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A Spiking Neuron Model of Cortical Correlates of Sensorineural Hearing Loss: Spontaneous Firing, Synchrony, and Tinnitus

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Hearing loss due to peripheral damage is associated with cochlear hair cell damage or loss and some retrograde degeneration of auditory nerve fibers. Surviving auditory nerve fibers in the impaired region exhibit elevated and broadened frequency tuning, and the cochleotopic representation of broadband stimuli such as speech is distorted. In impaired cortical regions, increased tuning to frequencies near the edge of the hearing loss coupled with increased spontaneous and synchronous firing is observed. Tinnitus, an auditory percept in the absence of sensory input, may arise under these circumstances as a result of plastic reorganization in the auditory cortex. We present a spiking neuron model of auditory cortex that captures several key features of cortical organization. A key assumption in the model is that in response to reduced afferent excitatory input in the damaged region, a compensatory change in the connection strengths of lateral excitatory and inhibitory connections occurs. These changes allow the model to capture some of the cortical correlates of sensorineural hearing loss, including changes in spontaneous firing and synchrony; these phenomena may explain central tinnitus. This model may also be useful for evaluating procedures designed to segregate synchronous activity underlying tinnitus and for evaluating adaptive hearing devices that compensate for selective hearing loss.

1 Introduction

Persistent tinnitus is a frequently occurring symptom in individuals with sensorineural hearing loss and can be extremely distressing. Such hearing loss, characterized by the loss of inner and outer hair cells, occurs in normal aging and with noise-induced trauma. As such, persistent tinnitus is quite common, especially among older adults. The hair cells tuned to high frequencies are especially vulnerable to damage, and as a result, hearing loss is most common in the high frequencies. The tinnitus percept may be either tonal or more broadband in nature and is typically within the region of hearing loss (Eggermont, 2003). Animal studies have identified many changes that occur throughout the auditory system with noise-induced high-frequency hearing loss. At the level of the auditory nerve, these include increased synchrony capture of high-frequency fibers by lower frequencies and broadening of tuning curves (Norena & Eggermont, 2003; Norena, Tomita, & Eggermont, 2003). In both the auditory nerve and the inferior colliculus, spontaneous firing rates are lower in the damaged animal than in the healthy animal (Chen & Jastreboff, 1995), while spontaneous rates are elevated at the higher levels of dorsal cochlear nucleus and primary auditory cortex (A1) (Seki & Eggermont, 2003). In addition, at the cortical level, there is increased synchrony in firing in the damaged region (Eggermont, 2003). Furthermore, the tonotopic map is reorganized such that neurons formerly tuned to high frequencies now respond best to frequencies near the edge of the region of hearing loss (Seki & Eggermont, 2003).

Tinnitus associated with hearing loss has been characterized as a phantom sensation (Rauschecker, 1999), akin to phantom limb syndrome. Like phantom limb sensations, it is a percept arising from a damaged sensory system. We speculate that cortical correlates of hearing loss such as increased synchrony, elevated spontaneous firing rates, and shifted tonotopic maps may be responsible for the tinnitus percept. For example, areas of A1 that were originally associated with frequencies now in the region of hearing loss may now be driven by other frequencies or by spontaneous input generated by feedback within the cortex. We present in this article a model that captures some of these cortical correlates and in the future hope to develop this model as a platform to test behavioral therapies for tinnitus.

2 Related Work

Given our long-term goal of developing models that lead to rehabilitation strategies for tinnitus, it is important that our model capture the relevant features of A1. In this section, we review what is known about the anatomical and functional organization of A1, particularly with regard to plasticity and reorganization after damage.

2.1 Functional and Anatomical Organization of A1. Little work has been done to describe in detail the functional and anatomical organization of A1 in humans. For this reason, we rely primarily on the literature describing this organization in other mammals, including rabbits, rats, and cats.

