–V interpeak latency	68	-0.222	0.069
LiSN-HC	74	0.079	0.503

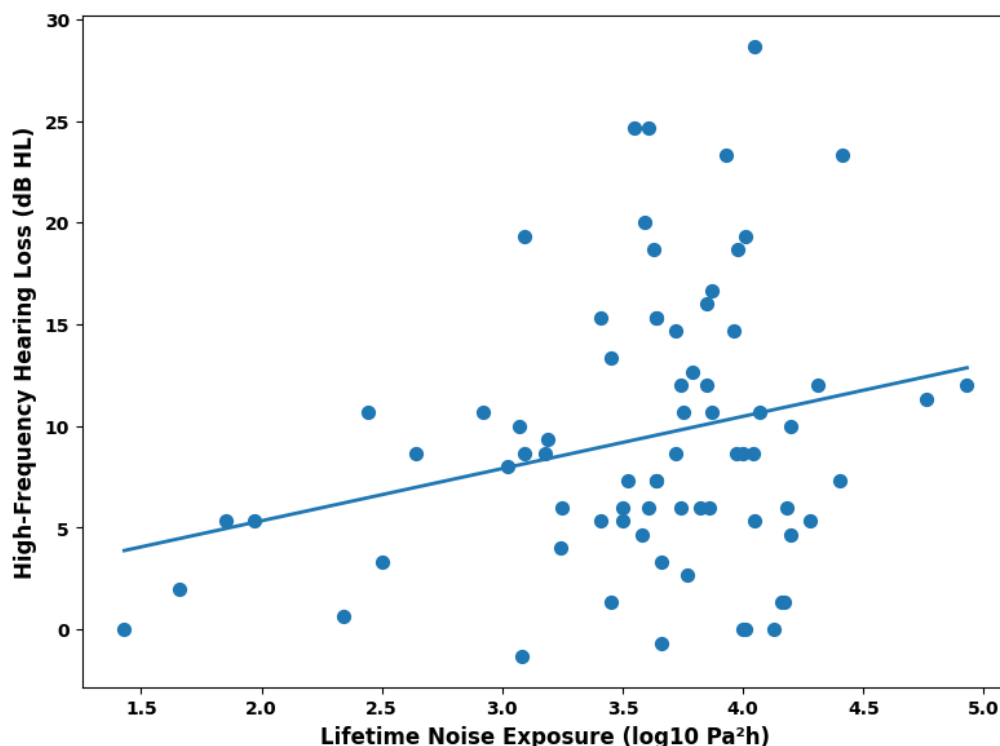


Figure 3. Relationship between lifetime noise exposure and high-frequency hearing loss

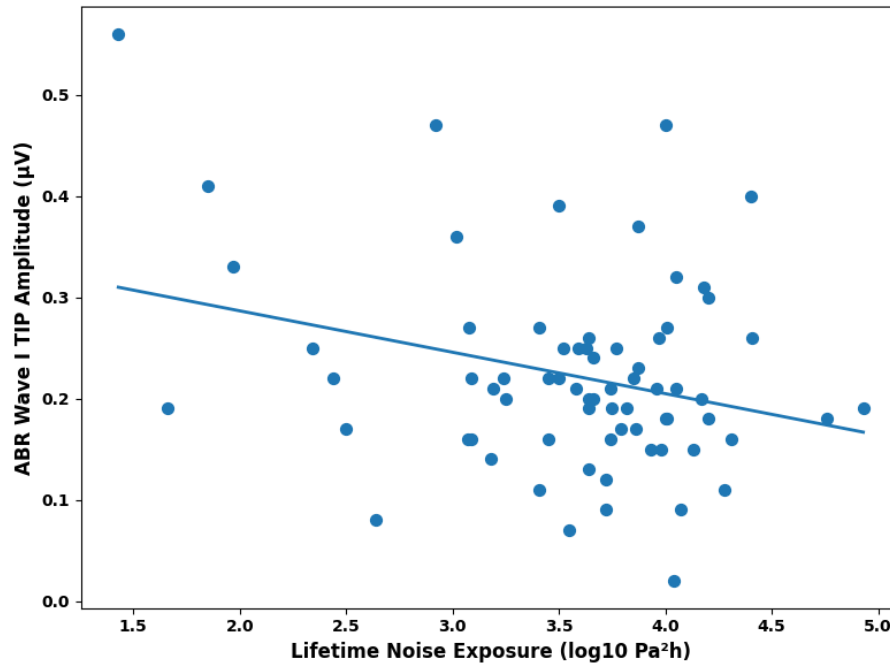


Figure 4. Relationship between lifetime noise exposure and ABR Wave I TIP amplitude

3.4 Predictors of Frequency-Specific Hearing Loss

Multiple linear regression models were constructed to assess whether the lifetime noise exposure predicted hearing sensitivity, while controlling for the effects of age and gender. The results are presented in Table 4.

