Characterizing individual differences in frequency coding: Implications for hidden hearing loss

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A long-standing debate in hearing research has focused on whether frequency is coded in the peripheral auditory system via phase-locked timing information in the auditory nerve (temporal code), or via tonotopic information based on the firing rates of auditory-nerve fibers tuned to different frequencies (rate-place code). Because frequency discrimination is generally much more accurate than intensity discrimination, it has been thought that frequency is likely to be coded via a temporal code, whereas intensity is represented via a rate code. However, direct empirical tests of this assumption have produced mixed results. This paper reviews a way in which the coding of both frequency and intensity might be reconciled within a single mechanism, and then uses an approach based on simple signal detection theory to predict the effects of a loss auditory-nerve synapses (synaptopathy) on some basic psychoacoustic phenomena, such as detection thresholds, frequency discrimination, and intensity discrimination. The predictions provide a baseline with which to compare future empirical findings based on the perceptual consequences of synaptopathy, or "hidden hearing loss."

INTRODUCTION

The coding of frequency is critical to many aspects of auditory perception, such as speech perception, music perception, and auditory scene analysis. A long-standing question in auditory science is how frequency is coded in the peripheral auditory system. The two most common candidates involve a code based on the tonotopic representation of frequency along the cochlea's basilar membrane, leading to differences in firing rate in auditory nerve fibers tuned to different characteristic frequencies (rate-place code), and a code based on the phase-locked timing of auditory nerve spikes (temporal code) (e.g, Siebert, 1970; Heinz *et al.*, 2001a).

In general, the information carried in the timing information is far greater than that carried in the rate-place information, assuming optimal processing of that information. Processing of timing information would require some neural mechanism that can precisely measure the time intervals between neural spikes with a resolution of microseconds and for delays as large as tens of milliseconds. Although evidence for

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neural coding accuracy down to microseconds has been found for the binaural system when processing interaural time differences (ITDs) (e.g., Yin and Chan, 1990; Brand *et al.*, 2002), similarly direct evidence has not been identified for the processing of frequency. There is, however, a body of more indirect evidence, pointing to a role for the temporal code. First, frequency discrimination becomes much worse at high frequencies, with difference limens (as a percentage of the reference frequency) increasing by about an order of magnitude between 2 and 8 kHz (e.g., Moore, 1973; for a review, see Micheyl *et al.*, 2012). This deterioration at high frequencies is difficult to explain based simply on a peripheral rate-place code, but may be explained in terms of the upper limits of phase-locking in a temporal code (Heinz *et al.*, 2001b). Second, studies have generally found little to no relationship between pure-tone frequency discrimination at low or high frequencies and frequency selectivity, suggesting that a rate-place code based on tonotopic representation is unlikely to limit performance (Tyler *et al.*, 1982; Moore and Peters, 1992). Third, detection thresholds for frequency modulation (FM) depend on modulation rate and carrier frequency in a way that is not found for amplitude modulation (AM). At low carrier frequencies (< 4 kHz) and slow modulation rates (< 5 Hz), listeners are generally very sensitive to FM, whereas at higher frequencies and/or at higher modulation rates, performance deteriorates. This pattern of results, along with other evidence from the interference of AM on FM detection, has led to the proposal that slow-rate FM at low carrier frequencies is coded via a timing code that is temporally sluggish (i.e., unresponsive to rapid changes in frequency), whereas fast-rate FM, or FM at high carrier frequencies, relies on an FM-to-AM transformation via the auditory filters (e.g., Moore and Sek, 1995).

Perhaps because of the apparent need for fine timing information to code frequency, it has been hypothesized that temporal fine structure and temporal envelope coding may be particularly affected by a form of hearing loss, termed “hidden hearing loss” (Schaette and McAlpine, 2011) or “synaptopathy” that results from a loss of synapses between the hair cells and auditory nerve fibers (e.g., Kujawa and Liberman, 2009). Several studies have now suggested a link between synaptopathy and certain behavioral deficits observed in temporal coding in the absence of traditional clinical hearing loss (Plack *et al.*, 2014; Bharadwaj *et al.*, 2015).

