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# THEORETICAL MODELING OF THE SPATIAL DISTRIBUTION OF FREQUENCY SENSITIVITY IN THE BASILAR MEMBRANE

**Tichaona Chikore**[1,2], **Farai Nyabadza**[2]

[1]Department of Applied Mathematics, University of Johannesburg, Johannesburg, South Africa
[2]Institute of Applied Research and Technology, Emirates Aviation University,
Dubai International Academic City, UAE

**Abstract.** Hearing loss, often caused by prolonged exposure to loud noises, is a critical public health issue, with the basilar membrane in the cochlea playing a central role in this process. The basilar membrane's ability to differentiate sound frequencies is vital for auditory perception, yet its response to noise exposure and the subsequent damage to cochlear hair cells are not fully understood. This gap provides a platform to propose a dynamic-cum-statistical modelling approach to understand the basilar membrane's response to various sound frequencies and the impact of noise-induced disturbances on hearing. The model incorporates a position-dependent sensitivity function along the basilar membrane and accounts for both normal and toxic frequencies. By integrating sensitivity and damage functions, the model demonstrates how different frequencies are processed along the basilar membrane and evaluates the potential for hair cell damage. We establish the log-normal nature of accumulated damage, and exploit this feature to develop quantifiable measurables that can be subjected to statistical analysis of probability, mean, variance, and standard deviation. Theoretical results indicate mechanisms for probability risk assessment, measuring membrane vulnerability through spatial mapping, and sensitivity dynamics to exposure to toxic frequencies. The results emphasize the importance of considering cumulative damage and spatial variability in sensitivity when assessing the risk of hearing loss due to sound exposure. Further refinement of the model to account for individual variations in basilar membrane structure and noise exposure patterns is recommended.

**Keywords**: Frequency, cumulative distribution frequency, basilar membrane, noise exposure.
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**Corresponding author:** Farai Nyabadza, Department of Applied Mathematics, University of Johannesburg, Johannesburg, South Africa, Tel.: +27 11 559 2305, e-mail: *fnyabadza@uj.ac.za*
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## 1 Introduction

The human auditory system is a remarkable feat of biological engineering, with the basilar membrane in the cochlea serving as a critical component in the detection and differentiation of sound frequencies (Kalchev, 2024; Peterson et al., 2024; Polak et al., 2022). This membrane, an elongated and flexible structure, exhibits a complex spatial arrangement that allows it to respond to a wide range of sound frequencies. Understanding how the basilar membrane achieves this frequency discrimination is fundamental for exploring the latent dynamics of auditory perception and has implications for both auditory research and clinical diagnostics.

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The basilar membrane is not a uniform entity but is instead characterized by a gradient of sensitivity along its length. This gradient is organized tonotopically, meaning that different regions of the basilar membrane are specialized for different frequency ranges (see also Brugge & Howard (2002)). High-frequency sounds are primarily detected at the base of the basilar membrane, while low-frequency sounds are processed towards the apex. This spatial arrangement is crucial for the auditory system's ability to differentiate between various pitches and tones in complex sound environments. In Figure 1 that follows extracted from National Library of Medicine (1991), we show the physical structural nature of the cochlea around the basilar membrane:

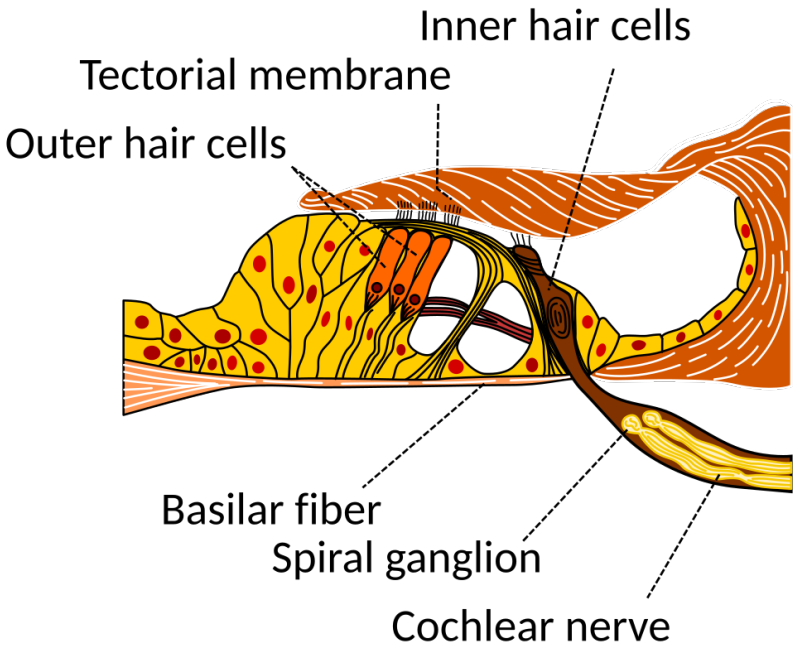


**Figure 1:** The hair cells support membrane in the cochlea as illustrated in National Library of Medicine (1991)

Hearing loss is a significant public health concern, often resulting from damage to cochlear hair cells. These cells are responsible for translating sound vibrations into neural signals, and their impairment can lead to various degrees of hearing loss. A crucial aspect of this process is the role of the basilar membrane in the cochlea, which is responsible for filtering sound frequencies. The basilar membrane's response to sound, particularly loud sounds, is vital in understanding how hair cells are damaged and subsequently lead to hearing loss. To model the basilar membrane's response to sound frequencies, it is essential to consider both the physical structure of the membrane and its functional response characteristics. The basilar membrane's ability to respond to different frequencies can be likened to a series of filters, each tuned to a specific range of frequencies (Moini & Piran, 2020). This frequency-tuned filtering is achieved through the variation in the mechanical properties of the membrane along its length. For high frequencies, the membrane is stiffer and less flexible, while for low frequencies, it becomes more flexible and responsive (Russell et al., 2007, 2024).

In the basilar membrane high frequencies are detected at its base, while low frequencies are processed at the apex. This tonotopic organization allows for precise frequency discrimination. However, exposure to loud sounds can disturb the basilar membrane's normal functioning, causing mechanical stress that adversely affects hair cells (Waqas et al., 2018). In mathematical terms, the sensitivity of the basilar membrane to different frequencies can be modeled using a sensitivity function that varies along the length of the membrane. This function needs to account for the non-uniform distribution of sensitivity and the gradual shift in frequency tuning from the

base to the apex (Robles & Ruggero, 2001; Chatterjee & Zwislocki, 1997). The mathematical formulation of this sensitivity function involves defining a position-dependent scaling factor that reflects the varying sensitivity at different points along the basilar membrane. Additionally, a function describing the sensitivity to frequency deviations from a baseline frequency is included to capture the response characteristics accurately.

This study's primary contribution is the development of a quantifiable mathematical model that clarifies the effects of loud sounds on the basilar membrane and and the ultimate resultant hearing loss, assuming that the cumulative damage in each segment follows a log-normal distribution (Dunn & Shultis, 2023). The model integrates mechanical dynamics, statistical approaches and sensitivity functions to develop a mathematical formalization of the membrane's response to various sound frequencies. By quantifying how different frequencies impact the membrane's sensitivity and contribute to hair cell impairment, the study provides a detailed understanding of sound-induced disturbances. The model effectively demonstrates how exposure to high-intensity sounds can lead to hearing loss by affecting the mechanical and biological processes of the basilar membrane. This comprehensive approach gives an understanding of the mechanisms behind hearing damage and informs strategies for sound exposure management.

The paper is structured as follows: The introduction to this study is in Section 1 followed by the model formulation in Section 2. In Section 3 we state our results as propositions, and discuss them in Section 4. The conclusion to this work is found in Section 5.

## 2 The model

We make use of indicator functions, topological, and statistical concepts, and ordinary differential equations to describe the dynamics in the basilar membrane.

### 2.1 Model formulation

To formulate the model we begin by defining the sensitivity function $f(x,\nu)$, where $x$ denotes the position along the basilar membrane, and $\nu$ represents the sound frequency. This function is designed to capture how the sensitivity of the $i^{th}$ segment of the basilar membrane at position $x$ changes in response to the frequency $\nu$. We set $g(\nu-\beta(x))$ to be the function that describes the sensitivity to frequency deviations from a baseline frequency $\beta(x)$. The function $g(\cdot)$ peaks when $\nu$ is close to the characteristic frequency, reflecting where the membrane is most responsive. Considering that the sensitivity response depends on the magnitude of the sensitivity of a system to deviations in frequency from a baseline frequency, we propose a piecewise function to capture variations in sensitivity, depending on how large the deviation is. This is reasonable because in biological systems such as the basilar membrane's response to sound frequencies, small deviations might be handled differently compared to large deviations, which might trigger protective mechanisms or saturate the system's response. Hence, we have that for thresholds $\delta_1$ and $\delta_2$, in its simplest form:

$$g(\nu-\beta(x)) = \begin{cases} k_1(\nu-\beta(x)), & \text{if } \nu-\beta(x) \leq \delta_1, \\ k_2(\nu-\beta(x)), & \text{if } \delta_1 < \nu-\beta(x) \leq \delta_2. \end{cases} \tag{1}$$

