Effects of Amplitude Modulation on the Coding of Interaural Time Differences of Low-Frequency Sounds in the Inferior Colliculus. II. Neural Mechanisms

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D’Angelo, W. R., S. J. Sterbing, E.-M. Ostapoff, and S. Kuwada. Effects of amplitude modulation on the coding of interaural time differences of low-frequency sounds in the inferior colliculus. II. Neural mechanisms. J Neurophysiol 90: 2827–2836, 2003. First published July 30, 2003; 10.1152/jn.00269.2003. In our companion paper, we reported on interaural time difference (ITD)-sensitive neurons that enhanced, suppressed, or did not change their response when identical AM was added to both ears. Here, we first examined physical factors such as the difference in the interaural correlation, spectrum, or energy between the modulated and unmodulated signals. These were insufficient to explain the observed enhancement and suppression. We then examined neural mechanisms by selectively modulating the signal to each ear, varying modulation depth, and adding background noise to the unmodulated signal. These experiments implicated excitatory and inhibitory monaural inputs to the inferior colliculus (IC). These monaural inputs are postulated to adapt to an unmodulated signal and adapt less to a modulated signal. Thus enhancement or suppression is created by the convergence of these excitatory or inhibitory inputs with the inputs from the binaural comparators. Under modulation, the role of the monaural input is to shift the threshold of the IC neuron. Consistent with this role, background noise mimicked the effect of modulation. Functionally, enhancement and suppression may serve in detecting the degree of modulation in a sound source while preserving ITD information.

INTRODUCTION

A major finding of the companion paper (Sterbing et al. 2003) was that there were three classes of neurons: those that enhanced, those that suppressed, and those that did not change their response in the presence of modulation. For the 272 inferior colliculus (IC) neurons in our sample, relative to an unmodulated signal, modulation created a continuum from complete enhancement to complete suppression. Here, we examine the possible factors and mechanisms that underlie the effects of modulation.

An important question is if the effects of modulation can be attributed to physical factors. A common model of interaural time difference (ITD) processing is cross-correlation (Licklider 1959; Sayers and Cherry 1957; Yin et al. 1987). So, we compared the interaural correlation between unmodulated and modulated signals. A second factor is the spectral differences between the unmodulated and modulated ITDs (Geisler et al. 1969; Ingham et al. 2000). Compared with the unmodulated ITD, the spectrum of the modulated ITD is wider. Further, we considered the energy difference between the two signals.

Besides physical factors, what are the neural factors that would operate differentially to an unmodulated and a modulated signal? One factor may be the differences in neural adaptation to these signals. In one view, the role of adaptation is to make the neuron more sensitive to changes in the signal (Bibikov and Nizamov 1996). For example, in the frog midbrain, the addition of noise enhanced the discharge rate and the synchronization to an amplitude modulated signal (Bibikov 2002). Therefore adding background noise may have the enhancing and suppressing effects that we have observed with modulation. However, it should be noted that the frog data did not show suppressive effects with noise.

Another neural factor is the anatomical substrate that could underlie enhancement and suppression. Many of the ITD-sensitive neurons in the IC could receive converging inputs from bin- and monaural sources in the lower brain stem (Oliver and Huerta 1992; Oliver and Shneiderman 1991). Furthermore, the monaural sources can be excitatory or inhibitory and from the ipsi- or contralateral side. Such convergence is necessary to explain our enhancing and suppressive effects. To test the potential influence of these inputs on our neurons, we selectively modulated the signal to one ear or the other.

Our findings indicate that the enhancement and suppression cannot be completely explained by physical factors. Instead, it appears that modulation activates excitatory or inhibitory monaural inputs that adapt rapidly to an unmodulated stimulus. The enhancing or suppressive effects are created by the convergence of these excitatory or inhibitory inputs, respectively, onto ITD-sensitive neurons in the IC. The addition of background noise mimicked the effect of modulation.

METHODS

Recordings were made in unanesthetized, female Dutch-belted rabbits (1.5–2.5 kg) with healthy external ears. Surgeries were performed under anesthesia [ketamine (44 mg/kg) and xylazine (6 mg/kg) im]. All animal procedures were approved by the Committee for Animal Care and Use at the University of Connecticut Health Center and conformed to the guidelines for laboratory animal care and use published by the National Institutes for Health.

