59.14

A MODEL OF DIRECTION SELECTIVITY IN MACAQUE V1 BASED ON THE MORPHOLOGY OF THE MEYNERT CELL.

R. P. N. Rao[1]*, T. J. Sejnowski[1], and M. S. Livingstone[2]. [1]Computational Neurobiology Lab, The Salk Institute, 10010 N. Torrey Pines Road, La Jolla, CA 92037 and [2]Department of Neurobiology, Harvard Medical School, Boston, MA 02115.

A recent physiological study has suggested a relatively simple model for the genesis of directional selectivity in V1 of alert fixating macaque monkeys (Neuron, Vol. 20, 1998). In this model, which is inspired by the morphology of Meynert neurons in layers 5/6, the inhibitory inputs to a direction-selective cell are assumed to be located primarily on or near the soma while the excitatory inputs are assumed to be located mainly on the elongated basal dendrite(s) of the cell. We have constructed a compartmental model based on the structure of a Meynert cell described by Valverde (Cerebral Cortex, Vol. 3, 1985, p. 244). The model neuron included a low density of Na^+ channels in the soma, basal and apical dendrites and a higher density in the axon. The axon and soma also contained fast K^+ channels, which were excluded from the dendrites. The dendrites contained high voltage-activated Ca^{2+} channels and slow calcium-dependent and voltage-dependent K^+ channels. Inputs to the soma were inhibitory, involving both fast $GABA_A$ and slower $GABA_B$ synapses, while inputs along the long basal dendrite were excitatory, involving AMPA and NMDA synapses. When stimulated, the model neuron generated direction selective responses, the preferred direction of stimulation being from the distal end of the dendrite towards the soma. Removal of $GABA_B$ synapses resulted in the loss of direction selectivity, suggesting a role for these synapses in mediating the delayed inhibition required by the model. Our current efforts are focused on investigating the role of mechanisms such as synaptic depression in explaining the shifting excitatory response time course exhibited by some direction-selective neurons.

Supported by the Sloan Foundation, HHMI and NIH grant RO1 EY10203.