The primary auditory cortex displays a roughly cochleotopic organization along the latero-medial axis with expanded representations of behaviorally significant characteristic frequencies. (For a review, see Read, Winer, & Schriener, 2001.) The organization along the orthogonal, isofrequency axis is less clear. It appears to be a fairly complicated arrangement with several overlapping, patchy maps of various parameters, including bandwidth, binaural response type, intensity selectivity, timbre, and more complex spectrotemporal features (Read, Winer, & Schriener, 2002; Velenovsky, Cetas, Price, Sinex, & McMullen, 2003). Along this axis, narrow and broadband regions alternate, but there are also patches with consistent binaural response type, intensity threshold, operating range, and response to frequency modulation. The bandwidth and threshold topographies are correlated, in that broadband areas have higher response thresholds. In addition, binaural properties vary in a periodic fashion, but their relationship to other parameters is unclear (Read et al., 2002). Langner, Sams, Heil, and Schulze (1997) in magnetoencephalography (MEG) studies found evidence that periodicity, and timbre vary along the isofrequency axis.

In addition to short-range lateral excitatory and inhibitory lateral connections, long-range lateral connections exist. These long-range lateral A1-to-A1 connections tend to be within the isofrequency contours, and there are few that cross the cochleotopic dimension. There are connections between patches with similar narrow-band spectral tuning properties, which could be a system for processing with high spectral resolution (Read et al., 2001).

2.2 Plasticity and Reorganization After Damage. Neuroplastic reorganization has been postulated to contribute to the tinnitus percept. In this section, we review evidence for plasticity in the adult primary auditory cortex. In the next section, we review models of cortical reorganization and discuss their plausibility as candidate mechanisms underlying tinnitus.

Plasticity is necessary in development in order to achieve such important skills as sound localization. Since humans have a large degree of variation in shape and size of pinnae, as well as a lesser degree of variation in shape and size of the head, each person has a different head-related transfer function that changes during development. Thus, plasticity is needed to develop the highly accurate sound localizations that most adults possess. (For an excellent review of auditory cortical plasticity, see Rauschecker, 1999.)

But is auditory cortical plasticity active in adults? Musical skill is associated with enhanced auditory cortical representation for musical notes as measured with MEG (Pantev et al., 1998), but that enhancement is correlated with the age of first learning the instrument (earlier learners have

more enhancement). Further experiments show that highly skilled musicians have enhanced auditory cortical representations for the timbre of their own instrument relative to other instruments and pure sine waves (Pantev, Roberts, Schulz, Engelien, & Ross, 2001). This indicates that the expansion is probably due to increased exposure and salience of those tones, rather than that people who become musicians are just naturally endowed with enhanced auditory systems. Further experiments (Menning, Roberts, & Pantev, 2000) show that intensive discrimination training in adults can induce similar expansion of cortical representation for the frequency trained. Thus, the plasticity is not limited to childhood and can be induced in normal adults.

Animal studies allow for a more direct and precise measurement of cortical representations and changes to them. Kilgard and Merzenich (1998) showed that receptive field size in the auditory cortex of the adult rat can be narrowed or broadened when appropriate auditory stimuli are paired with stimulation of the nucleus basalis. Although direct stimulation of the nucleus basalis is not a natural phenomenon, this does confirm that auditory cortical receptive fields are subject to plastic change.

Reorganization of cortical frequency response characteristics in A1 depends on the size of the cochlear or spiral ganglion lesion. Lesions covering a small area (1 mm of cochlea, or approximately 1 octave) are associated with rapid subcortical changes in frequency tuning (Snyder, Sinex, McGee, & Walsh, 2000) and no change in cochleotopic maps of frequency selectivity near threshold (Rajan & Irvine, 1998). Rajan (1998) investigated the effects of receptor organ damage on the organization of auditory cortex in adult cats. He found that limited damage to auditory receptors causes loss of functional surround inhibition in the cortex, unmasking of latent inputs, and significantly altered neural coding. However, these changes do not lead to plasticity of the cortical map, defined by the most sensitive input from the receptor surface to each cortical location. Thus, in sensory cortex, loss of surround inhibition as a consequence of receptor damage does not always produce a change in topographic mapping, especially when that receptor damage is not extensive.

Larger lesions, however, have been shown to produce cochleotopic map reorganization in auditory cortex. Norena et al. (2003) found that after a 1 hour exposure to a 120 dB sound pressure level pure tone, significant peripheral damage covering two to three octaves resulted. This trauma induced a shift in characteristic frequencies of cortical neurons toward the lower frequencies, as well as an increase in bandwidth of the tuning curve and other changes.