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The experimental procedures are nearly the same as those described in our companion paper (Sterbing et al. 2003). Here, we describe only those procedures specific to the present study.

**Background noise**

For some units, the response to the binaural beat was recorded in the presence of ambient background noise. A low-pass noise (20 kHz cutoff) was generated using a Grason-Stadler 455C (West Concord, MA) and further band-pass filtered (Krohn-Hite, Model 3550, Brockton, MA) between 200 and 2,000 Hz. The loudspeaker (ADS, L200, Wilmington, MA) was placed ~1 m directly over the rabbit's head. Measured at the position of the rabbit's head, the level of the noise used in the experiments ranged from 60 to 85 dB SPL. Because the custom-fitted ear molds provided an attenuation of ~55 dB between the two ears (100–2,100 Hz), the noise level at the eardrum was considerably less (Kuwada et al. 1987).

**Cross-correlation model**

A model of interaural cross-correlation with biologically based preprocessing (Bernstein and Trahiotis 1996; Bernstein et al. 1999) was implemented using the binaural cross-correlogram toolbox for MATLAB (courtesy of Dr. Michael Akeroyd, Laboratory of Experimental Psychology, University of Sussex). Tones with a fixed delay and modulated tones with the same delay were used in the simulations. Modulation frequencies between 20 and 50 Hz with carriers between 300 and 1,500 Hz were tested. The phase of modulation was the same in each ear, and the peak amplitude of the unmodulated and modulated signal was kept constant. The signal to each ear was the same in each ear, and the peak amplitude of the unmodulated and modulated signals, squares them, and then low-pass filters each at 2 kHz. Binaural processing was simulated by taking the cross-product of the left and right signals. The delay dimension covered ±1 ms.

**RESULTS**

**Analysis of the modulated and unmodulated signals**

We first asked if the different neuronal types (enhanced or suppressed under stimulation with modulation) could be due to an altered coincidence detection mechanism created by differences in the interaural correlation of the signals. We examined this possibility within a model (Bernstein and Trahiotis 1996; Bernstein et al. 1999) and found that the interaural correlation with and without modulation was identical. Thus from a signal-processing viewpoint, these two signals provided the same ITD information. This is in agreement with the location of the peak and the shape of the IC population response for the two signals (Sterbing et al. 2003). However, this does not predict the overall increase in rate of the population response. Furthermore, the model does not address the extreme changes in the behavior of individual units that led to the net increase in rate of the population response.

Next we asked whether these neuronal types were a consequence of the differences in the energy of the unmodulated and modulated signals. For enhanced neurons, the greater response to modulation cannot be due to the energy in the signal because it has less energy than the unmodulated condition by ~4.2 dB. For suppressed neurons, the reduction in the response to modulation could be due to the reduced energy in the modulated signal. However, if we presented the unmodulated signal with less energy than the modulated signal, responses remained suppressed (n = 9). Figure 1 illustrates an example of a neuron that was suppressed for the binaural beat with modulation (---) compared with the binaural beat without modulation (—) with both signals presented at the same peak level. To test if the suppression was due to less energy in the modulated condition, we recorded the response to the unmodulated beat with a peak level 10 dB less than the modulated condition. The response (· · ·) was still greater than the response to the modulated binaural beat (···). For neither the enhanced neurons nor the suppressed neurons did the difference in energy explain the changes in response with the addition of modulation to the binaural beat.

To determine the contributions of the spectral components of the modulated signal, we tested in a subset of neurons (n = 19) their response to the carrier, lower sideband, and upper sideband and compared their sum to the response to the modulated binaural beat. For example, a neuron that was tested with a carrier of 900 Hz and AM of 23 Hz was also tested separately with tonal binaural beats at 877, 900, and 923 Hz. Four examples of this analysis are illustrated in Fig. 2 for two peak-type neurons, one that enhanced (Fig. 2A) and the other that suppressed (Fig. 2B) their responses to modulation and similarly for two trough-type neurons (Fig. 2, C and D). In Fig. 2, left, the ITD functions to the modulated signal and, separately, those for the three components of the modulated signal are plotted. The ITD function to the modulated signal and the sum of the functions for the three components is replotted in Fig. 2, right. Note that for one suppressed neuron (Fig. 2D), the...
y scale is doubled (right) to incorporate the summed response. In general, for enhanced neurons (Fig. 2, A and C), the summed responses to the three components of the modulated signal were below that to the modulated signal, and the reverse was true for the suppressed neurons (Fig. 2, B and D). In our sample, in no case was the peak response to the modulated signal explainable by addition of the responses to the carrier and sidebands. Thus we conclude that the differences in the responses are not due to the simple addition of the responses to the frequency components of the modulated signal. This is expected as the sidebands are most likely not independently resolved with low modulation frequencies because they are located within the hypothetical critical bands of the cochlea (for review, see Scharf 1970).