The slopes $k_1$ and $k_2$ are fractional weights for different sensitivity levels and the thresholds $\delta_1$ and $\delta_2$ are critical in defining how the system responds to frequency deviations. These thresholds ($\delta_1$ and $\delta_2$) can be interpreted as threshold below which the system's sensitivity is moderate and a point beyond which the system's response is sensitive respectively. A direct consequence of the physical and mechanical properties of the cochlea, particularly the basilar membrane is a gradual decline in frequency sensitivity as $x$ increases along the basilar membrane. Hence, we propose a characteristic function of the form $\beta(x) = \frac{\beta_0}{1+\kappa x}$, where $\beta_0$ is the characteristic frequency at base,

and $\kappa$ controls the slope of decrease. In this respect, the sensitivity function can be expressed as:

$$f(x,\nu) = \alpha(x) \cdot g(\nu - \beta(x)), \tag{2}$$

where the indicator function

$$\alpha(x) = \begin{cases} 1 & \text{if } \nu > \beta(x) \\ 0 & \text{if } \nu \leq \beta(x), \end{cases}$$

represents a position-dependent scaling factor that reflects the segment's sensitivity. It is an indicator function that adjusts sensitivity along the basilar membrane, accounting for the natural variation in sensitivity to different frequencies across the membrane's length. It reflects the membrane's higher sensitivity to low frequencies at the apex and high frequencies at the base. The overall sensitivity of the basilar membrane to a given frequency $\nu$ can be obtained by integrating the sensitivity function over the length of the membrane. This is expressed as:

$$S(\nu) = \int_0^L f(x,\nu)\, dx, \tag{3}$$

where $L$ represents the total length of the basilar membrane. This integral provides a measure of how the entire basilar membrane responds to the frequency $\nu$, capturing the collective sensitivity of the membrane. To account for dynamic changes in sensitivity occurring with varying sound frequencies over time, the model includes an ordinary differential equation that describes how the sensitivity $S(t)$ evolves given by:

$$\frac{dS(t)}{dt} = \gamma \cdot \left(f(x,\nu) - S(t)\right), \tag{4}$$

where $\gamma$ is a rate constant that reflects the speed at which the basilar membrane adjusts its sensitivity to changes in position $x$. The properties of positivity and boundedness of solutions are trivial and are omitted in this discussion. To represent the basilar membrane's response to a spectrum of frequencies, a frequency distribution profile is introduced. This profile, denoted as $D(\nu)$, is given by:

$$D(\nu) = \int_0^L f(x,\nu) \cdot h(x)\, dx, \tag{5}$$

where $h(x)$ represents the distribution of frequencies at position $x$ along the membrane and can be an exponential decay or Gaussian function to reflect the spatial frequency mapping in the cochlea.

We consider the basilar membrane as a continuous one-dimensional topological space $B$ that can be partitioned into measurable segments $b_1, b_2, \ldots, b_k$. Each segment $b_k$ represents a specific location along the basilar membrane where sound frequencies are mapped. To model how different frequencies are allocated to each of the various segments along the basilar membrane, let $\{\beta_1, \beta_2, \ldots, \beta_k\}$ denote a set of normal (baseline) frequencies and $\{s_1, s_2, \ldots, s_k\}$ denote a set of frequencies from a source which can cause damage to the hair cells. Setting

$$\{t_1, t_2, \ldots, t_k\} = \{s_1, s_2, \ldots, s_k\} - \{\beta_1, \beta_2, \ldots, \beta_k\}, \tag{6}$$

then each segment $b_k$ of the basilar membrane is characterized by two sensitivity functions:

- $s_k(\beta_i)$: The sensitivity of segment $b_k$ to normal (baseline) frequency $\beta_i$, reflecting how well the segment responds to this frequency.
- $d_k(t_j)$: The sensitivity of segment $b_k$ to toxic frequency $t_j$, representing the potential damage inflicted by this frequency.