In this paper we review two recent studies, one empirical and one theoretical, that address the question of how frequency and intensity changes are coded. Finally, we present a simple analysis based on signal detection theory for predicted effects on signal detection, as well as frequency and intensity coding, of hidden hearing loss.

EMPRICAL TEST OF TEMPORALLY CODED SLOW FREQUENCY MODULATION

Whiteford and Oxenham (2015) carried out a correlational study involving 100 young normal-hearing listeners. They measured detection thresholds for FM, AM, dichotic FM (introducing dynamic ITD cues), and dichotic AM (with dynamic interaural level difference, ILD, cues), all with a carrier frequency of 500 Hz and a slow (1-Hz) or fast (20-Hz) modulation rate. In addition, frequency selectivity around 500 Hz was

estimated using a forward-masking paradigm. The hypothesis was that slow-rate FM and ITD coding are both governed by the same temporal (phase-locking) code, and so should be correlated, whereas fast-rate FM is determined by FM-to-AM translation, and so should be correlated with the threshold predicted from fast-rate AM thresholds combined with the measure of frequency selectivity. Whiteford and Oxenham (2015) found a reasonable correlation (around r = 0.5) between essentially all measures of modulation detection, slow and fast, FM and AM, and dichotic and diotic. Although the correlation between slow FM and dichotic FM thresholds was consistent with the hypothesis, the fact that the correlations were similar for all modulation-detection tasks was not. In addition, the measure of frequency selectivity was not correlated with either fast or slow FM, even when AM sensitivity was accounted for. In other words, the results provided no support for the idea that slow FM is coded differently from other forms of modulation.

Whiteford and Oxenham's (2015) negative result may be because thresholds are not limited by peripheral sensory factors, such as auditory-nerve coding, but are instead limited by higher-level (e.g., cortical) sensory or cognitive factors. Alternatively, similar peripheral mechanisms may limit both FM and AM perception at both low and high modulation rates, leading to the common source of variance. This common variance may reflect a common neural code, or it may simply reflect a common mode of transmission; for instance, damage to the auditory nerve would result in poorer transmission of both rate-place and timing codes. A next step for this line of investigation is to study correlations using a more diverse population of subjects, to study the effects of ageing and the effects of hearing loss. For instance, it has been suggested that ageing results in a selective deficit in temporal fine structure processing (Moore *et al.*, 2012). If so, then stronger correlations between diotic and dichotic slow-rate FM detection thresholds might be observed in a population that had a wider age range. Similarly, cochlear hearing loss due to dysfunction of the outer hair cells leads to a loss of sensitivity and poorer frequency selectivity (e.g., Moore *et al.*, 1999). Therefore, including subjects with a range of cochlear hearing losses may result in a clearer correlation between fast-rate FM detection thresholds and estimated frequency selectivity.

The next section reviews one possible way in which AM (fluctuations in intensity) and FM (fluctuations in frequency) might be coded similarly, and yet remain consistent with the finding that frequency coding appears more accurate than intensity coding.

A COMMON CODE FOR FREQUENCY AND INTENSITY?

Even if a temporal code is admitted for representing frequency at the level of the auditory periphery, it is unlikely that such a code survives the transformations between the cochlea and primary auditory cortex. Instead, by the time the processing reaches auditory cortex, any timing information extracted from the temporal fine structure of tones has probably been transformed into some form of rate-based population code (e.g., Wang *et al.*, 2008). This leaves a potential problem: Frequency difference limens (FDLs), as well as FM detection thresholds at slow rates and low carrier frequencies,

are generally much smaller than would be predicted by a just-detectable change in excitation pattern, based on measured intensity difference limens or AM detection thresholds at similar rates and carrier frequencies (Glasberg and Moore, 1986; Lacher-Fougere and Demany, 1998). If both intensity and frequency are coded by a rate-place in auditory cortex, then how can the apparent discrepancy between the accuracy of intensity discrimination and frequency discrimination be resolved?