The differences between the responses to the unmodulated and modulated binaural beat should become smaller as the two stimuli become more similar to each other. We manipulated the similarity of the conditions by systematically decreasing modulation depth. Decreasing modulation depth decreases the amplitude of the sidebands such that at 0% modulation depth the signal is identical to the unmodulated signal. Figure 3 illustrates the effects of modulation depth on an enhanced and a suppressed neuron. The response of the enhanced neuron (Fig. 3A) was nearly zero for the unmodulated condition and similarly so at shallow depths. However, the response steadily increased as the depth approached 100%. In contrast, for the suppressed neuron (Fig. 3B), the response was robust for the unmodulated binaural beat and similarly so at shallow depths. However, the response steadily decreased as the depth approached 100%. Figure 4 plots the normalized peak response versus modulation depth for all the enhanced (Fig. 4A) and suppressed (Fig. 4B) neurons in our sample (n = 9). Although the slopes vary, for most neurons, the response is graded with modulation depth.
Background noise produces similar effects to modulation

The differences in response to the unmodulated and modulated signals may be due to less adaptation in the modulated condition, i.e., the responses to modulated tones are more robust and sustained than those to tones. An example of this is presented in Fig. 5, where the response to a monaural, unmodulated tone (Fig. 5A) is basically an onset response, whereas that to a monaural, modulated tone is robust and sustained. So for enhanced neurons, modulation may simply increase sustained excitation, and for suppressed neurons, modulation may increase sustained inhibition.

We tested this idea by recording the responses to the binaural beat with and without background noise. To an unmodulated binaural beat, noise should increase the response for an enhanced neuron and decrease the response for a suppressed neuron. Figure 6 illustrates the responses in background noise for an enhanced and a suppressed neuron. For the first neuron (Fig. 6, A–C), modulation evoked a stronger response than the unmodulated signal, thus fitting our definition of enhancement. When the unmodulated signal was presented in noise (Fig. 6C), the response resembled in magnitude the response to the modulated signal (Fig. 6B). For the suppressed neuron (Fig. 6, D–F), the unmodulated response in noise (Fig. 6F) again resembled the response to the modulated signal (Fig. 6E). Thus consistent with our adaptation hypothesis, for both types of neurons, background noise made the unmodulated response similar to the modulated response. We tested 18 of the 272 neurons in background noise and 13 fit the adaptation hypothesis.

Are the effects of modulation dependent on which ear receives modulation?

If enhancement or suppression is due to an adaptive mechanism of the inputs to the IC, then only modulating the signal to one ear should decrease the enhancement or suppression seen when both ears receive modulation. Moreover, if adaptation is equal for both sides, then adding the responses to modulation to each side alone should combine to approximate the binaurally modulated response. To investigate this idea, we...
measured the responses to the binaural beat when both ears received modulation (our standard condition) and compared them to the responses to the binaural beat when the signal to only one ear or the other was modulated. In the subset of our sample that was tested \((n = 55)\), we found three major classes of neurons. Figure 7, A and B, illustrates examples of the first class of neurons we found: those that fit the scheme that adaptation effects are additive across the inputs from each ear. In the enhanced neuron (Fig. 7A), modulation to either ear alone decreased the response rate compared with the binaurally modulated condition. Consistent with our scheme, summing the responses very closely approximated the response when modulation was presented to both ears. Similarly, for the suppressed neuron (Fig. 7B), modulation to either ear alone increased the response rate compared with when the modulation was presented to both ears. Summing the degree of suppression when only either ear was modulated approximated the response to the binaurally modulated condition.