The model predicting low-frequency hearing loss was not significant, with $R^2 = 0.052$, adjusted $R^2 = 0.011$, $F = 1.27$ and $p = 0.292$. The coefficient for lifetime noise exposure was positive but not significant: $B = 1.18$ dB HL ($\log_{10}$ Pa²h) ($p = 0.134$).

In the case of high-frequency hearing loss, the overall model was statistically significant, accounting for 16.0% of the variance, with an adjusted R^2 of 0.124, an F value of 4.46 and a p value of 0.006. The coefficient B of the association between lifetime noise exposure was positive ($B = 1.99$), but the association became more moderate after adjustment and failed to reach the threshold of 0.05 ($p = 0.085$). Age was a strong independent predictor with an estimated 0.30 dB HL increase per year of age; $p = 0.008$.

The extended-high frequency model was also statistically significant, explaining 20.1% of the variance, R^2 ; adjusted = 0.167, $F = 5.86$, and $p = 0.001$. The most powerful independent predictor was age ($B = 1.03$ dB HL/year, $p < 0.001$). In this adjusted model, lifetime noise exposure and gender were not independently significant.

Table 4. Multiple linear regression models predicting frequency-specific hearing loss

Outcome	Predictor	B	SE	Standardized β	p-value
Low-frequency hearing loss	Lifetime noise exposure	1.185	0.781	0.180	0.134
	Age	0.060	0.075	0.096	0.425
	Male gender	0.341	1.023	0.039	0.740
High-frequency hearing loss	Lifetime noise exposure	1.992	1.140	0.195	0.085
	Age	0.298	0.109	0.307	0.008
	Male gender	0.593	1.493	0.044	0.693
Extended-high-frequency hearing loss	Lifetime noise exposure	2.497	2.755	0.099	0.368
	Age	1.026	0.264	0.426	<0.001
	Male gender	-1.290	3.610	-0.039	0.722

Model statistics: low-frequency hearing loss, $R^2 = 0.052$; high-frequency hearing loss, $R^2 = 0.160$; extended-high-frequency hearing loss, $R^2 = 0.201$.

3.5 Neural Auditory Response and Functional Listening Models

A separate regression model was tested to determine if lifetime noise exposure also predicted ABR Wave 1 TIP amplitude while accounting for age and high frequency hearing loss. As noted in Table 5, the model accounted for about 9.2% of the variance in amplitude for Wave I. Reduced Wave I amplitude was independently associated with lifetime noise exposure ($B = -0.036 \mu\text{V}$ per $\log_{10} \text{Pa2h}$, standardized $\beta = -0.252$, and $p = 0.044$). This model was not significantly affected by age or high-frequency hearing loss. This modified outcome confirmed the inverse exposure-response trend depicted in Figure 4.

The overall model predicting Wave V latency was significant ($R^2 = 0.123$, adjusted $R^2 = 0.084$, $p = 0.031$). There was a significant relationship between age and Wave V latency ($B = 0.011 \text{ ms/year}$; $p = 0.020$); lifetime noise exposure was not significantly associated with Wave V latency.

The multivariable model accounted for 20.9% of variance in LiSN-HC performance, with an adjusted R^2 of 0.163, and a global p of 0.003. After adjustment, there was no association between lifetime noise exposure and LiSN-HC. The LiSN-HC score was significantly associated with hearing loss in a positive manner: $B = 0.123$, $p = 0.017$, and the TEA score was significantly associated with hearing loss in an inverse manner: $B = -0.371$, $p = 0.022$. This observed relationship between high-frequency hearing loss and LiSN-HC is shown in Figure 5.

Table 5. Multivariable models of neural and functional auditory outcomes

Outcome	Predictor	B	SE	Standardized β	p-value
ABR Wave I TIP amplitude	Lifetime noise exposure	-0.036	0.018	-0.252	0.044
	Age	-0.0001	0.002	-0.007	0.958
	High-frequency hearing loss	-0.002	0.002	-0.110	0.390
ABR Wave V latency	Lifetime noise exposure	-0.017	0.046	-0.043	0.717
	Age	0.011	0.005	0.293	0.020
	High-frequency hearing loss	0.005	0.005	0.124	0.329
LiSN-HC	Lifetime noise exposure	0.035	0.495	0.008	0.945
	High-frequency hearing loss	0.123	0.051	0.285	0.017
	Age	0.067	0.049	0.159	0.175
	TEA score	-0.371	0.158	-0.255	0.022

Model statistics: ABR Wave I amplitude, $n = 70$, $R^2 = 0.092$, adjusted $R^2 = 0.051$, $p = 0.093$; ABR Wave V latency, $n = 71$, $R^2 = 0.123$, adjusted $R^2 = 0.084$, $p = 0.031$; LiSN-HC, $n = 74$, $R^2 = 0.209$, adjusted $R^2 = 0.163$, $p = 0.003$.