Micheyl *et al.* (2013) recently proposed a solution to this apparent discrepancy. Their solution was based on the potential for correlations between the responses of neurons to the same stimulus, even in the absence of stimulus variability. This so-called "noise correlation" (Cohen and Kohn, 2011) generally decreases the benefit of pooling information across neurons. For instance, consider the case where an increment in the intensity of a stimulus is to be detected via a change in the firing rate of a population of neurons. The sensitivity of a single neuron is given by difference in mean firing rate in response to the baseline and the incremented stimuli (M_{R2}-M_{R1}), divided by the standard deviation (σ, i.e., the trial-to-trial variability of the neural response). This provides a measure of sensitivity, d', for each individual neuron: $d' = (M_{R2}-M_{R1})/\sigma$.

Assuming independence between all neurons, the optimal decision rule is to combine the information from across all N neurons (e.g., Green *et al.*, 1959):

$$d'_{TOT} = \sqrt{\sum_{i=1}^{N} d'^2_i} \qquad \text{Eq. (1)}$$

So, for instance, doubling the number of independent neurons leads to an increase in d' of a factor of $\sqrt{2}$, or about 1.4. However, if the neurons are all completely correlated (noise correlation coefficient = 1), then no benefit is derived from combining the information from multiple neurons, as the total information is the same as the information from just a single neuron. Therefore, as the degree of correlation increases from 0 to 1, the increase in sensitivity as a function of N decreases from a factor of $\sqrt{N}$ to 1 (no change).

When the task involves detecting a change in frequency, however, the situation is different. Now, a noise correlation can in some cases *improve* performance. For instance, consider two neurons with characteristic frequencies (CFs) on either side of the test-tone frequency. When the frequency of the tone is increased, the response of the neuron with the higher CF will increase, whereas the response of the neuron with the lower CF will decrease. Thus, an optimal combination of information will involve some form of *subtraction* of the two responses, as opposed to the *addition* that would be required in the intensity-discrimination condition. When responses are added, noise correlation increases in the internal noise; when responses are subtracted, any noise correlation can be potentially subtracted out and hence eliminated. Thus, in the case of frequency discrimination, noise correlation may improve performance. This difference between frequency and intensity coding is illustrated in Fig. 1, which shows the responses of two sample neurons with some degree of correlation. The spike rate

of one unit (*j*) is plotted as a function of the spike rate of another unit (*i*). If the spiking rate of the units were uncorrelated, the distributions would be circles; the oval distributions show that there exists a positive correlation (perfect correlation would be represented by a straight line along the major diagonal). Panel A illustrates the case of frequency discrimination, where unit *j* has a CF higher than the test frequencies, and unit *i* has a CF lower than the test frequency. When the stimulus frequency is increased from the reference (blue) to the higher frequency (red), the average firing rate of *j* increases, whereas the average firing rate of *i* decreases. In this situation, the fact that the firing-rate distributions are oval means less overlap (and hence better discriminability) between the two joint distributions than would be the case with independent firing rates. The opposite is true for the case of intensity discrimination (Panel B). Here the oval distributions lead to more overlap (and hence worse discriminability) than would be the case with independent firing rates. Using this kind of approach, Micheyl *et al.* (2013) showed that the same model could account for human performance in both intensity and frequency discrimination, using the same rate-place neural coding, by assuming a degree of correlation that was within the range of those observed in auditory cortical recordings. The work thus shows that it is not necessary to assume different neural codes to account for human frequency and intensity discrimination abilities.