For other neurons, the response to modulating the contralateral ear alone or modulating either ear alone approximated the response to the binaurally modulated condition. Figure 7C shows a neuronal response that represents the second class of neurons we found: where modulation to only the contralateral ear evoked a similar response as the binaural modulation. Furthermore, modulation to only the ipsilateral ear had little, if any, effect and resembled the response to the unmodulated signal. Similarly, for the suppressed neuron (Fig. 7D), modu-
translation to only the contralateral ear evoked a similar response as the binaural modulation. As in the enhanced neuron (Fig. 7C), modulation to only the ipsilateral ear again had little effect. The final class of enhanced and suppressed neurons were those where modulation to either ear alone evoked similar responses to the binaurally modulated condition. For both the enhanced (Fig. 7E) and suppressed neuron (Fig. 7F), modulation to either the contralateral or ipsilateral ear evoked responses comparable to those evoked by the binaurally modulated signal.

The distribution of the different classes of enhanced (n = 41) and suppressed (n = 14) neurons described in the preceding text is shown in Fig. 8. Combining enhanced and suppressed neurons, ~31% showed additive properties. Modulation to either ear alone approximated the response to the binaurally modulated signal in ~29% of the neurons. In ~33% of the neurons, modulation to the contralateral ear alone approximated the response to the binaurally modulated signal. For only 7% of the neurons, modulation of the signal to the ipsilateral ear only produced the same effect as binaural modulation.

Can the responses to monaural stimulation predict the effects of modulation?

Is the enhancement or suppression seen with the modulated binaural beat also reflected in a neuron’s response to a monaural signal that is modulated? To address this question, we compared the binaural beat responses of enhanced and suppressed neurons to their responses to unmodulated and modulated monaural signals (n = 16). Our first example (Fig. 9A) is a neuron that showed similar response rates to the unmodulated and modulated binaural beat. Here, the prediction is that the response to an unmodulated monaural tone would be similar to that of a modulated monaural tone. In this example, this prediction holds because the monaural responses are similar whether they were unmodulated or modulated. Our second example (Fig. 9B) is an enhanced neuron. There was no response to either unmodulated or modulated monaural tones delivered to the ipsilateral ear. Although there was also no response to an unmodulated tone delivered to the contralateral ear, a modulated signal to this ear evoked a robust sustained response. Thus the prediction here is that a binaural stimulus with modulation only to the contralateral ear should evoke an enhanced response and that modulation only to the ipsilateral ear should evoke a response similar to the unmodulated binaural beat. Both predictions hold because the response to the binaural beat with only contralateral modulation is greater than the response to the unmodulated binaural beat and the binaural beat with

![Graph showing the distribution of neuron types](image)
modulation only to the ipsilateral ear approximates that to the unmodulated binaural beat. What is unresolved is why when both ears are modulated, the response is greater than that when only the contralateral ear is modulated. Ipsilateral modulation has an enhancing effect when combined with contralateral modulation and yet ipsilateral modulation by itself evokes little, if any, response.

Figure 10 provides examples of the poor relationship between monaural and binaural response properties. For the enhanced neuron in Fig. 10A, the monaural responses, whether unmodulated or modulated, were extremely weak, at best. This fits with the result that modulating the signal to either ear alone within the binaural beat evoked responses similar to the unmodulated binaural beat. However, the monaural responses poorly predict the large enhancement to the modulated binaural beat. Thus overall, the monaural responses are a poor predictor of the binaural response. For another enhanced neuron (Fig. 10B), the monaural/binaural relationship is different from the enhanced neuron in A. Here, the monaural responses display similar profiles with no obvious differences for the unmodulated and modulated signals. Yet when modulated separately within the binaural beat, the responses approximate the enhanced response when both ears are modulated. Thus the monaural responses have no predictive power. A similar conclusion is reached for the suppressed neuron in Fig. 10C. Because the modulated monaural responses are greater than the unmodulated monaural responses, especially to contralateral stimulation, it would be expected that the response to the modulated binaural beat should be enhanced. In fact, independent of the side that is modulated, the response to the modulated binaural beat is suppressed.