To ensure that the distribution of frequencies does not exceed a predefined damage threshold for each segment, we let $\phi_k$ be the damage threshold for segment $b_k$, then the allocation of toxic frequencies is valid if:

$$\sum_{t_j \in T} d_k(t_j) < \phi_k \text{ for all } k \tag{7}$$

where $T$ is the set of toxic frequencies. This constraint ensures that the cumulative damage caused by toxic frequencies remains below the threshold, protecting the hair cells from excessive harm. We determine the cumulative damage for each segment and compare it against a defined damage threshold $\phi_k$. If the accumulated damage $D_k(t)$ for any segment $b_k$ exceeds $\phi_k$, we consider that segment as experiencing hearing loss.

# 3 Quantifying toxicity exposure, sensitivity, and damage

In this section, we derive and formalize key results related to the accumulation of damage on the basilar membrane due to toxic frequency exposure. We present each result as a proposition, followed by a proof to rigorously establish the claims.

## 3.1 Log-normal distribution of accumulated damage

In Proposition 1 we establish that the accumulated damage on the basilar membrane over multiple exposure cycles follows a log-normal distribution (see also Limpert et al. (2001)).

**Proposition 1.** *The accumulated damage on the basilar membrane over various exposure cycles follows a log-normal distribution.*

*Proof.* Within a segment $b_k$ at any time $t > 0$, we consider exposure cycles $E_i$ for $i = 1, 2, \ldots, n$ defined as the susceptibility of the segment to potential damage due to toxic frequencies $t_i$ during the $i^{th}$ exposure cycle. Each cycle $E_i$ corresponds to the vulnerability of the segmented basilar membrane to a specific toxic sound frequency $t_i$, with the damage level $D_i$ reflecting the cumulative effect of these exposures. We further assume that the time interval for each exposure cycle is $\tau$, such that the total time $t = n\tau$ a relatively short interval, such that the decline in sensitivity of the segment over one cycle is minimal. The cumulative damage $D_n$ after $n$ cycles is assumed to be proportional to the intensity of the exposure, given by the sum of the differences $D_i - D_{i-1}$ between consecutive cycles. This can be modeled using a function $\psi(D_{i-1})$, which represents the progressive sensitivity decline due to previous damage. The damage in the $i^{th}$ cycle is then given by:

$$D_i - D_{i-1} = E_i \cdot \psi(D_{i-1}),$$

where $E_i$ is the exposure during the $i^{th}$ cycle and $\psi(D_{i-1})$ is a function representing sensitivity decline, and assuming $\psi(D)$ is linear for simplicity, we observe that the accumulated damage $D_n$ over $n$ cycles follows a log-normal distribution and it is given by:

$$\sum_{i=1}^{n} E_i = \sum_{i=1}^{n} \frac{D_i - D_{i-1}}{\psi(D_{i-1})} = \int_{D_0}^{D_n} \frac{dD}{\psi(D)}.$$

Assuming $\psi(D) = \delta D$ (linear), this simplifies to:

$$\sum_{i=1}^{n} E_i = \frac{1}{\delta} \int_{D_0}^{D_n} \frac{dD}{D} = \frac{1}{\delta} \ln\left(\frac{D_n}{D_0}\right). \tag{8}$$

This shows that:

$$\ln\left(\frac{D_n}{D_0}\right) \sim N(\mu, \sigma^2),$$

where $\mu$ and $\sigma^2$ are the mean and variance of the logarithm of the damage. Therefore, $D_n$ follows a log-normal distribution. □

Equation 8 shows that the cumulative exposure (and thus the total damage) is proportional to the natural logarithm of the ratio of the final damage $D_n$ log-normally distributed. This means that while the logarithm of damage $\ln(D_n)$ would be normally distributed, the actual damage $D_n$ itself follows a log-normal distribution. This distribution reflects the idea that damage accumulates in a multiplicative way over time since, small changes in each cycle can lead to larger cumulative effects, which leads to a log-normal distribution.

