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Modelling the role of a sustained chloride current in rod bipolar cell sensitization

Dim light vision relies on rod bipolar cells to relay light information from rod photoreceptors to downstream neurons in the retina. Rod bipolar cells release glutamate in proportion to their photoreceptor response-driven depolarization, but this depolarization can also be modulated by the activity of retinal interneurons. Blocking or eliminating rod bipolar cell $GABA_C$ receptors, a specialized GABA-sensitive but non-inactivating chloride channel, drastically decreases rod bipolar cell responses and the overall sensitivity and operational range of the rod bipolar cells. We believe that this occurs due to tonic $GABA_C$-driven hyperpolarization of the rod bipolar cell resting potential. To test this hypothesis, we used a conductance-based model to simulate the responses of rod bipolar cells to flashes of light under various levels of background illumination with either no, variable, or maximal rod bipolar cell chloride conductance and compared this model to experimental electroretinography data from $GABA_C$ knockout mice, wildtype mice, or mice injected intravitreally with exogenous GABA. Our results indicate that chloride currents are used in addition to potassium currents for hyperpolarization of rod bipolar cell resting potential and provide clues for how this sustained $GABA_C$ input from retinal interneurons might be regulated in normal dim light vision.

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