MODEL-BASED PREDICTIONS OF INTENSITY DISCRIMINATION FOR NORMAL- AND IMPAIRED-HEARING LISTENERS

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ABSTRACT

Interpretation of psychophysical data from impaired-hearing individuals on intensity discrimination tasks has been confounded by the fact that some impaired individuals’ performance is near-normal in quiet, whereas for others, the difference limen is elevated. This paper presents a theoretical analysis of the effects of cochlear impairments on intensity discrimination using a combination of signal detection theory and a computational auditory model. Not only were we able to replicate the trends observed in experimental data, but, by using a model, we could also establish a link between the psychophysical predictions and the underlying physiology. In doing so, we are able to support the hypothesis that the observed behavior is due to the spread of excitation.

1. INTRODUCTION

It has often been difficult to link psychophysical performance to underlying physiological signal processing mechanisms. Specifically, conflicting data has made it difficult to conclusively state the effect that hearing loss has on intensity discrimination. Based on evidence from normal-hearing listeners suggesting that the spread of excitation to higher-frequency regions was important for intensity discrimination (e.g., [13]), Florentine and colleagues proposed analyzing the impaired-hearing data according to the configuration of the impairment rather than analyzing the data collectively [3].

Schroder and colleagues extended this work by investigating the effects of various impairments on the near-miss to Weber’s Law observed in normal-hearing subjects [11]. It has been hypothesized that the near-normal performance in some impaired cases may be due to either greater intensity resolution (resulting from loudness recruitment) or the normal spread of excitation. To test this hypothesis, Schroder and colleagues measured difference limens (DLs) for normal and impaired subjects in quiet and high-pass noise conditions [11]. They observed that impaired individuals with near-normal DLs in quiet exhibited reduced discrimination ability in the noisy condition, as did the normal-hearing subjects. In contrast, those listeners whose DLs were elevated in the quiet condition were affected very little by the high-pass noise.

The psychophysical data collected by Schroder and colleagues supported their hypothesis that near-normal impaired-hearing performance was due to the spread of excitation rather than loudness recruitment. However, there was no mechanism by which to conclusively prove this hypothesis using human psychophysical procedures alone. In this paper, we investigate the effects of impairment on intensity discrimination theoretically, replicating the aforementioned experiment using a computational auditory model in combination with signal detection theory. Similar approaches have previously been used to generate theoretical predictions of psychophysical data (e.g., [12, 5, 6, 7]). Using this method, we can not only generate predictions of psychophysical data, but can also establish a connection between those results and the underlying physiological responses predicted by the model.

2. MODEL-GENERATED NEURAL RESPONSES

Several computational models of peripheral auditory processing have been developed to simulate neural response patterns (e.g., [1, 10, 15]). We have chosen Patterson and colleagues’ physiologically-based Auditory Image Model (AIM) [10]. Predictions of neural activity patterns are generated first by dividing the auditory frequency spectrum into discrete channels using a transmission-line filter bank and then using the output of each channel to drive an inner hair cell, based on the Meddis hair cell model [8]. Each channel corresponds to an auditory filter whose center frequency (CF) and bandwidth are determined by the Equivalent Rectangular Bandwidth scale [4]. The model was configured to have 102 channels with CFs ranging from 100 to 10000 Hz. The model output provided a representation of the neural activity pattern in terms of a set of time-varying average neural firing rates, one per channel.

We modified AIM, a deterministic model, to mimic the stochastic behavior of the auditory system by appending a non-stationary Poisson process. This approach has been used frequently in the past (e.g., [1]) to incorporate sources of neural or internal noise into a model and has been successfully integrated with AIM to generate predictions of frequency difference limens [7].