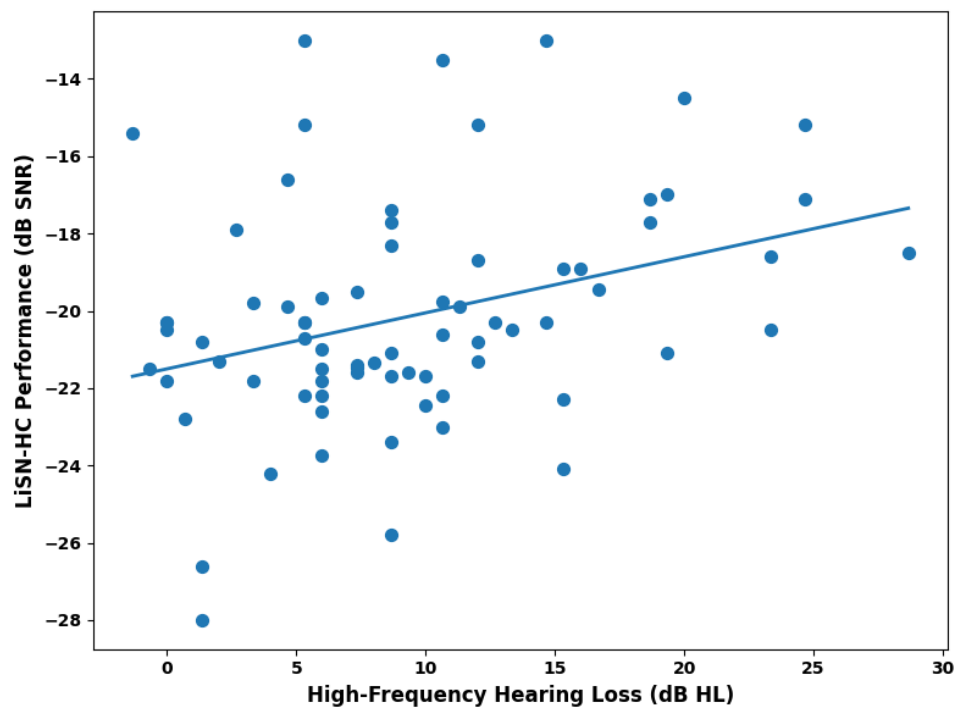


Figure 5. Relationship between high-frequency hearing loss and speech-in-noise performance measured using LiSN-HC

3.6 Overall Pattern of Findings

The two analyses revealed a distinct relationship between acoustic-exposure and auditory-health. Bivariate association of greater lifetime noise exposures with greater high-frequency hearing loss was significant and reduced ABR Wave I amplitude was significant in both unadjusted and adjusted results. Demographic adjustment, however, resulted in a greater amount of variability in the high and extended-high-frequency hearing sensitivity due to age. Speech-in-noise performance was not significantly related to lifetime noise exposure; however, high-frequency hearing loss and attentional performance were both predictive of variability in LiSN-HC. The results suggest that cumulative acoustic exposure was more strongly associated with high-frequency auditory sensitivity and early neural response amplitude, whereas age and cognitive-auditory factors played an important role in other aspects of auditory function.

4. Discussion

The present study focused on the relationship between cumulative acoustic exposure and frequency-specific hearing sensitivity, auditory brainstem responses and speech-in-noise performance. The unadjusted and adjusted analyses revealed greater lifetime noise exposure to be significantly associated with poorer hearing thresholds at the high frequencies, and with decreased amplitude of ABR Wave I. After multivariable adjustment, however, age proved to be a stronger predictor of the high- and extended-high-frequency hearing loss. Lifetime noise exposure was not independently associated with speech-in-noise performance; however, high-frequency hearing loss and attentional performance were. Acoustic exposure could selectively impact peripheral and early neural auditory function, but not impact all hearing outcomes equally, as indicated by these results.