The responses to monaural stimulation could often be used to predict the observed enhancement or suppression of the modulated binaural beat. For 8 of the 16 neurons, the responses to both the contralateral and ipsilateral monaural stimulation were helpful in predicting the binaural beat responses. For 4 of the 16, the response to contralateral monaural stimulation provided a partial prediction. For these units, the response to ipsilateral monaural stimulation did not help to predict the binaural beat response. For the other 4 of the 16 units, the

![Graphs illustrating the response properties of neurons under different stimulation conditions.](http://jn.physiology.org/)

**Fig. 10.** The ITD functions and histograms for 3 neurons whose response to monaural stimulation could not be used to explain the response to binaural stimulation. Same format as Fig. 9. **A:** a neuron that showed enhancement with modulation. **B:** another neuron that showed enhancement with modulation. **C:** a neuron that showed suppression with modulation.
monaural responses could not be used to predict the change in ITD response.

**DISCUSSION**

In this study, we examined potential mechanisms underlying the enhancing and suppressing effects of modulation on ITD sensitive neurons in the IC. Physical differences in the modulated and unmodulated signals, including the interaural correlation, energy, and spectrum, did not provide clear answers. Our results are consistent with adaptive mechanisms. The question then becomes where is this adaptation occurring and what is the anatomical basis for this adaptive circuitry.

**Adaptive mechanisms**

A primary question is whether the effects of AM are processed at the initial site of binaural interaction [viz., the superior olivary complex (SOC)] and are simply mirrored by their targets in the IC or if these effects are created at higher centers. First, it appears that modulation does not appreciably alter the adaptation of auditory nerve fibers (AN) and primary-like neurons in the cochlear nucleus (CN). At the level of the AN, it appears that the peak rate would remain constant for unmodulated and modulated tones if the signals were equated for peak level, as in the present study (Joris and Yin 1992). Modulation of primary-like cells in the CN evokes responses similar to those of the AN (Frisina et al. 1990; Rhode and Greenberg 1994). Neurons with primary-like responses are strongly correlated with bushy cells (Rhode and Smith 1986), and it is these cells that provide the inputs to the SOC (Cant and Morest 1984).

At the medial (MSO) and lateral (LSO) superior olive, sensitivity to ITDs in low-frequency signals is brought about by the convergence of action potentials phase-locked to the carrier frequency of the signals to each ear (for review, see Kuwada et al. 1997). Logically, if the signal is amplitude modulated, the number of phase-locked action potentials to the carrier would decrease because, for some number of cycles of the carrier, the amplitude of the signal is below threshold. Consequently, there are fewer chances for coincidence and the output of the coincidence detector would be decreased relative to an unmodulated signal. This may, in part, explain our suppressed neurons. However, this mechanism cannot account for neurons that showed complete or nearly complete suppression, nor can it account for the enhanced neurons.

The effect of modulation on low-frequency, ITD-sensitive neurons in the SOC has not been studied. Thus whether MSO and LSO neurons show differences to unmodulated and modulated stimuli, i.e., more or less adaptation than their inputs, is not known. However, comparisons between the MSO and the IC indicate that adaptation to dynamically varying ITDs is less pronounced in the MSO (Spitzer and Semple 1998). In summary, there is little, if any, evidence to suggest that robust enhancement or suppression is due to adaptive mechanisms at the level of the primary binaural comparators.

If the effects of modulation cannot be attributed to the primary binaural comparators, then adaptive mechanisms external to the binaural pathways may be involved. For example, a highly adaptive response to an unmodulated tone and a sustained, nonadaptive response to a modulated tone (e.g., Fig. 5) could explain enhancement and suppression. For enhancement, a sustained excitatory input created by a modulated tone would depolarize an IC neuron and amplify the signals from the binaural comparator (viz., MSO and LSO). Conversely, for suppression, a sustained inhibitory input would hyperpolarize an IC neuron and attenuate the binaural inputs.

It is not known which of the excitatory and inhibitory monaural inputs to the IC are adaptive to tones and relatively unadaptive to modulated tones. This is because the IC receives projections from both a broad class of cell type called stellates [ventral CN (VCN)] and from fusiform cells [dorsal CN (DCN)]. Stellate cells can display highly adaptive responses to tones (e.g., onset, onset-chop, transient-chop) but highly nonadaptive, phase-locked responses to modulated tones (Frisina et al. 1990; Rhode and Greenburg 1994). Even neurons that show little adaptation to tones (e.g., sustained chop from VCN and pause-build from DCN), can temporally encode the modulation envelope with high fidelity (Kim et al. 1990). These could be the sources of the highly adaptive response to tones yet robust responses to modulated tones found commonly in the IC (e.g., Fig. 5) (Krishna and Semple 2000; Langner and Schreiner 1988; Rees and Möller 1983; Rees and Palmer 1989). Similar inputs that project to the IC via the ventral nucleus of the lateral lemniscus (VNLL) may provide the sustained inhibitory input that could explain the suppression of ITD sensitivity seen in IC neurons to modulated sounds.