3. IMPAIRING THE MODEL

To theoretically study the effects of auditory impairment on intensity discrimination, a model of impaired auditory processing must be implemented, analyzed, and used to generate performance predictions that can be compared to results predicted by a normal-hearing model. Because the differences between the normal and impaired models can be precisely controlled, physiological impairments were simulated by modifying AIM. For the first impairment, outer hair cell (OHC) feedback was eliminated by setting the feedback gain parameter of the OHC circuit to zero. In the second condition, a 20-dB flat hearing loss was simulated by shifting the thresholds of the auditory fibers and compressing the dynamic range. The third condition simulated a high frequency loss by linearly suppressing the response in channels corresponding to fibers with best frequencies (BFs) between 1000 and 3000 Hz such that the threshold at 3000 Hz was shifted 40 dB. Channels below 1000 Hz were allowed to respond normally; channels above 3000 Hz had a flat 40 dB loss induced.
The impairments were verified by comparing rate-intensity, neural tuning, response area, and absolute threshold curves for the normal and impaired models. The rate-intensity curves (Figure 1a) indicate a shift in threshold (~20 dB) for two impaired conditions. It is expected that the threshold of the high frequency loss condition will also shift as the test frequency increases. The steeper slopes for two of the impaired conditions also illustrate the phenomenon of loudness recruitment [9]. The neural tuning curves (Figure 1b) reveal that the removal of OHCs eliminates the sharply tuned tip observed in the normal response; however, away from this tip, the response of the fiber appears to be near-normal. In contrast, for a flat loss, the entire tuning curve is shifted, indicating that the fiber’s response to frequencies away from its BF are also significantly affected. The high frequency loss appears to have no effect on the tuning curve because the test fiber lies in the unimpaired region. However, if a fiber from the impaired region were selected, the results would show a shift similar to that for a flat loss. Two important features of the response area curves (Figure 1c) differ across conditions: 1) the extent of fiber response area and 2) the response magnitude. When OHC feedback is removed, the response area is normal, although the magnitude is reduced in the region near the tone frequency. For a flat loss, the magnitude response is suppressed in all fibers and the spread is reduced by 2 to 3 kHz. The high frequency loss condition, on the other hand, has a near-normal magnitude response near the test frequency which decays rapidly to the spontaneous rate between 2 and 3 kHz. Finally, Figure 1d plots the absolute threshold curves. These curves more clearly illustrate the effect of the high frequency loss in comparison to the other two impaired conditions. The similarity between audiograms for the OHC-loss and flat loss conditions will minimize this confounding factor when interpreting results.

4. CALCULATION OF THE DIFFERENCE LIMEN

Theoretical predictions of the intensity difference limen were generated using two different detectors, each representing one possible mechanism by which the auditory system could use the information encoded in neural responses to differentiate between two stimuli. The same approach has been used previously to generate predictions for frequency discrimination [7]. One detector, referred to as the optimal detector, utilized the entire neural response predicted by the computational model. The form of the detector was derived using signal detection theory for a binary hypothesis test. The density functions under each hypothesis were determined and used to formulate the log likelihood ratio:

\[
\ln \Lambda = \sum_{n=1}^{N} \frac{\ln r_n(x_n, I + \Delta I)}{\ln r_n(x_n, I)} \frac{n_j}{n_0} \leq \beta, \tag{1}
\]

where \(r_n(x_n, I)\) is the time-varying firing rate in the \(i^{th}\) filter generated in response to a tone with intensity \(I\) at \(x_n\), the time of the \(n^{th}\) spike and is proportional to the probability density function. This log likelihood ratio is an expression for the optimal detector for a single fiber in the \(i^{th}\) channel.

The second detector, based only on the total number of spikes in each channel, removed temporal information by integrating over the neural firing rate and used this average rate to drive a stationary Poisson process. Because the probability of a spike in this case is uniform across time, the log likelihood ratio in Equation 1 can be simplified algebraically to

\[
\ln \Lambda = N \frac{n_j}{n_0} \leq \beta, \tag{2}
\]

where \(N\) is the total number of spikes observed in a given fiber. Assuming channel independence, the overall detectability, \(\delta'\), for both detectors is equal to the square root of the sum of the squares of the detectability computed for each individual fiber. An adaptive procedure was used to determine \(\Delta I_{IN}\), defined as the difference in intensity required to obtain \(\delta' = 1\).

5. RESULTS AND DISCUSSION

Model-based predictions for intensity discrimination were determined as a function of both stimulus frequency and intensity in quiet and simulated high-pass noise conditions. High-pass noise was modeled by eliminating the information encoded in channels whose CFs were greater than 1.5 times the frequency of the test tone, \(f_0\). The cutoff frequency was selected to closely replicate the experimental paradigm used by Schroder and colleagues in their human experiments [11]. Eliminating channels produces essentially the same effect as adding high-pass noise with a lower-frequency cutoff of 1.5\(f_0\) since the noise masks any intensity information encoded in the fibers with higher CFs. In all simulations, the stimuli were 200 ms in duration with a 20 ms rise/fall time.
The same normal- and impaired-hearing subjects were measured in a study conducted by Schroder and her colleagues, in which the performance of a count-based detector was evaluated. Data showing that it is possible for some impaired individuals to achieve normal intensity DLs, even when the spread of excitation is somewhat restricted, as in the flat loss condition, is critical to achieving normal performance in quiet. 

When the spread of excitation is more restricted, as in the flat loss, the count-based detector predicts elevated DLs, whereas the optimal detector predicts normal intensity DLs across frequency. For a flat loss, the count-based detector predicts elevated DLs, whereas the optimal detector predicts near-normal DLs. This suggests that, when the spread of excitation is somewhat restricted, as in the flat loss condition, temporal information is critical to achieving normal performance. These predictions generally agree with experimental data showing that it is possible for some impaired individuals to achieve normal intensity discrimination.