The direction of the relationship between lifetime noise exposure and high-frequency hearing loss was positive and similar to other studies that found that suprathreshold and frequency-specific measures may be able to detect early auditory damage before a generalized hearing loss was apparent (Bhatt et al., 2022). Acoustic stress may result in a cumulative effect in high-frequency regions, and subtle threshold elevations may be an early sign of dysfunction related to exposure. No significant differences between the exposure tertiles, although there was a significant continuous correlation, indicates that information may be lost when continuous exposure is broken into tertiles. There was also significant variation in extended-high-frequency hearing loss, but this was not independently associated with lifetime exposure after adjustment. This is possible due to the environment and age dependence of extended-high-frequency thresholds (Gottfriedová et al., 2024a). This is similar to the results in younger PLD users that have been obtained using extended-high-frequency testing (Gottfriedová et al., 2024b).

In adjusted models, the best predictor of both high-frequency and extended-high-frequency hearing loss was age. This aligns with the existing literature on the impact of demographic and age-related factors on extended-high-frequency sensitivity in middle-aged and older adults (Helfer et al., 2024). The present results underline the importance of considering cumulative noise exposure without age. Cochlear degeneration due to aging may mask, amplify or overlap noise-related cochlear changes, emphasizing the importance of multivariable adjustment when estimating independent effects of noise exposure.

The most striking electrophysiologic observation was that there was an inverse correlation between lifetime noise exposure and the amplitude of ABR Wave I. The amplitude of Wave I is a widely studied potential non-invasive marker for cochlear synaptopathy, but is complicated to interpret in humans, due to the influence of recording conditions, cochlear status and subject individuality on the amplitude of Wave I (Bramhall, 2021). The relationship persisted after controlling for age and high-frequency hearing loss, indicating that cumulative exposure may have a relationship to the early characteristics of auditory nerve responses other than threshold sensitivity. Electrophysiological profiles are similar to this work and do support the use of exposure history as well as cochlear and brainstem measures in assessing suspected noise-related synaptopathy (Kaf et al., 2022). However, caution must be used when interpreting the ABR findings as human cochlear synaptopathy cannot be confirmed with a single electrophysiological marker.

There was a different pattern for speech-in-noise performance. There was no independent relationship of lifetime exposure to LiSN-HC, while high-frequency hearing loss and TEA scores accounted for performance. These findings aid the theory that the understanding of speech in complex environments is less the acoustic environment and more the interaction of peripheral hearing, central auditory processing, and cognition (Billings et al., 2024). More recent studies also show connections between the ABR characteristics, cognitive functions and perception of speech in noise, even when hearing thresholds are normal (DiNino et al., 2025). Speech perception has been discussed with regard to individual differences, particularly in relation to the potential for synaptopathy induced by noise exposure, but such relationships have not been found in all listeners (DiNino et al., 2021).

Tinnitus is relevant due to evidence in population studies linking cumulative and leisure noise exposure to the prevalence of tinnitus (Feder & Marro, 2025). Other auditory effects of exposure have also been associated with tinnitus presence and noise annoyance, and therefore with tinnitus distress (Hahad et al., 2025). All in all, the results corroborate the use of a multi-dimensional exposure history and hearing thresholds, neural responses, and listening measures. Limited causal inferences and generalizability due to small sample, cross-sectional design, and missing ABR observations. Longitudinal studies in the future should include exposure assessment, symptom measures and larger samples to better elucidate the relationship between noise exposure and peripheral, neural and functional auditory outcomes.

5. Conclusion

This study provides evidence for the measurable variation in auditory health that is correlated with cumulative acoustic exposure, especially in high-frequency hearing sensitivity and early neural auditory responses. High frequency hearing loss was also positively correlated with lifetime noise exposure, suggesting that chronic noise exposure may affect

peripheral hearing, and lifetime noise exposure was independently inversely correlated with ABR Wave I amplitude, suggesting that chronic noise exposure may also affect auditory-nerve function. But, after multivariable adjustment, age was a better predictor of high frequency and extended high frequency hearing loss, highlighting that age-related hearing loss is a more powerful predictor than exposure-related hearing loss. Speech-in-noise performance was not independently predicted by LNE but correlated with high-frequency hearing loss and attentional performance, indicating that functional listening ability may be related to interactions between sensory and cognitive processes. The results support the use of a multidimensional auditory assessment instead of only using traditional hearing thresholds. Lifetime exposure history, frequency specific hearing, electrophysiology and speech in noise testing may be useful in enhancing the ability to detect subtle hearing dysfunction when combined. However, the small number of subjects engaged in this study, the cross-sectional design and lack of electrophysiological measurements hinder causal interpretation. Larger numbers of participants, more comprehensive symptom measures, and repeated exposures should be measured in future longitudinal studies to further clarify mechanisms and enhance the prediction of auditory risk associated with noise.

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