Plastic reorganization in the visual system has been much more extensively studied than in the auditory system and may provide insights relevant to the auditory system.

Chino, Kaas, Smith, Langston, and Chen (1992) found rapid reorganization of cat visual cortex after partial deafferentation of the retina, but only

if the undamaged eye was removed. Thus, bilateral deafferentation is necessary for reorganization to occur. The reorganization occurs within hours and is complete (there are no "silent" areas of cortex that respond to nothing). Because it is so rapid, the reorganization must be due to reweighting of existing connections rather than growth of new connections.

Whether thalamo-cortical afferents are plastic in the adult after loss of sensory input, but in the absence of total deafferentation, remains unclear. The large shifts in tuning curves resulting in gross topographic map reorganization could be due to other factors, such as unmasking of weak, previously noneffective afferent connections, plasticity on long-range horizontal connections, or cortico-thalamic connections. In support of the latter possibility, Ivanco (1997) observed long-term potentiation (LTP) *in vivo*, in awake, behaving rat in cortico-thalamic auditory pathways but not thalamo-cortical pathways. The shifts in tuning curves could also be due to a downregulation in GABAergic inhibition known to result from loss of sensory input (Mossop, Wilson, Caspary, & Moore, 2000) at several levels of the auditory system that could unmask broadly tuned excitatory afferent connections or multisynaptic chains of lateral excitatory connections.

Generally it appears that large lesions are necessary for primary sensory cortex map reorganization to occur, but smaller amounts of damage can cause other changes that may be important to the understanding of tinnitus.

2.3 Modeling. Very little work has been done on modeling plastic reorganization in the auditory cortex. Mercado, Myers, and Gluck (2001) simulated experience-dependent plasticity in a Kohonen map representation of auditory cortex. They simulated training an animal on a single frequency and the subsequent overrepresentation of that frequency in cortex by training the network on input at only one frequency, thereby increasing the representation of that frequency in the network. Jenison (1997) modeled the reorganization of the tonotopic map after partial deafferentation via plasticity of thalamic afferent connections to a two-dimensional sheet of cortical neurons with short-range lateral excitation and long-range lateral inhibition. After a simulated cochlear lesion, the thalamic afferent connections are reorganized using Hebbian learning, while the lateral connections were held constant. After recovery (relearning), the cortical units that had responded to the damaged portion of the cochlea responded to neighboring areas, thus successfully simulating the cortical reorganization seen in animal studies of noise-induced hearing loss. However, neurophysiological work suggests that changes to thalamocortical connection strengths are not enough to fully explain the tonotopic map reorganization seen in animal studies of noise-induced hearing loss. Paired thalamo-cortical recording studies (Miller, Escabi, Schreiner, 2001) suggest that A1 neurons receive afferent input from about 30 thalamic neurons. Thalamocortical pairs have either the same best frequencies (± 0.05 octaves) or best frequencies with a maximal difference of $\pm 1/3$ octave (Miller, Escabi, Read, & Schreiner,

2001). In contrast, tonotopic map reorganization can result in tuning curve shifts of multiple octaves. Such large shifts in tuning are not likely mediated by increased excitatory drive from thalamocortical afferent convergence. A1 neurons also receive a large degree of nonthalamic input, including cortical afferent input (Miller, Escabi, Read et al., 2001).

Tonotopic reorganization may explain some perceptual phenomena associated with hearing loss, including hyperacusis for frequencies near the edge of hearing loss (Eggermont, 2003). However, it may not be necessary, or even sufficient, to explain the emergence of tinnitus. More likely correlates of tinnitus are the increased spontaneous firing and increased synchrony. Several modelers have proposed mechanisms of tinnitus based on subcortical lateral inhibition, which enhances the response of neurons adjacent to a deafferented region (Bruce, Bajaj, & Ko, 2003; Gerken, 1996). This may give rise to an enhanced sensory-driven response to stimuli near the edge of hearing loss, but would not account for spontaneously generated activity in this frequency range in the absence of hearing loss. Further, subjective ratings of tinnitus indicate that the perceived sound is typically well within the range of hearing loss (Norena, Micheyl, Chery-Croze, & Collet, 2002), not at the edge of the loss region.

