

Reconsidering binaural phenomena in terms of interaural neural fluctuation differences

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ABSTRACT

Binaural phenomena have been interpreted based on a variety of acoustical parameters, including interaural differences of time, level, and envelope. Models based on these parameters often include physiological transformations, such as peripheral filters, including nonlinear filter-based models. However, the transduction of the mechanical response of the cochlea into an electrical response is generally not included in binaural models, except for the bandwidth limitations imposed by the low-pass filtering of the IHC membrane filter. Inner-hair-cell (IHC) saturation and its interaction with basilar-membrane compression have a qualitative effect on the signals that provide the inputs to the binaural nervous system. Saturation of the IHC voltage strongly shapes the responses to stimulus parameters that are conveyed to the central nervous system. IHC saturation influences the temporal pattern of auditory-nerve (AN) responses, and in particular affects the amplitude of low-frequency fluctuations in AN rate functions in response to complex sounds. Here, we re-examine some classical binaural phenomena in terms of the interaural differences of neural fluctuation amplitudes of AN rate functions. We also examine response profiles across populations of model midbrain neurons that are sensitive to both neural fluctuation frequency and interaural differences.

Keywords: Interaural Differences, Binaural Detection, Physiological Modeling, Computational Modeling

1. INTRODUCTION

Neural models for binaural detection and sound localization typically focus on sensitivity to interaural differences of time (ITDs) and level (ILDs) and spectral cues for elevation. Based on psychophysical and physiological responses to tones, the neural coding of ITDs is generally attributed to the cells in the medial superior olive (MSO) and coding of ITDs to cells in the lateral superior olive (LSO) (1). Cells in the MSO receive excitatory inputs from both ears, and they also receive inhibitory inputs from both ears via the medial and lateral nuclei of the trapezoid body, providing the substrate for more complex processing than straightforward excitatory-excitatory (EE) processing (2). Physiological recordings from LSO cells that receive excitation from the ipsilateral ear and inhibition from the contralateral ear support models for excitatory-inhibitory (IE) mechanisms; however, the LSO and MNTB are relatively small in humans as compared species typically used in physiological studies (3-6).

Because of the difficulty of recording in the lower brainstem, most *in vivo* binaural physiology are from the midbrain, in the inferior colliculus (IC) in mammals, rather than in the lower brainstem. Most IC recordings have been based on manipulations of ITDs and ILDs of tones or interaural correlations of noise (e.g. 7-9). The ITD and ILD sensitivity of IC cells is often assumed to be “inherited” from the MSO and LSO, although the fact that IC cells receive both excitatory and inhibitory inputs from other brainstem nuclei. These inputs include excitatory projections from predominantly monaural nuclei such as the anteroventral cochlear nucleus (AVCN), and inhibitory projections (via the ventral nucleus of the lateral lemniscus) from octopus cells in the posteroventral cochlear nucleus (10). Thus, EI interactions that occur at the level of the IC provide a potential basis for binaural sensitivity.

IC neurons that are sensitive to interaural differences are generally also sensitive to changes in the fluctuations of their inputs, as often illustrated by tuning to amplitude-modulated (AM) stimuli (11). AM tuning can generally be elicited by either diotic stimuli or by monaural stimuli presented to the contralateral ear, thus the mechanisms for AM tuning must not be dependent upon binaural inputs. Because the IC is the first level of the ascending pathway where strong rate-based tuning to AM is observed, it is also clear that this tuning is not “inherited” from the inputs to the IC. Several models for

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AM tuning have been proposed (12); these models generally depend upon interactions between excitatory and inhibitory inputs to the IC (13). Interactions between ITD and AM stimulus manipulations have previously been demonstrated (14-15). Here we consider evidence that these excitatory-inhibitory interactions, in addition to EE mechanisms, may be an important component in understanding binaural detection phenomena.