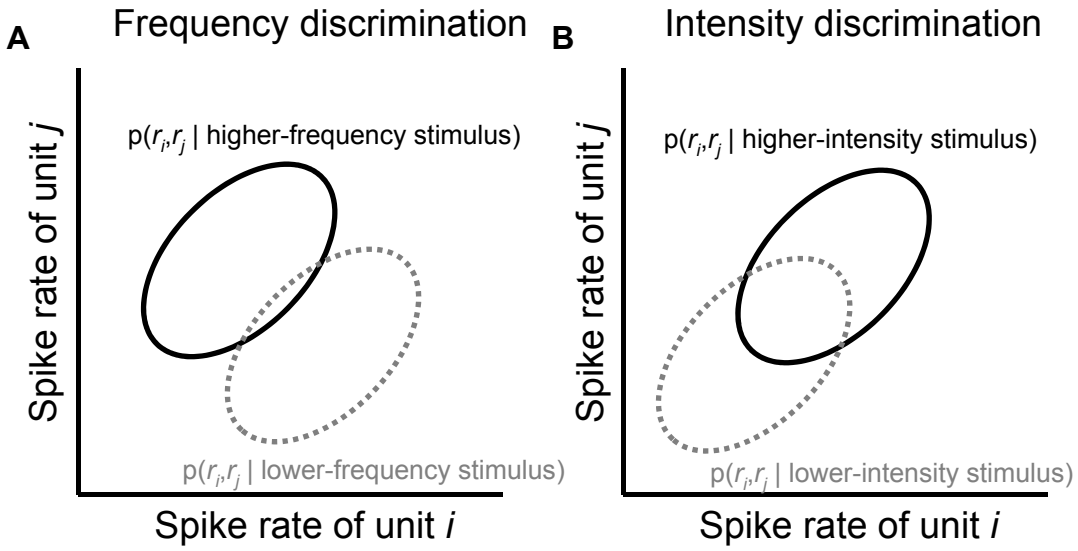

Fig. 1: Schematic diagram of the response distributions of two sample neurons (*i* and *j*) to illustrate the effects of noise correlation between neurons in frequency and intensity discrimination task. Redrawn from Micheyl *et al.* (2013).

PREDICTED EFFECTS OF HIDDEN HEARING LOSS

As the recent work of Liberman, Kujawa, and colleagues has shown (e.g., Kujawa and Liberman, 2009; Sergeyenko *et al.*, 2013; Fernandez *et al.*, 2015), noise exposure that causes only a temporary shift in thresholds (measured behaviorally and neurally) can nevertheless lead to permanent loss of synapses (of 50% or more) between the inner hair cells and auditory nerve fibers. This synaptopathy, has been termed "hidden hearing loss" (Schaette and McAlpine, 2011; Plack *et al.*, 2014), because it would not be detected by a traditional audiogram.

The reasons why absolute thresholds remain unaffected by hidden hearing loss are not completely clear. One possibility is that the synaptic loss seems to be concentrated in fibers with low spontaneous firing rates and high thresholds (Furman *et al.*, 2013), meaning that the high-spontaneous-rate fibers with low thresholds, which are presumably responsible for detecting low-intensity sounds, are less affected. There has been some speculation as to what perceptual abilities might be most affected by hidden hearing loss, including poorer temporal processing (similar to that found in people with auditory neuropathy or dys-synchrony), deficits in processing supra-threshold sounds, particularly at higher sound levels, and understanding speech in noise (Plack *et al.*, 2014; Bharadwaj *et al.*, 2015).

At this point it may be useful to generate some basic expectations regarding performance in perceptual tasks, based on signal detection theory (Green and Swets, 1966), along with some highly simplified assumptions concerning peripheral auditory processing. The analysis below follows in the tradition of Viemeister (1988), who calculated the number of auditory nerve fibers required to achieve human levels of intensity discrimination, based on the response properties of single neurons.

Model assumptions

In estimating the effect of losing synapses (and hence functionally losing auditory nerve fibers), the simplest assumptions are that: 1) the response of each auditory nerve fiber is independent from the responses of the others, and 2) the information from all the auditory nerve fibers is optimally combined. In this case, the sensitivity of the system is described by the d'_{TOT} shown in Eq. 1, where d'_i is the sensitivity of an individual auditory nerve fiber, i. For this initial analysis, a further simplifying assumption is that all auditory nerve fibers carry equal information or, equivalently, a loss of functional auditory nerve fibers affects the entire population proportionally.