Although no modeling work has been done on illusory auditory percepts in cortex, Wilson's computational account of illusory visual percepts such as migraine auras and Charles Bonnet syndrome may give some insights into what is causing the tinnitus percept. Wilson, Blake, and Lee (2001) explained binocular dominance wave propagation (oscillating illusions caused by static patterns) in terms of recurrent excitation and mutual inhibition. He described the illusions as being caused by a dense set of competing stimuli. Inhibitory interactions among V4 concentric units cause the hallucinations, and the oscillations are caused by spike frequency adaptation. Selective attention results from biasing these competitive interactions in parallel networks.

3 Model

Our neural network model is built on a spike response model neuron that captures the shapes of excitatory and inhibitory postsynaptic currents and of action potentials (Gerstner & Kistler, 2002; Bruce et al., 2003). The input to our model is generated by a lateral inhibitory network (LIN) (Bruce et al., 2003) model of midbrain auditory processing. This model captures several important aspects of subcortical changes after high-frequency hearing loss: it produces output with decreased spontaneous firing in the impaired region and, via lateral inhibition, exhibits enhanced stimulus-generated firing of neurons tuned to the edge frequencies due to loss of surround inhibition. Our cortical model, which is the novel component of this work, includes a layer of ventral medial geniculate input neurons and a cortical layer of neurons with geniculate afferent input, and lateral excitatory and inhibitory

connections. The widespread afferent cortical connections result in broad frequency tuning sharpened by lateral inhibition. Simulated high-frequency peripheral damage in the input model results in enhanced firing of neurons at the edge frequency region in the cortical model, thus capturing some of the tonotopic map reorganization seen after high-frequency hearing loss. We hypothesized that the changes in synchrony and spontaneous firing rates are due to compensatory changes in the gain of lateral connections in the cortex after the reduction of afferent input. Our simulations show that both increasing the gain on lateral excitatory connections and decreasing the gain on lateral inhibitory connections result in increased spontaneous firing rates and increased synchrony in the cortical hearing loss region (Wierenga, Ibata, & Turrigiano, 2005).

3.1 Model Neuron. The cortical neuron membrane potentials ($v_i(t)$) are described by the following equation (for all potentials less than the spiking potential),

$$\tau dv(t)/dt + v(t) = \mathbf{V}i_a(t) + \mathbf{U}i_e(t) - \mathbf{W}i_i(t), \quad (3.1)$$

where τ is the membrane time constant (τ was set to 5 milliseconds for these simulations), \mathbf{V} is the matrix of thalamic afferent connection strengths, \mathbf{U} is the matrix of lateral excitatory connections, \mathbf{W} is the matrix of lateral inhibitory connections, $i_a(t)$ is the vector of thalamic afferent, $i_e(t)$ is the vector of excitatory lateral input, and $i_i(t)$ is the vector of inhibitory lateral input at time t (time step size was 2 milliseconds).¹ The input is a postsynaptic current in response to spiking activity at the input neuron. The postsynaptic currents are described by the following equation:

$$i(t) = (\alpha/10\tau)^2 t \exp(-\alpha t/\tau). \quad (3.2)$$

For excitatory neurons, postsynaptic currents are sharp and fast ($\alpha = 5$), and for inhibitory neurons, postsynaptic currents are slower ($\alpha = 1$; see Figure 1A). When the membrane potential reaches the spiking threshold, a spike is generated (membrane potential spikes and then is reset to a low value), and a refractory period is entered (see Figure 1B). Input to the model is created by running Poisson-generated spontaneous input plus an optional tonal stimulus through a spiking LIN (see Bruce et al., 2003). A hearing impairment is modeled at the input level by decreasing both the spontaneous rate and the stimulus-driven response rate in the deafferented region.

¹ This step size was chosen for the purposes of the synchrony analysis. See section 4.3 for further discussion.