One strategy for understanding binaural detection has been to take advantage of differences in performance of psychophysical tasks across different stimulus waveforms, e.g. differences in binaural detection across reproducible ('frozen') tokens of gaussian noise. Models based on interaural cross-correlations, which can explain mean thresholds for many low-frequency binaural tasks (e.g. 16-17). These models are challenged by results from "molecular" level psychophysical results (18), such as detection in reproducible-noise (19-22). Detailed detection performance across ensembles of reproducible maskers, for both diotic and dichotic 500-Hz tone-in-noise detection, is best explained by envelope cues in the form of interaural envelope differences (IEDs) (23-25). In contrast, these detailed reproducible-noise datasets are not well explained by models based on interaural time differences (21, 24), which are generally assumed to be important for binaural detection of low-frequency tones. ILD-related cues, and even optimal linear combinations of ITD and ILD cues, are not as successful as the IED cue in explaining detailed binaural detection of 500-Hz tones in wideband or narrowband gaussian noise (24).

The IED result makes sense given the EI interactions that occur at several stages of the auditory pathway, and the fact that starting in the periphery, auditory neurons encode the envelope of complex sounds, including noise. Analysis of IEDs reveals that they are a nonlinear combination of ITD and ILD (24). However, IEDs can be more simply understood as a simple EI interaction between neural inputs that follow the envelope of the stimulus. The fact that the auditory CNS is particularly sensitive to slow fluctuations in stimulus envelope provides the motivation to pursue a better understanding of this representation.

Here, we go beyond IEDs to consider interaural differences of neural fluctuations (IDNFs), the low-frequency fluctuations in the responses of AN fibers, as opposed to envelope fluctuations in the stimuli. Cochlear filtering creates relatively large, low-frequency fluctuations in the mechanical response of the inner ear. This response is then transduced by the inner hair cells (IHCs) into a voltage that drives release of neurotransmitter and excitation of AN fibers. The transduction process involves a nonlinear function that slowly saturates over a range of moderate sound levels (26-28). This saturating nonlinearity has a strong effect on the amplitudes of response fluctuations. In general, in response to complex sounds with nonuniform spectra, CFs near spectral peaks will have responses that are more saturated than fibers tuned away from the peaks.

Neural fluctuations are not simply related to stimulus energy – a tone, or for example a harmonic in a complex tone or a harmonic near a spectral peak of a voiced sound, or any narrowband stimulus component near CF, will "capture" the response of the IHC (29-30, review 31). Capture of the AN response by a single harmonic results in a response that is dominated by one frequency component, and thus the envelope of the post-stimulus-time-histogram (PSTH) of the response is "flat," resembling that of a response to a pure tone. In contrast, the PSTH of AN responses for fibers tuned away from harmonics lock onto amplitude fluctuations caused by beating between stimulus components. An important point here is that the "neural fluctuations" in the AN responses do not simply reflect the stimulus envelope. Neural fluctuations are strongly shaped by the IHC transduction nonlinearity, and therefore are also affected by cochlear amplification (and potentially by gain control) that determines the operating point of this nonlinearity.

Neural fluctuations are of particular interest for coding of complex sounds, including binaural stimuli, because IC cells with binaural sensitivity are typically also sensitive to "AM," i.e. to the fluctuation amplitudes of their neural inputs. The goal of this paper is to explore whether binaural models that are sensitive to neural fluctuations can also explain basic IC physiological properties, such as phase-sensitivity. The focus is on IC cells with characteristic frequency near 500 Hz, for which we have reproducible-noise data; however, the same principles and general model structure can be applied to higher CF cells that have stronger ILD sensitivity and weak ITD sensitivity.

2. METHODS

The peripheral model responses are from the Zilany et al., (32) AN model. IC model cells are based on the strategy of Krips and Furst (33), with coincidence-detecting neurons that receive excitatory and inhibitory inputs. Responses of four model structures are illustrated here: IC Model 1) The simplest

model had the structure of the same-frequency inhibitory-excitatory model for AM tuning in the IC (SFIE, 13), receiving contralateral excitation and contralateral inhibition. This model is AM sensitive, but of course does not have any sensitivity to binaural differences. IC Model 2) Simply adding inhibitory inputs that are driven by the ipsilateral ear yields a cell with AM tuning and sensitivity to ITD and ILD. IC Model 3) The third model considered here has an EE stage that receives excitatory inputs driven by both ears. IC Model 4) Finally, we combine an EE stage with inhibitory inputs from the contralateral sides.