Predictions for detecting a signal in quiet or in noise

Many studies have shown that the sensitivity to a signal in noise or quiet is proportional to the signal intensity, for a given signal duration and frequency (e.g., Green *et al.*, 1959; Hicks and Buus, 2000). For instance, a doubling in sensitivity should lead to a halving in the sound intensity, or a 3-dB decrease in level, required for detection threshold. Taking our simplified assumptions along with Eq. 1, we can see that decrease in the number of functional auditory nerve fibers by a factor F will lead to a decrease in the overall d'_{TOT} by a factor $\sqrt{F}$. In other words a 50% (factor of

2) loss in auditory nerve fibers will lead to a reduction in sensitivity by a factor of √2. Because *d′* and intensity are proportional, a √2 decrease in *d′* implies a √2 increase in the intensity required to achieve threshold. This translates into a 1.5-dB increase in threshold. In other words, the model predicts that a 50% loss of fibers would lead to only a 1.5-dB change in threshold – one that is probably not measurable with standard audiometric equipment. Similarly, a dramatic 90% loss of fibers would still only predict a 5-dB increase in thresholds in quiet or in noise. The relationship between predicted threshold change (where a negative number implies a loss of sensitivity or increase in threshold) and proportional loss of synapses is shown in Fig. 2 for losses between 0 and 99% of synapses.

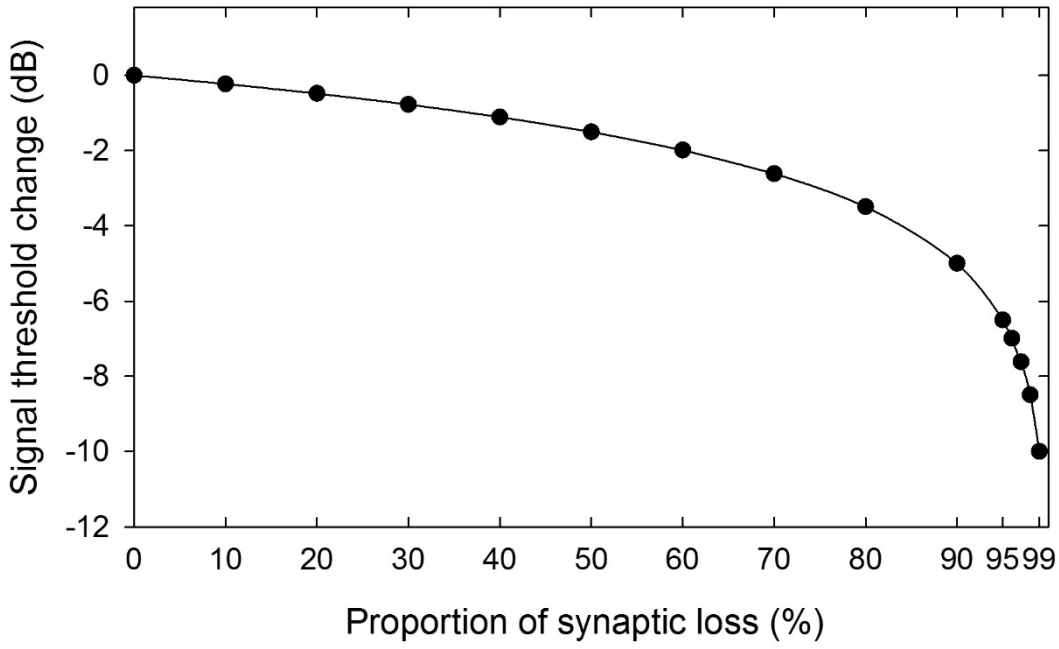

Fig. 2: Illustration of the predicted change in threshold, as a function of the proportion of lost synapses. Negative numbers imply a worsening, or increase, in threshold.As shown, even a 99% loss of synapses results in only a 10-dB change in threshold.

Predictions for auditory discrimination of frequency or interaural time differences

Similar predictions can be derived for any auditory task where the simplifying assumptions are reasonable and where the relationship between *d′* and the relevant stimulus parameter is known. For frequency discrimination, *d′* is generally proportional to the difference in frequency, *Δf* (e.g., Dai and Micheyl, 2011). Thus, by the same logic as outlined above, any decrease in *d′* due to loss of fibers would

result in a proportional increase in the Δf at a given threshold. For instance, a 50% loss of fibers would result in a predicted decrease in d' of $\sqrt{2}$, and so frequency-discrimination thresholds should increase by the same amount. Although a change in threshold from, say, 1% to 1.4% might be measurable within an individual subject, the large individual differences observed in normal-hearing listeners (e.g., Whiteford and Oxenham, 2015) would make it difficult to distinguish from other factors in the general population.