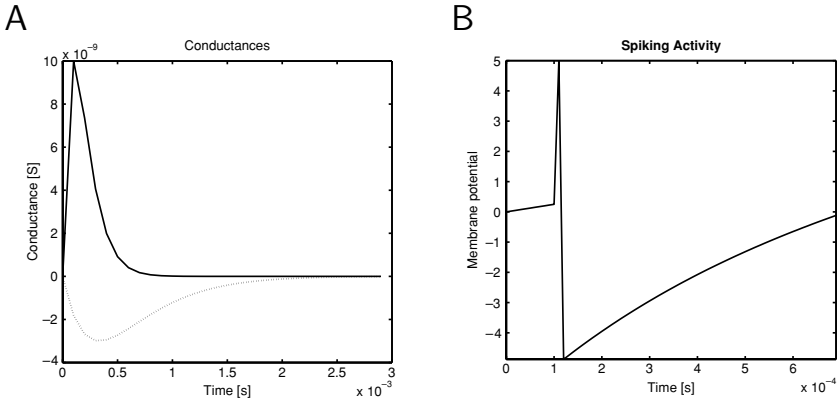


Figure 1: Model neuron. (A) Postsynaptic currents in response to an excitatory (solid) or inhibitory (dotted) input spike at time 0. (B) Spiking activity. The membrane potential of a neuron in response to a constant input current.

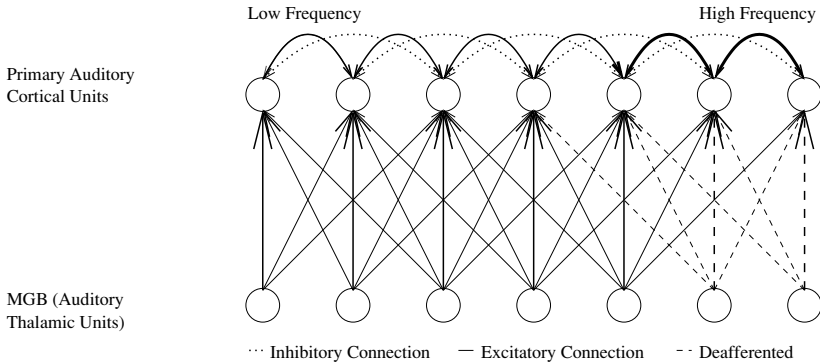


Figure 2: The structural architectures of the three models was identical. The cortical layer of units receives two kinds of input: afferent input from the thalamic layer and lateral input (both excitatory and inhibitory) from other cortical units. In all cases, connection strength was a function of distance. Solid lines represent normal excitatory connections, and dotted lines represent inhibitory connections. For the impaired models, afferent input was damaged at the higher frequencies (dashed lines).

3.2 Cortical Model. The model network consists of two layers: an input layer, analogous to auditory thalamus, and a cortical layer. The cortical layer receives diffuse, broadly tuned excitatory input from the thalamic layer, and both excitatory and inhibitory recurrent input. (See Figure 2.) The current cortical model lacks back projections to the thalamus. These projections

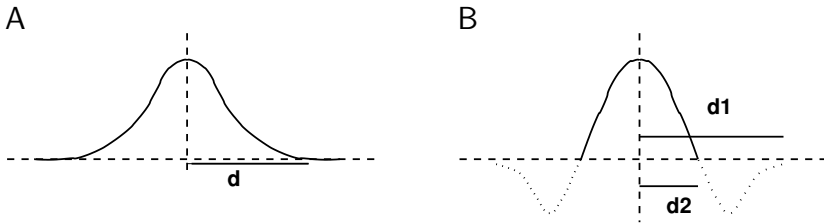


Figure 3: Connection strength as a function of distance. (A) Thalamo-cortical afferent connections. The range of connections (d) is $\pm 2/3$ of an octave, or 8 units. (B) Cortico-cortical lateral connections: d_1 is $\pm 3/4$ of an octave, or 9 units, and d_2 is $\pm 1/6$ of an octave, or 2 units.

are present in the mammalian auditory system in large numbers. As noted earlier, they are a good candidate for plasticity (Ivanco, 1997), which may further contribute to the development of the tinnitus percept. However, the function of these cortico-thalamic projections is not well understood at this time, so they have not been included.

The connection strengths of both thalamic afferents and lateral connections are a function of distance. Each model neuron in each layer is spatially arranged along a cochleotopic axis. The strength of a thalamic afferent connection is a gaussian function of lateral distance between the two neurons at their corresponding locations along the cochleotopic axis. The strength of a lateral connection is a Mexican hat function of the distance between the two neurons (see Figure 3). A cortical neuron receives afferent input from thalamic neurons with best frequencies within a range of $\pm 1/3$ octave of the cortical neuron's best frequency (Miller, Escabi, Read et al., 2001). The full range of the lateral connections is $\pm 3/4$ octaves, with the excitatory portion being $\pm 1/6$ octave. The lateral excitatory connection strengths are much weaker than the thalamic afferent connection strengths, but the lateral inhibitory connection strengths are matched to the excitatory thalamic afferent connection strengths. Thus, in the normal model, the lateral connections act as a mechanism to sharpen tuning.