In our second simulation, we mimicked an experiment devised by Schroder and her colleagues in which the performance of the same normal- and impaired-hearing subjects were measured in quiet and high pass noise conditions. They observed that for listeners with a flat hearing loss, DLs were near-normal in quiet, but elevated in noise. In contrast, individuals with high frequency loss had elevated thresholds in quiet and were generally unaffected by noise in the region of the loss. Schroder and her colleagues concluded that normal-hearing individuals and those hearing impaired subjects who had near-normal DLs in quiet must be taking advantage of the spread of excitation to fibers with BFs at higher frequencies since these fibers are most affected by the high-pass noise. Impaired-hearing subjects with elevated thresholds in quiet were not using those higher frequency CF fibers originally, thus, their performance was relatively unaffected by the noise.

Figure 3 plots the percent change in DL between the quiet and high-pass noise conditions for the normal and impaired cases in our simulation. The most noticeable trend in the data is a decrease in the percent change as the frequency increases from 500 Hz to 8000 Hz. This behavior is due to the configuration of the model which only simulates nerve fibers with center frequencies up to 10 kHz. Despite this limitation, the relative performances under each condition can provide some insight into the effect of high pass noise on discrimination for each impairment configuration.

As Figure 2 illustrates, a high frequency loss can significantly affect theoretical intensity discrimination performance, even when the threshold at the signal frequency is normal (e.g., at 1000 Hz). On the other hand, when the outer hair cells are eliminated, near-normal intensity DLs are predicted across frequency. For a flat loss, the count-based detector predicts elevated DLs, whereas the optimal detector predicts near-normal DLs. This suggests that, when the spread of excitation is somewhat restricted, as in the flat loss condition, temporal information is critical to achieving normal performance. These predictions generally agree with experimental data showing that it is possible for some impaired individuals to achieve normal intensity discrimination.

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In comparing Figures 2 and 3, it appears that the closer the difference limen is to normal in quiet, the greater the effect of high pass noise. The high frequency loss condition is particularly revealing. At lower frequencies, where the DL is near-normal in the quiet condition, the percent change in the DL is relatively large, just like in the normal-hearing case. By contrast, at higher frequencies, where the DL is elevated in quiet, the change in DL is nearly zero. These results, similar to the observations made by Schroder and colleagues, support the hypothesis that the spread of excitation is an important factor in determining intensity discrimination ability.

Examining the physiological responses predicted by the model can provide additional theoretical support for these conclusions. As noted previously, removal of the OHCs affects the magnitude of the impaired response near the test frequency but not the spread of excitation across fibers. Thus, assuming that intensity DLs are correlated with this spread, it is reasonable that both the normal case and this impaired case will be similarly affected by high-pass noise. In contrast, the spread of excitation is more limited for a flat loss. Clearly, when high-pass noise is added, the DLs should be affected to a lesser extent than normal since many of these fibers convey very little or no useful information in the quiet condition. Applying the same argument to the high frequency loss condition, it is expected that high pass noise will have a smaller effect on performance at 1000 Hz since the spread of excitation is already severely limited in quiet. At lower test frequencies, the spread will not be as significantly affected at this stimulus level and more nor-
normal performance is expected and observed in Figure 3. Likewise, as the test frequency increases, the effect of the high frequency loss on the spread of excitation will become more pronounced and the change due to the addition of high pass noise will near zero.

Figure 4 compares normal and impaired Weber functions generated by the count-based detector. Results for the normal model are generally consistent with experimental data, showing a near miss to Weber’s law in quiet and more closely conforming to Weber’s law in noise. Results for the first two impairment configurations indicate near-normal Weber functions in both quiet and noisy conditions except at low SPLs where elevated thresholds result in larger DLs. The high frequency loss condition behaves normally in noise at 500 and 1000 Hz. However, in quiet, the DLs are nearly constant at higher SPLs and do not show the near miss normally observed. These results can be attributed to the fact that normally, as the intensity of the stimulus increases, the spread of excitation to fibers with BFs above the stimulus frequency also increases. This spread allows more fibers to contribute to overall intensity discrimination. Since, by definition, a high frequency loss reduces the utility of fibers with higher frequency BFs, even in quiet, the impaired listener will not benefit in the same way. As a function of frequency, the level at which this effect is first observed decreases as the frequency increases. This level is dependent on the specific configuration of high frequency loss since it is determined by the level at which the normal spread of excitation extends into this frequency region. Correspondingly, the absolute difference between quiet and noisy conditions decreases as frequency increases.

In summary, this theoretical approach provides a useful tool for studying the effects of auditory impairments on psychophysical behavior. Its applicability to other tasks is also promising. For example, the knowledge gained from this and similar studies may be useful in determining the best signal processing to implement in a new digital hearing aid. In addition, by simulating the proposed hearing aid and using this module to pre-process the input into the computational model, this theoretical approach may be used to quickly evaluate potential remediation strategies. Thus, while human experiments will always be critical to the study of auditory processing in both normal and impaired systems, this theoretical approach provides another tool to supplement this research.

6. REFERENCES