### 3.2 Probability of exceeding damage threshold

To investigate exposure to toxic frequencies over a time period $t$, we propose iteration of the accumulated damage $\Delta t$ at each time step $\tau$, updating the accumulated damage for each segment $k$ using the equation

$$D_k(t) = D_k(t - \Delta t) + d_k(t_j) \cdot \Delta t. \tag{9}$$

Since the accumulated damage $D_k(t)$ follows a log-normal distribution, then $\ln(D_k(t))$ is normally distributed, that is:

$$\ln(D_k(t)) \sim N(\mu_k, \sigma_k^2),$$

where $\mu_k$ represents the mean and $\sigma_k$ the standard deviation of the logarithm of the damage for segment $k$, that can be estimated using historical damage data. Given a set of $n$ damage observations in segment $k$ containing $D_{k,1}, D_{k,2}, \ldots, D_{k,n}$, we can estimate the mean $\mu_k$ and variance $\sigma_k^2$ as:

$$\hat{\mu}_k = \frac{1}{n}\sum_{i=1}^{n} \ln(D_{k,i}), \quad \hat{\sigma}_k^2 = \frac{1}{n-1}\sum_{i=1}^{n} \left(\ln(D_{k,i}) - \hat{\mu}_k\right)^2. \tag{10}$$

We use the cumulative distribution function (CDF) of the log-normal distribution to calculate the probability that the accumulated damage is less than or equal to a given threshold $\phi_k$, is used. The probability is given by:

$$F(\phi_k) = \Phi\left(\frac{\ln(\phi_k) - \hat{\mu}_k}{\hat{\sigma}_k}\right), \tag{11}$$

where $\Phi$ denotes the CDF of the standard normal distribution. This result provides a rigorous statistical framework for predicting, assessing, and managing the risk of damage to the basilar membrane. We quantify the probability that the accumulated damage in a specific segment of the basilar membrane exceeds a given threshold, as detailed in the following proposition.

**Proposition 2.** *The probability that the accumulated damage $D_k(t)$ for segment $k$ of the basilar membrane exceeds a given threshold $\phi_k$ is given by:*

$$P(D_k(t) > \phi_k) = 1 - \Phi\left(\frac{\ln(\phi_k) - \hat{\mu}_k}{\hat{\sigma}_k}\right), \tag{12}$$

*where $\Phi$ CDF of the standard normal distribution, and $\hat{\mu}_k$ and $\hat{\sigma}_k$ are the estimated mean and standard deviation of $\ln(D_k(t))$.*

*Proof.* Since $D_k(t)$ follows a log-normal distribution, $\ln(D_k(t))$ is normally distributed:

$$\ln(D_k(t)) \sim N(\hat{\mu}_k, \hat{\sigma}_k^2).$$

The probability that $D_k(t)$ exceeds $\phi_k$ is the complement of the CDF of $\ln(D_k(t))$:

$$P(D_k(t) > \phi_k) = 1 - P(\ln(D_k(t)) \leq \ln(\phi_k)) = 1 - \Phi\left(\frac{\ln(\phi_k) - \hat{\mu}_k}{\hat{\sigma}_k}\right).$$

□

This probability highlights the likelihood of the damage surpassing the threshold, which is essential for assessing the potential risk to the segment $k$ of the basilar membrane.

### 3.3 Threshold optimization

We derive a method to determine the safe threshold $\phi_k$ for accumulated damage in a segment of the basilar membrane, ensuring that the probability of exceeding this threshold remains below a specified risk level. To establish a safe threshold $\phi_k$ such that the probability of exceeding it ($P_{<\phi_k}$) remains below a specified risk level one can solve for $\phi_k$ using the inverse CDF of the standard normal distribution, $\Phi^{-1}$.

**Proposition 3.** *The threshold $\phi_k$ such that the probability of the accumulated damage $D_k(t)$ not exceeding $\phi_k$ ( $P_{<\phi_k}$), is below a specified risk level is given by:*

$$\phi_k = \exp\left(\hat{\mu}_k + \hat{\sigma}_k \cdot \Phi^{-1}(1 - P_{<\phi_k})\right),$$

*where $\Phi^{-1}$ is the inverse CDF of the standard normal distribution.*

*Proof.* Given the desired probability $P_{<\phi_k}$, we solve for $\phi_k$ such that:

$$P(D_k(t) \leq \phi_k) = P_{<\phi_k}.$$

Using the CDF of the log-normal distribution:

$$\Phi\left(\frac{\ln(\phi_k) - \hat{\mu}_k}{\hat{\sigma}_k}\right) = P_{<\phi_k}.$$

Solving for $\phi_k$:

$$\begin{aligned} \tfrac{\ln(\phi_k)-\hat{\mu}_k}{\hat{\sigma}_k} &= \Phi^{-1}(P_{<\phi_k}), \\ \ln(\phi_k) &= \hat{\mu}_k + \hat{\sigma}_k \cdot \Phi^{-1}(P_{<\phi_k}), \quad \text{such that} \\ \phi_k &= \exp\left(\hat{\mu}_k + \hat{\sigma}_k \cdot \Phi^{-1}(P_{<\phi_k})\right). \end{aligned} \tag{13}$$