The ITD and ILD sensitivity of each model was evaluated, along with its ability to predict the variance across frozen noise waveforms, i.e. the average detection patterns across subjects, for a 500-Hz tone in wideband (100-3000 Hz) gaussian noises in the NoSo (diotic noise and tone) and NoSpi (diotic noise, with tone that is inverted to one ear) configurations (22). The difference in threshold between these two stimulus configurations (34) is referred to as the binaural masking level difference (BMLD).

Model parameters were not adjusted to fit detailed response properties of individual neurons. Instead, the model structure was of interest, and the general trends in the different binaural response sensitivities, as well as the ability of each model to explain the BMLD data. The coincidence windows used for the EE stages was 0.5 ms. The CFs of all peripheral inputs to the model were tuned to 500 Hz. Inhibitory inputs were delayed by 2 ms. A single input, driven by the AN model, was used for each excitatory input (intermediate stages representing the cochlear nucleus were not included in this preliminary model.) After the EE stage, inhibitory inputs from one or both ears converge on the IC cell. The coincidence window over which an inhibitory inputs suppressed the cell's response was 0.5 ms. The total number of inhibitory inputs, when included, was 20; when the inhibition arose from both ipsilateral and contralateral sides, 10 inputs from each side were included.

3. RESULTS: MODEL RESPONSES

IC Model 1: This model is an implementation using a Krips & Furst (33) EI neuron of the SFIE model for AM tuning in the IC (13), receiving contralateral excitation and contralateral inhibition. This model is AM sensitive, but of course does not have any sensitivity to binaural differences. As a result, the ITD and ILD curves are flat (Fig. 1).

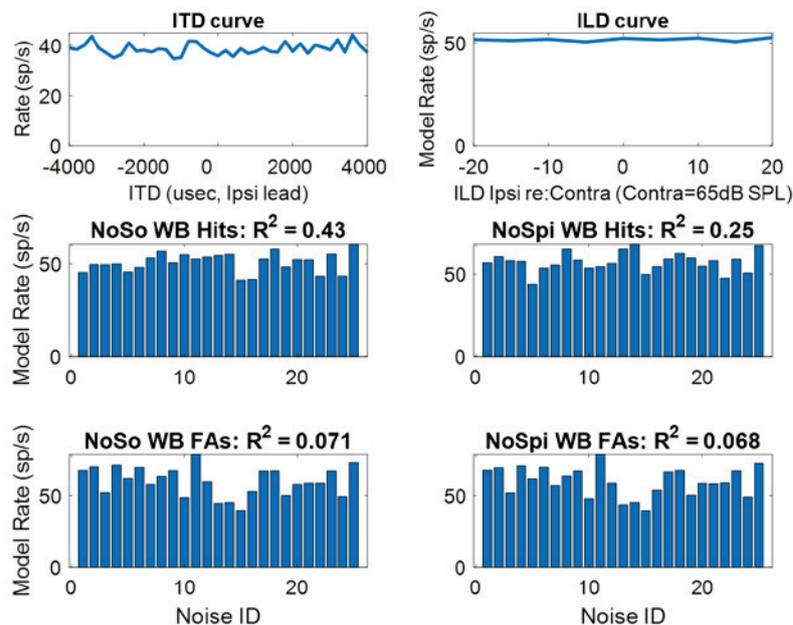


Figure 1 – Responses of IC Model 1. The top two panels show IC Model average discharge rate (spikes/sec) as a function of ITD and ILD. The bottom 4 panels show IC Model rate in response to each of the 25 noises in the ensemble of reproducible noises from Evilsizer et al. (22). The proportion of the variance in the average human listener's detection pattern (hits or false alarms for each noises waveform) that is explained by the model rate profile is indicated in the title above each panel.