**Neural circuits underlying enhancement and suppression**

What are the potential sources of low-frequency inputs to the IC that could have these adaptive characteristics? Excitatory projections to the IC arise from the contralateral and ipsilateral CN (Oliver 1987). The contralateral projection arises from the DCN and VCN and distributes widely throughout the IC (Fig. 11, projections 1 and 2). In contrast, the ipsilateral projection arises mainly from the VCN and is confined to the low-frequency region of the IC (Fig. 11, projection 3). Interestingly, when matched for low frequency, these ipsilateral and contralateral projections are of approximately equal density (Oliver 1987).

The VNLL is a prime candidate for monaural inhibitory projections to the IC (Glendenning et al. 1981). This nucleus...
provides a glycinergic/GABAergic input (Riquelme et al. 2001) to the ipsilateral IC (Fig. 11, projection 4) and receives a widely distributed input from the contralateral VCN (Fig. 11, projection 5) and an input restricted to low frequencies from the ipsilateral VCN (Fig. 11, projection 6). The low-frequency part of the VNLL projects to the low-frequency part of the IC (Malmierca et al. 1998).

The inputs from the binaural comparators (MSO and LSO) and from monaural sources (VCN, DCN, VNLL) converge within the low-frequency part of the IC but do not completely overlap (Glendenning and Masterton 1983; Loftus et al. 2002; Oliver et al. 1997, 1999). This incomplete overlap is consistent with our finding that both peak- and trough-type neurons can be suppressed, enhanced, or not affected by modulation.

How do these neural circuits operate for different combinations of enhancement? Enhancement could be produced by the convergence of excitatory inputs from the binaural comparator and from the CN. For the additive type (Fig. 7A), each of the inputs from the contralateral and ipsilateral CN enhances the response and sum to approximate the binaural modulated response. Neurons, for which modulation to either ear evokes enhancement similar to that when both ears are modulated (Fig. 7E), suggest a ceiling effect. We would predict that if this was so, lowering the stimulus intensity would make these neurons more like the additive type. Finally, neurons, for which modulation to the ipsilateral ear had no effect and modulation to the contralateral ear evokes enhancement similar to that when both ears were modulated (Fig. 7C), are consistent with an excitatory projection from the contralateral CN.

Inhibitory monaural inputs outlined in Fig. 11 could explain the observed suppressive effects to modulation. In this scheme, inhibitory inputs from both CNs are conveyed to the IC through the ipsilateral VNLL. For the additive type (Fig. 7B), each of the inputs from the contralateral and ipsilateral CN via the VNLL suppresses the response and add to approximate the binaural modulated response. Neurons that are suppressed under modulation to either ear, similarly to when both ears are modulated (Fig. 7E), suggest a ceiling effect in the VNLL. We would predict that if this was so, lowering the stimulus intensity would make these neurons more like the suppressive additive type. Finally, neurons that were suppressed under modulation to the contralateral ear similarly to when both ears were modulated (Fig. 7D), and showed no change under modulation to the ipsilateral ear, are consistent with an inhibitory projection from the contralateral CN via the ipsilateral VNLL.

Finally, there were many neurons that did not change their response appreciably to the modulated signal. For these neurons, we predict that they receive predominantly binaural inputs.

Function of the enhancement and suppression

We have shown that there is a continuum of ITD-sensitive IC neurons from those that suppress to those that enhance their response to modulated sounds. Moreover, suppressed neurons show a more vigorous response than enhanced neurons to unmodulated sounds. Thus these two populations could differentially sense modulation by independently decreasing and increasing their firing rate to a modulated signal. Moreover, as the depth of modulation decreased, enhanced neurons would decrease and suppressed neurons would increase their firing rates. Such polar mechanisms are postulated to enhance contrast in the visual and somatosensory systems (Kuffler 1953; Mountcastle and Powell 1959). In summary, a role for the excitatory and inhibitory monaural inputs onto ITD-sensitive neurons may be to detect the presence and degree of modulation in the sound source while preserving ITD information.

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DISCLOSURES

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