We hypothesize that as a response to regional hearing loss and the consequent reduction in stimulus-driven thalamic afferents, compensatory changes occur in the connection strengths of lateral connections in that region. When hearing is impaired in a region of the frequency map, that region receives less afferent input. By changing the connection strengths of the lateral inputs, it is possible to raise the level of total input back to unimpaired levels.

There are three possible ways of changing lateral connection strengths to increase activity levels: increasing excitatory strength, decreasing inhibitory strength, or both. All three possibilities were tried, and the best results were obtained using the combination of increased excitation and decreased

Table 1: Model Parameters.

Parameter	Value
Neurons per layer	100
Neurons per octave	Approximately 11
Spontaneous activity rate	200 spikes/sec.
Peak tonal activity rate	500 spikes/sec.
Correlation measured with lags of	+/-50 ms

inhibition. Only decreasing inhibition produced qualitatively similar results to those presented here, but the levels of synchrony were lower. Increasing lateral excitation to the necessary level to achieve the unimpaired level of input without altering inhibition created an unstable, overactive network. Model parameters are summarized in Table 1.

The results presented here are from a model with increased strength of lateral excitatory inputs and decreased strength of lateral inhibitory inputs to the cortical units, which receive impaired thalamic afferent input, thus raising their total input levels to the input levels seen in the unimpaired region.

4 Results

We compared the performance of three model architectures on five different conditions. The three models were a “normal model” with input from a normally responding subcortical model; an impaired model with no compensatory changes to lateral connections, which had input from a damaged cochlear model but a cortical model identical to the normal model; and an impaired model with changes, which had input from the damaged cochlear model and compensatory changes in lateral connection strengths in the cortical model. Each model was tested on five input conditions: spontaneous input (no input tone) and with responses to four different input tones: one in the normal hearing region, one to either side of the edge of hearing loss, and one in the impaired region. There were 10 different randomly generated input sequences for each of the five input conditions.

4.1 Spontaneous Activity. The responses of the three models to spontaneous activity from the cochlear model can be seen in Figure 4. As one might expect, the response of the normal model (solid line) is essentially flat across the entire frequency spectrum. As the effect of hearing impairment in the cochlear model is lowered, spontaneous activity in the impaired region, the response of the impaired model with no changes to lateral weights (dashed line), also shows a drop in spontaneous activity. However, the impaired model with changes to lateral weights shows an increase in spontaneous

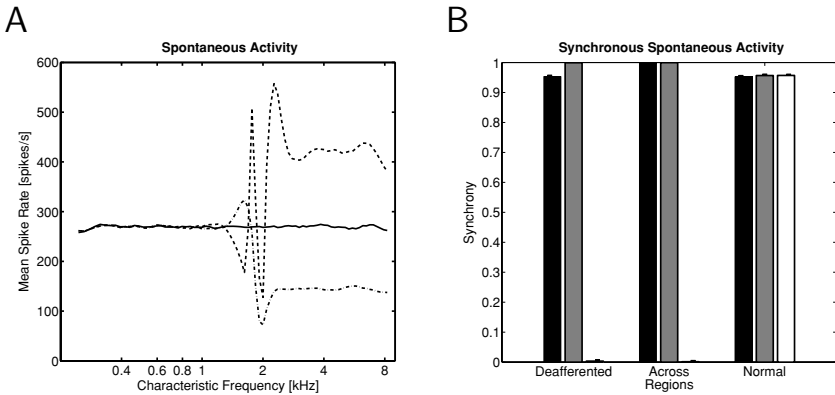


Figure 4: Response of the cortical models to spontaneous input (spike rate versus characteristic frequency). (A) Solid line: normal model; dash-dotted line: impaired model with no changes; dashed line: impaired model with changes. (B) Synchrony in spontaneous activity is greater in the impaired model with changes (gray bars) than in the normal model (black bars) or impaired model without changes (white bars). The difference is greatest in the region of hearing loss.

activity in the impaired region, as is consistent with physiological experimental findings.