This provides the safe threshold $\phi_k$ for the damage level in segment $k$. □

To incorporate the CDF into the ordinary differential equation (4), the sensitivity function $f(x, \nu)$ is adjusted to reflect the probability of accumulated damage. The modified equation becomes:

$$\frac{dS(t)}{dt} = \gamma \cdot (F(\phi_k) \cdot f(x, \nu) - S(t)), \tag{14}$$

where $F(\phi_k)$ represents the CDF of the accumulated damage, and $f(x, \nu)$ is the sensitivity function dependent on position $x$ and frequency $\nu$. The term $F(\phi_k)$ adjusts the sensitivity based on the probability that the accumulated damage is below a threshold $\phi_k$. A higher $F(\phi_k)$, indicating lower damage, results in a greater contribution of $f(x, \nu)$ to the time-dependent sensitivity $S(t)$. Conversely, a lower $F(\phi_k)$, indicating higher damage, reduces this contribution. Over time, as the basilar membrane is exposed to potentially damaging frequencies, $F(\phi_k)$ may decrease, leading to a corresponding reduction in $S(t)$. This dynamic interaction implies that $S(t)$, the sensitivity of the basilar membrane over time, is influenced not only by the frequency $\nu$ but also by the cumulative probability of damage, modulating the sensitivity in response to ongoing exposure.

To model how the local sensitivity function $f(x, \nu)$ is affected by damage $D(x, t)$ at position $x$, we introduce a damage-dependent local sensitivity function:

$$f(x, \nu, D(x, t)) = f_0(x, \nu) \cdot g(D(x, t)), \tag{15}$$

where $f_0(x, \nu)$ represents the baseline local sensitivity before any damage, and $g(D(x, t))$ is a function that reduces sensitivity based on the level of damage $D(x, t)$ and we assume that $g(D(x, t))$ decreases as $D(x, t)$ increases. Hence, $g(D(x, t))$ could be modeled as an exponentially decreasing function:

$$g(D(x, t)) = e^{-\beta D(x,t)}, \tag{16}$$

where $\beta$ is a constant controlling the sensitivity reduction rate.

### 3.4 The Damage-Weighted Sensitivity Function (DWSF)

Consequent of Equation 16 , we have that $S(\nu)$ is modified to incorporate the impact of accumulated damage, providing a more comprehensive view of how sensitivity is affected over time. This damage $S_d(\nu)$ can be expressed as:

$$S_d(\nu) = \int_0^L f_0(x,\nu) \cdot e^{-\beta D(x,t)}\, dx. \tag{17}$$

The damage $D(x,t)$ at any point $x$ is a random variable and can be described by the CDF $F(\phi_k)$. Assuming the damage at different points $x$ follows a similar distribution, the sensitivity function $S_d(\nu)$ can be written as:

$$S_d(\nu) = \int_0^L f_0(x,\nu) \cdot \left[\int_0^{\phi_k} e^{-\beta\phi_k}\, dF(\phi_k)\right] dx, \tag{18}$$

where $\phi_k$ represents the threshold of interest, and the inner integral computes the expected reduction in sensitivity due to the damage distribution. The inner integral represents the expected value of the exponential function considering the damage distribution. The outer integral then averages this expected value over the length of the membrane, weighted by the sensitivity function $f_0(x,\nu)$. If the CDF $F(\phi_k)$ is derived from a log-normal distribution, the inner integral can be expressed using the properties of the log-normal distribution:

$$E[e^{-\beta D(x,t)}] = e^{-\beta\hat{\mu}_k + \frac{\beta^2\hat{\sigma}_k^2}{2}} \cdot \Phi\left(\frac{\ln(\phi_k) - (\hat{\mu}_k - \frac{\beta\hat{\sigma}_k^2}{2})}{\hat{\sigma}_k}\right). \tag{19}$$

Thus, the sensitivity that incorporates damage $S_d(\nu)$ becomes:

$$S_d(\nu) = \int_0^L f_0(x,\nu) \cdot e^{-\beta\hat{\mu}_k + \frac{\beta^2\hat{\sigma}_k^2}{2}} \cdot \Phi_k\left(\frac{\ln(\phi_k) - (\hat{\mu}_k - \frac{\beta\hat{\sigma}_k^2}{2})}{\hat{\sigma}_k}\right) dx. \tag{20}$$