The model predicts a substantial amount of the variance in the NoSo detection pattern for hits, as expected (23). The fact that this monaural model predicts some of the variance in the NoSpi detection pattern is due to the correlation of the envelope cues between the NoSo and NoSpi configurations.

IC Model 2: This model is the same as Model 1, except that half of the inhibitory inputs are driven by the ipsilateral ear, and half by the contralateral ear. This binaural model has phase sensitivity, shown in the cyclic ITD curve. This model also predicts the NoSo detection results, with similar quality as Model 1. However, it does not predict the detection pattern for the NoSpi detection, suggesting that a model excited by the contralateral ear and inhibited by both ears cannot describe binaural detection, despite having reasonable ITD sensitivity.

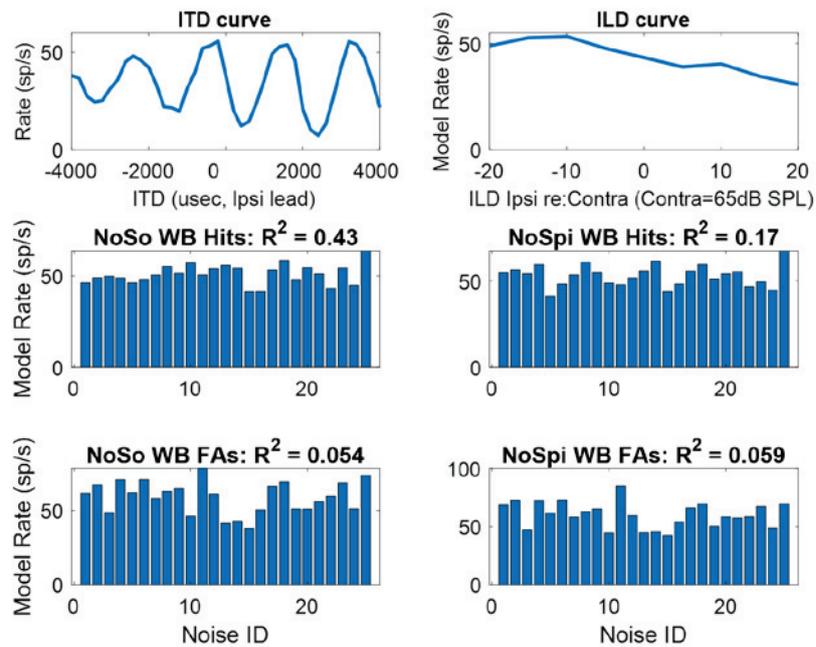


Figure 2 – Responses of IC Model 2. Same format as Fig. 1.

IC Model 3: The third model considered here has an EE stage that receives excitatory inputs driven by both ears. This model received no inhibitory inputs, and is thus most closely related to a classical EE cross-correlation-based model. Although the model has strong phase sensitivity, as shown by the cyclic ITD curve, it cannot predict the differences in detection performance across reproducible waveforms for either NoSo or NoSpi stimulus configurations.