4.2 Response to Tonal Input. The models were tested on four different types of tonal input. Their response profiles can be seen in Figure 5.

In Figure 5A, the tone is in the range of normal hearing. As this input is in the frequency range not affected by the impairment or by the changes in lateral weights, the differences between the three models are the same as those described for spontaneous inputs. The stimulus-driven activity is the same across the three models; they differ only in their responses to the spontaneous activity.

Figures 5B and 5C show the responses to tonal input near the edge of the hearing loss. The model with compensatory changes shows a particularly elevated response to tonal input just below the edge of the hearing loss. Figure 5D shows the models' response to a tone in the impaired region. Note that spontaneous activity and edge activity remain elevated. Further, note that while the impaired model without changes shows basically no stimulus-driven response, the impaired model with changes does show stimulus-driven response.

Another interesting aspect of the impaired model with changes is that it shows increased activity at the edge of the hearing loss. This could be an analogue of the hyperacusis that is frequently reported by tinnitus sufferers.

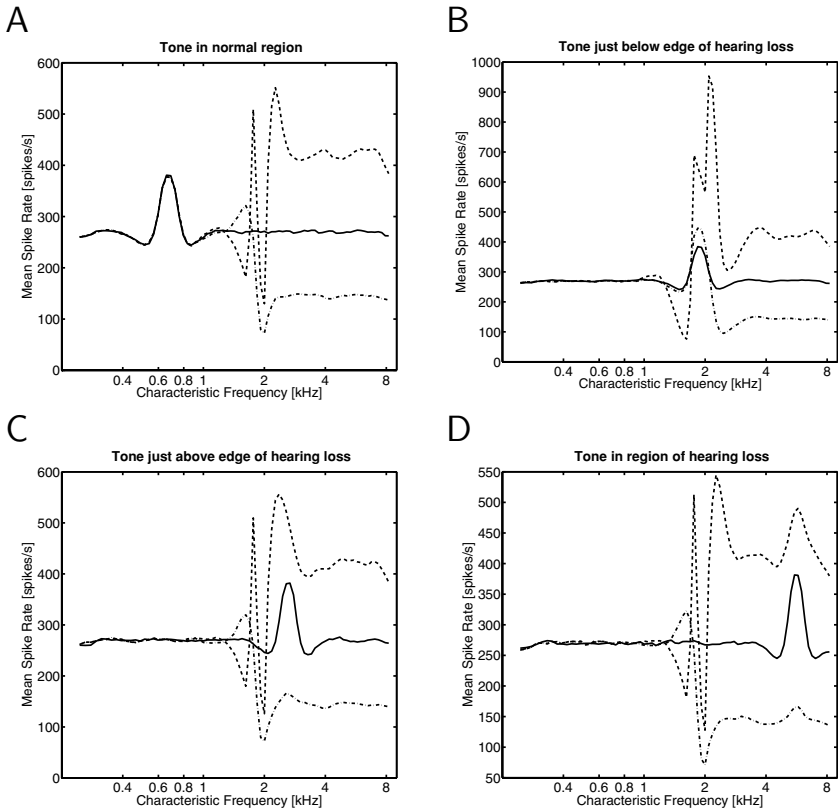


Figure 5: Response of the cortical models to a tonal input (spike rate versus characteristic frequency). Solid line: normal model; dash-dotted line: impaired model with no changes; dashed line: impaired model with changes. (A) Tone in normal hearing range. (B) Tone just below the edge of hearing loss. (C) Tone just above the edge of hearing loss. (D) Tone in the range of hearing loss.

The impaired model without changes also shows this feature, but to a much lesser degree.

4.3 Synchrony of Spontaneous Activity. Another change that occurs in animals with hearing loss is an increase in synchronous firing in the spontaneous activity of primary auditory cortex (Eggermont & Komiya, 2000; Norena & Eggermont, 2003; Seki & Eggermont, 2003). We evaluated the synchronous firing of our three models by computing the cross correlations of the spike trains of various cortical neurons across in a manner similar to that used by Norena and Eggermont (2003). We used a time step size of 2 ms, analogous to how they divided spike trains into 2 ms bins. We

Table 2: Normalized Synchrony Results with Standard Error.

