

## 25.001 Gain control mechanisms on a computational model of the retinal rod-driven ON bipolar

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### Resumo

**Introduction:** The retina grows predominantly by stretch and neuronal hypertrophy. In the outer retina, rod-driven ON bipolar cells (ON rBCs) extend their dendrites laterally and increase the number of contacted rods as the eye grows. This addition of membrane area and synaptic contacts may significantly impact the integration of rod signals and, in consequence, scotopic vision in general.

**Objectives:** To study the potential effects of neuronal growth and increases in rod convergence on the response properties of ON rBCs in the presence and absence of a local active conductance located at the dendritic tips of these neurons.

**Methodology:** An ON rBC was modeled with the NEURON simulation environment; biophysical and morphological data were gathered from literature. The model cell was synaptically connected to 24 through 240 model rods by means of a sigmoidal transfer function in order to study the effect of increasing synaptic convergence onto ON rBC response properties. Membrane potential changes at the ON rBC soma were then recorded for hyperpolarization of the model rods with voltage steps. To analyze the effects of neuronal growth onto the integration of rod-driven signals, the length of the ONrBC primary dendrites was increased from 2.5 to 25  $\mu\text{m}$  for each convergence. Finally, a delayed-rectifier voltage-gated potassium conductance ( $I_{KV}$ ) was inserted on the dendritic tips of the ON rBC, and its effects on rod-driven responses were studied for all dendritic lengths and degrees of rod convergence.

**Results:** Without  $I_{KV}$ , a 10-fold increase in rod convergence depolarizes the ON rBC resting membrane potential ( $V_{rest}$ ) by 31-35 mV, whereas increasing its dendritic length 10 times hyperpolarizes  $V_{rest}$  by 5-9 mV. These changes in  $V_{rest}$  impact rod-driven responses in ON rBCs: dendritic growth increases response amplitudes, whereas larger rod convergences lead to smaller response amplitudes. Synaptogenesis, but not dendritic growth, increased the gain of the rod:ONrBC synapse by steepening the slope of the rod:ONrBC input/output relation. This is analogous to an increase in sensitivity in real cells. In terms of response speed, dendritic growth delayed response peak time, whereas increases in convergence sped up time-to-peak. The insertion of  $I_{KV}$  on the dendrites of the model ON rBC hyperpolarized  $V_{rest}$  and stabilized response amplitudes for all dendritic lengths and rod convergences. Activation of  $I_{KV}$  during ON rBC responses also sped up and stabilized time-to-peak. Finally,  $I_{KV}$  prevented the gain of the rod:ONrBC input/output relation to change for all dendritic lengths and degrees of convergence tested.

**Conclusion:** These results suggest that (a) both dendritic growth and changes in synaptic convergence can alter ON rBC response amplitudes, speed and sensitivity significantly, (b) there is a causal relationship between synaptic convergence and sensitivity in the absence of gain control mechanisms, and (c)  $I_{KV}$  is a candidate for a local gain control mechanism. Dendritic  $I_{KV}$  may allow real ON rBCs to transmit a stable and coherent message to their synaptic counterparts throughout growth.

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**Palavras-chaves:** gain control, retina, vision