This function models the sensitivity of the basilar membrane at a specific frequency $\nu$ by incorporating the effects of accumulated damage over time. It adjusts the baseline sensitivity, $f_0(x,\nu)$, by factoring in the reduction in sensitivity due to damage, which is influenced by the mean and variance of the logarithm of the damage distribution. The function also incorporates the probability that the damage remains below a critical threshold $\phi_k$, using the cumulative distribution function of the standard normal distribution. To clarify the expectation $E[e^{-\beta D(x,t)}]$ in the context of the log-normal distribution, we acknowledge that since $D(x,t)$ is log-normally distributed, its logarithm follows a normal distribution. The expectation of $e^{-\beta D(x,t)}$ can be derived using the moment-generating function of the normal distribution, which leads to the result:

$$E[e^{-\beta D(x,t)}] = \exp\left(\mu\beta + \frac{1}{2}\sigma^2\beta^2\right).$$

This formula quantifies how damage at each segment and time contributes to the DWSF, considering the varying levels of accumulated damage across cycles. By integrating these effects over the length of the basilar membrane, the function provides a comprehensive measure of how sensitivity is altered by both local damage at specific points and the overall damage across the membrane.

## 4 Discussion and conclusion

This model explores the sensitivity of the basilar membrane to sound frequencies, focusing on how different segments respond to varying frequencies and the potential for damage or hearing

loss. It aims to develop a mathematical framework that captures the dynamic behavior of the membrane in response to both normal and harmful frequencies, taking into account the membrane's spatial sensitivity distribution. By understanding how different segments respond to these frequencies, the model helps predict and prevent damage to hair cells, which is crucial for assessing and mitigating hearing loss risk. The cochlea's basilar membrane exhibits a well-known tonotopic organization, where different positions along the membrane are tuned to specific frequencies. This tuning results from the mechanical properties of the membrane, which vary along its length. Understanding how different segments of the basilar membrane respond to normal and harmful frequencies is critical, as it can help predict and prevent damage to hair cells, which is a primary cause of hearing loss.

The damage accumulation is described by (9) and to ensure that $D_k(t)$ does not exceed a predefined damage threshold $\phi_k$, it is crucial to adjust $d_k(t_j)$ dynamically based on real-time sensitivity $S(t)$. If $S(t)$ indicates that a basilar membrane segment is highly sensitive, reducing $d_k(t_j)$ can mitigate the potential damage from continued exposure. In practical terms, this approach involves calculating $S(t)$ using the differential equation, evaluating potential damage $d_k(t_j)$ based on the sensitivity function $f(x, \nu)$ and historical damage data, and then adjusting the exposure $d_k(t_j)$ in real-time. This ensures that cumulative damage $D_k(t)$ remains within safe limits, thus protecting the basilar membrane from excessive stress and maintaining its health. By integrating these elements we can develop an effective strategy for preventing overstress and minimizing damage to the basilar membrane.

Computing $D(\nu)$ aids in the assessment of exposure by evaluating how different frequencies contribute to the overall exposure and potential risk of damage to the basilar membrane. By analyzing $D(\nu)$ for various frequencies, one can understand which frequencies have the greatest impact on the risk of damage. Additionally, $D(\nu)$ is instrumental in risk analysis, as it helps in understanding how exposure variability and sensitivity combine to influence the likelihood of damage. Higher $D(\nu)$ values could signal a greater potential for damage at specific frequencies, providing critical information for evaluating risk. Moreover, $D(\nu)$ supports optimization efforts by allowing adjustments to parameters which can be tuned to minimize the risk of damage. This makes $D(\nu)$ a practical tool for optimizing exposure settings based on its calculated values. Hence, $D(\nu)$ serves as a crucial metric for evaluating how different frequencies interact with the sensitivity of the basilar membrane, enabling more informed decisions in noise exposure management and hearing protection strategies.

Identifying that the accumulated damage follows a log-normal distribution is crucial for assessing and managing the risk of damage to the basilar membrane by providing a statistical framework based on accumulated damage data. Modeling the accumulated damage $D_k(t)$ as a log-normal distribution allows for a probabilistic understanding of how damage accumulates over time, reflecting the natural variability in biological systems. The estimation of key parameters—mean ($\mu_k$) and standard deviation ($\sigma_k$)— (possibly using historical data) is crucial for highlighting expected damage levels and variability across different segments of the membrane. By providing a mechanism to calculate the probability that the accumulated damage exceeds a given threshold $\phi_k$, the model helps identify segments at risk, guiding preventive measures and safety protocols.