	Normal	Impaired with Changes	Impaired without Changes
Deafferented	0.9533 +/-9e-4	0.9992 +/-1e-5	0.0033 +/-3e-5
Across	0.9985 +/-1e-4	0.9996 +/-2e-5	0.0014 +/-5e-4
Normal	0.9525 +/-7e-4	0.9566 +/-0.0012	0.9569 +/-7e-4

Note: Rows are regions of the model; columns are different models.

then calculated for each pair of neurons the cross correlations of the spike trains for every time lag within a 100 ms time window. We determined how many of the correlation coefficients were three standard deviations above the mean for that run. These were considered synchronous activity. These counts were then normalized for the sizes of the various regions to obtain comparable numbers.

We broke our results into three categories: those correlations computed only between neurons in the deafferented region, those computed between neurons in the normal range and those in the deafferented range, and those computed only between neurons in the normal region. The results can be seen in Figure 4 and Table 2. In all cases, synchrony was higher for the impaired model with changes than for the other models. The greatest increase in synchrony is in the deafferented region, as one would expect given that that is where the changes to the lateral connections were. Synchrony across regions was greater than synchrony in the normal region, as it included the impaired region. Finally, note that synchrony in the impaired model with no changes was extremely low in the deafferented region and in the across-region measurement. This is due to the overall low levels of activity in the deafferented region for that model.

5 Discussion and Conclusions

The dynamical modes of behavior of neural networks with Mexican hat profiles of lateral connectivity have been analyzed by Wilson and Cowan (1972, 1973) and Wilson (1999). As observed in our own simulations, when the weights on lateral connections are sufficiently strong, the network exhibits a spatially localized oscillatory response to external input, and when the level of lateral inhibition is weakened, the network exhibits oscillatory behavior over a long spatial extent (Wilson, 1999). Although the Wilson-Cowan model differs from ours in that it employs rate-coded neurons and separate populations of inhibitory and excitatory neurons, Gerstner and Kistler (2002) have proven the functional correspondence between rate-coded dynamical neurons of the Wilson-Cowan type and a spike response model similar to that employed in our simulations.

By changing the balance of excitation and inhibition on lateral connections in our cortical model, we were able to model several key features of cortical reorganization after peripheral impairment. Our model shows elevated spontaneous activity in the deafferented region, hyperexcitability at the edge of hearing loss, some spread of activation into the impaired region when driven by stimuli at the edge of hearing loss, and an increase in synchrony in spontaneous firing. In contrast, in our simulations of a model with hearing impairment but lacking compensatory changes in lateral connections, we observed no change in synchrony (simulation results not shown here) and decreased spontaneous firing rates. Thus, changes in the balance of excitation and inhibition of lateral connections in auditory cortex may be implicated in the sensation of tinnitus. These results are a promising step in the process of modeling, understanding, and eventually treating tinnitus.

6 Future Work

This work will be extended to automate the compensation method. Thus, the changes in connection strength will be generated through a homeostatic process designed to maintain a long-term average rate of activity.

A great deal is known about the organization of primary auditory cortex. For example, auditory neurons exhibit complex time-varying frequency-tuning profiles (Shamma & Versnel, 1995). Spatial summation across the lagged and nonlagged thalamic input neurons in our model will allow us to simulate the formation of spectrotemporal receptive fields at the cortical layer, as has been proposed in models of visual cortex (Bednar & Miikkulainen, 2003). Another key detail of auditory cortex is that along the dimension orthogonal to the tonotopic map axis, neuronal tuning curves vary in blob-like fashion according to their bandwidth and level (intensity threshold) tuning (for a review, see Read et al., 2002) as well as binaural responsiveness and other things (Velenovsky et al., 2003; de Venecia & McMullen, 1994). Further, long-range horizontal connections are patchy and tend to connect neurons with similar bandwidth tuning profiles (Read et al., 2002). There is also evidence of long-range horizontal connections along the axis of the tonotopic map between neurons tuned to harmonically related frequencies (Read et al., 2002). We hypothesize that plasticity on the long-range connections is a key link in the induction of tinnitus, and this is the main focus of our ongoing work in this area.

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