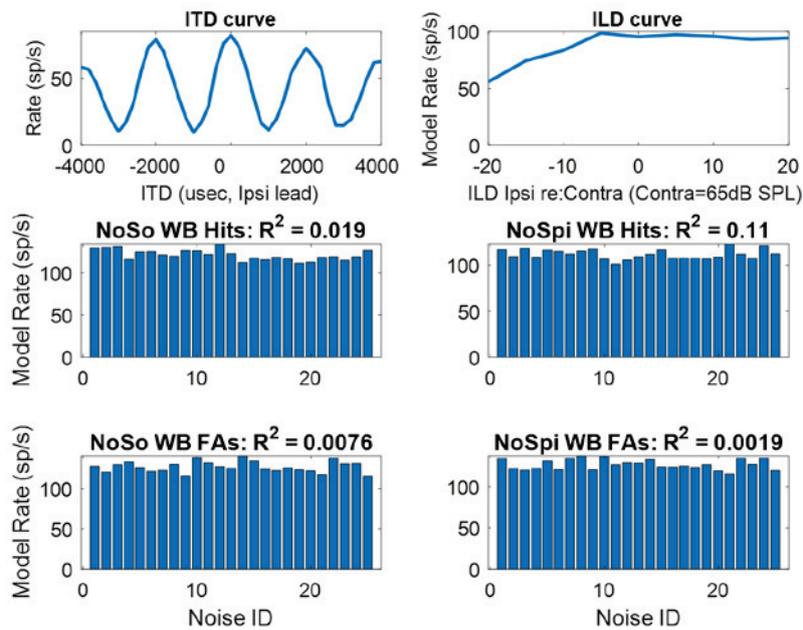


Figure 3 – Responses of IC Model 3. Same format as Fig. 1.

IC Model 4: Finally, we combine an EE stage with inhibitory inputs from the contralateral side. This model has both ITD and ILD sensitivity, and also explains substantial variance in both NoSo and NoSpi detection patterns. (Note that a similar model configuration, but with half of the inhibition from contralateral and half from the ipsilateral side failed to predict the NoSpi detection pattern. Not shown.)

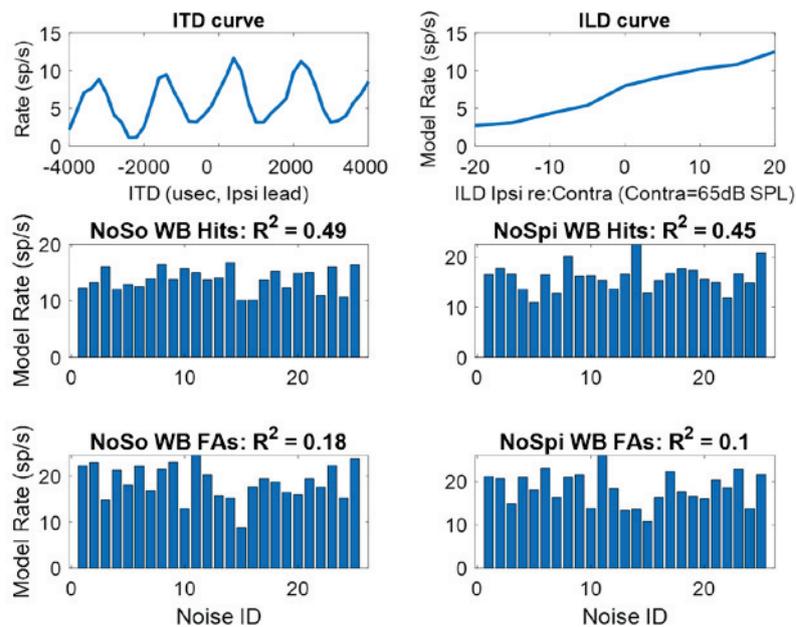


Figure 4 – Responses of IC Model 4. Same format as Fig. 1.

4. CONCLUSIONS

This study focused on several basic midbrain model configurations and their ability to explain both fundamental physiological measures of binaural sensitivity, such as ITD and ILD curves, and the detailed pattern of hits and false-alarms rates in a BMLD task using reproducible noise waveforms. One motivation for modeling the 500-Hz BMLD is that performance in this task is predictive of the binaural intelligibility level difference (35). Additionally, we had previously determined that envelope-related cues, not temporal fine structure, per se, was most important for describing binaural detection (24); therefore, combining envelope sensitivity and binaural sensitivity into a single model is a critical goal for explaining binaural processing at the level of the midbrain. The results here suggest that a minimal model, including only an EE stage with matched CF inputs at 500 Hz, and with additional contralateral inhibition, was able to predict several salient features of binaural sensitivity and both NoSo and NoSpi detection patterns.

Our approach takes advantage of the modeling structure proposed by Krips & Furst (33) which has the benefit of a straightforward implementation, given the availability of peripheral model response in the form of the average arrival rates of non-homogeneous Poisson processes. Another benefit of this approach is that the model responses are also non-homogeneous Poisson processes, and can therefore be used in future studies to directly predict psychophysical thresholds based on model cell responses properties.

ACKNOWLEDGEMENTS

Supported by NIH-R01-DC010813.

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