Analysis of the toxic frequency $t_j = s_j - \beta_j$ and its interaction with the sensitivity function $g(\nu - \beta(x))$ clarifies how frequency deviations impact the basilar membrane's sensitivity, which is key to auditory perception. The function $g(\nu - \beta(x))$ gauges how sensitivity changes as frequency $\nu$ deviates from the characteristic frequency $\beta(x)$ at position $x$, demonstrating dynamics underlying cochlear processing. The piecewise sensitivity function, with thresholds $\delta_1$ and $\delta_2$, highlights that small deviations $|\nu - \beta(x)| \leq \delta_1$ lead to moderate sensitivity, while larger deviations $\delta_1 < |\nu - \beta(x)| \leq \delta_2$ can provoke heightened sensitivity or protective mechanisms. These thresholds are critical in understanding the cochlea's transition between sensitivity levels. The nonlinear response of the basilar membrane, as demonstrated in Rhode (1971, 1978); Sellick et al. (1982); Robles (1986), show that the cochlea compresses a wide range of acoustic signals

into a narrower range detectable by hair cells. This non-linearity results in low-level distortion products (DPs), which have been linked to hearing thresholds (Kemp, 1979; Kim et al., 1980; Fahey & Allen, 1985; Gaskill & Brown, 1990; Nelson & Kimberley, 1992; Probst et al., 1991). Our study builds on this understanding by integrating the concept of nonlinear compression into our sensitivity function $g(\nu - \beta(x))$. This approach helps explain how frequency deviations affect cochlear sensitivity and guides the development of targeted hearing protection and damage prevention strategies, aligning with biological observations that cochlear sensitivity varies with frequency deviations.

The model incorporates the cumulative damage distribution into the sensitivity function of the basilar membrane. By representing damage $D(x,t)$ at each position $x$ as a random variable and utilizing its cumulative distribution function $F(\phi_k)$, we can more accurately describe how damage impacts sensitivity. The sensitivity function $S(\nu)$ is thus formulated as an integral that accounts for the expected reduction in sensitivity due to this damage distribution. Specifically, since the CDF $F(\phi_k)$ follows a log-normal distribution, the expected value of the damage's impact can be computed analytically. The formula for the expectation of $e^{-\beta D(x,t)}$ under a log-normal distribution is utilized to integrate the damage effect into the sensitivity function. This allows for a realistic assessment of how varying levels of damage, characterized by the distribution parameters, affect the sensitivity of the basilar membrane across different frequencies. By integrating over the length of the membrane, the final sensitivity function captures the collective impact of damage distribution on sensitivity, providing a comprehensive model for understanding how accumulated damage alters auditory sensitivity over time.

We highlight the empirical findings from Gajewski et al. (2004), which demonstrate that hearing thresholds often exhibit heavy-tailed and peaked distributions under conditions of bacterial infections and inner ear surgeries. These statistical properties align closely with our finding (Proposition 1) that the accumulated damage on the basilar membrane follows a log-normal distribution. Additionally, our model's assumption of multiplicative damage accumulation over exposure cycles resonates with the observed complex changes in hearing thresholds in their experiments. The use of a log-normal framework in their study further validates the relevance and applicability of our theoretical constructs to real-world biological systems. These parallels suggest that our model's predictions are consistent with known empirical phenomena, reinforcing its credibility and relevance. In Robles & Ruggero (2001); Henderson et al. (1993), there are detailed insights into cochlear mechanics and serve as a foundation for validating the link between structural damage and sensitivity reduction.

## 5 Conclusion

The model effectively captures the spatial distribution of sensitivity across the basilar membrane, which shows how different segments respond to various sound frequencies. This mapping is crucial for understanding the potential for frequency-specific damage, helping to identify regions most susceptible to harm. By using a log-normal distribution to represent damage accumulation, it aligns with biological evidence and enhances predictions of impairment based on sound exposure. However, the assumptions and reliance on estimated parameters may limit accuracy in certain cases. Future work could refine these assumptions, incorporate more empirical data, and consider additional factors like genetic predispositions, ultimately improving the model's precision and applicability in understanding and preventing cochlear damage.

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