

Poster presentation

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Role of plasticity in coincidence detection in the avian auditory brainstem

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Introduction

Interaural time difference (ITD) is the difference in the time of arrival of sounds to both the ears, used by birds and mammals as cues for locating the sound source. Nucleus magnocellularis (NM), one of the major neurons in the cochlear nucleus of the auditory brainstem, sends depressing excitatory synapses onto the NL neuron which acts as a coincidence detector. Coincidence detection means a NL neuron has a higher firing rate, when it receives simultaneous inputs from the NM neurons of both the left and right ear i.e. without any phase delay. Cook et al [1] have shown that the depression from NM to NL enhances coincidence detection among NL neurons. Another factor which affects coincidence detection is the inhibition from SON (superior olivary nucleus) onto the NM neurons. SON is another group of neurons in the auditory brainstem. Coincident firing of many NM fibers from any one side alone can evoke firing of NL neurons thus creating ambiguity among NL neurons in being able to discriminate between binaural coincidences from strong monaural excitation. It has been shown by Rubel et al [2] that the SON play a vital role in controlling the dynamics of the NM neurons when biased inputs are presented to either group of the NM neurons since SON inhibition is proportional to the strength of NM neurons. Thus an increased activity of NM neurons on one side recruits stronger inhibition on that side which eventually reduces NM activity.

Here we develop mathematical models to show how both NM depression and SON inhibition contribute towards enhancing coincidence detection. We constructed a firing

rate model for the NL neurons and showed that the NM depression makes the NL firing rate more phase dependent and less frequency dependent. These results are consistent with the experiment in [1]. Since the timing of these circuits is critical we also constructed a spiking model using integrate and fire neurons to describe the NM and NL activity. The analysis of this model gave us a feasible parameter range, namely the time constant of depression, the extent of depression per spike and synaptic conductance in order to obtain a range of NM frequencies at which the NL firing rate is more phase dependent. We also modeled the SON cell with a facilitating inhibition to the NM neurons. We were able to show that SON plays a role in eliminating ambiguity among the NL neurons during biased inputs to one side of the brain. This implies the potential of an alternate mechanism to make the NL firing rate frequency independent in the presence of input bias.

References

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