For the discrimination of intensity differences, d' has been found to be roughly proportional to the change in level (in dB), ΔL (Buus and Florentine, 1991; Buus *et al.*, 1995). Thus, according to our simplified model, a 50% loss in synapses would be predicted to produce a factor of $\sqrt{2}$ increase in the just-noticeable difference (JND). For instance, a JND of 1 dB would increase to 1.4 dB, which again would be barely measurable. It would take a more dramatic loss of 75% of synapses to double the JND to 2 dB.

The detection of interaural time differences (ITDs) is one psychoacoustic measure that almost certainly depends on auditory-nerve phase locking. Here again, d' is proportional to the ITD, so that a 50% reduction in fibers is predicted to lead to an increase in the threshold ITD by a factor of $\sqrt{2}$.

Predicting the effects on more complex tasks, such as speech understanding in noise, will take a more detailed approach. However, signal-detection-based approaches have been applied to the problem of speech understanding (e.g., Musch and Buus, 2001a; 2001b; Micheyl and Oxenham, 2012), so such approaches could likely be used to predict how speech intelligibility would be predicted to change in the face of auditory synaptopathy.

Model limitations

The predictions of the perceptual consequences of synaptopathy from the model outlined above are, of course, dependent on the model assumptions. All assumptions are highly simplified, and some are more justifiable than others, as outlined below.

The first assumption is that the responses from individual auditory-nerve fibers are independent. Based on available data, this assumption seems reasonable (in contrast to auditory cortical responses described in the previous section). However, if some correlation is assumed between neurons then the predicted effect of a loss of fibers becomes even smaller; as the assumed correlation increases from 0 to 1, the predicted change in d' decreases from a factor of $\sqrt{F}$ to no change.

The second assumption is that all fibers carry equal information. This is clearly not the case. For instance, at low intensities, most coding will be done by high-spontaneous-rate fibers, and fibers with low characteristic frequencies will have little influence on the coding of high-frequency sounds. In terms of high- vs. low-spontaneous-rate fibers, if synaptopathy does selectively affect low-spontaneous-rate fibers, then it may selectively and disproportionately impair processing at higher sound levels.

The third assumption is that the statistical distributions can be considered Gaussian and continuous. This assumption may fail in the cases where small numbers of neurons are involved and/or where the responses are more discrete in nature. For instance, if a brainstem neuron requires coincident input from two auditory-nerve fibers, then it will fail completely if just one of the fibers is no longer active.

Overall, the model should be treated as a very rough first approximation, but it nonetheless provides some insights into why a dramatic loss of fibers may result in behavioral changes that are barely measurable. More sophisticated and realistic models will likely provide an important tool in our quest to better understand the nature and consequences of different forms of damage to the human auditory system.

CONCLUSIONS

This paper reviewed two recent studies that investigated the possible neural codes underlying frequency and intensity coding in the auditory system. The first empirical study failed to find evidence that phase locking mediates the coding of slow-rate frequency modulation at low carrier frequencies (Whiteford and Oxenham, 2015). The second theoretical study showed how human performance in both frequency and intensity discrimination could be explained using a single rate-place code, if some degree of correlation between the responses of neighboring neurons is assumed. Regardless of the neural code used for frequency and intensity, decreasing the number of fibers carrying information, via synaptopathy or hidden hearing loss, will result in decreased performance. The final part of the paper outlined predictions of a highly simplified model based on signal detection theory that showed how a dramatic loss of auditory nerve fibers may only result in small, and in some cases unmeasurable, decreases in behavioral performance. Such modeling can be used as a 'baseline' with which to make specific predictions regarding the perceptual consequences of hidden hearing loss.

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