Computational model of *Hermissenda* type-B photoreceptors. I. Ionic currents underlying changes in excitability produced by one-trial conditioning

Yidao Cai, Douglas A. Baxter and Terry Crow
Department of Neurobiology and Anatomy, University of Texas at Houston Medical School, Houston, TX 77030

### Introduction

The visual system of *Hermissenda* is an important model system for studying cellular and molecular mechanisms of one-trial and multi-trial Pavlovian conditioning. Membrane conductance in type-B photoreceptors has been characterized and progress has been made on examining the role of specific conductances in Pavlovian conditioning. Several models of *Hermissenda* photoreceptors have been developed (e.g. Sakakibara 1993; Fost and Clark 1996; Blackwell 1998). However, these models did not include all identified currents. For example, the inward rectifier current ($I_{K,A}$) was not included in previous models, but recent experimental data suggested that this current contributes to the resting membrane potential and the firing frequency. The aim of this study was to develop a computational model of *Hermissenda* photoreceptors using all identified currents. The first goal of this study is to build a more complete model of type-B photoreceptors using all identified currents.

### Methods

#### 1. The multi-compartment model

The model consists of a somatic compartment, three axonal compartments, one terminal compartment, and one microvillar compartment (see Fig. 1). The morphological parameters used in the model were derived from empirical data.

#### 2. I. Ion currents

In addition to the leakage current, there are a total of seven ion currents implemented in the model (see Fig. 1). The morphological parameters used in the model were derived from empirical data.

- **A-type K** current ($I_{K,A}$)
- **Hyperpolarization-activated inward rectifier current** ($I_{h}$)
- **Ca**-activated K** current ($I_{K,Ca}$)
- **Ca**-activation of A** current ($I_{A}$)
- **K**, V** current ($I_{K,V}$)
- **Na** current ($I_{Na}$)

Wherever possible, the kinetic parameters of the ion channels used in the model were based on available data from type-B photoreceptors. Kinetic models were made to simulate the experimental data.

The Ca**-activated** K** channel was activated by $E_{Ca}$. The $E_{Ca}$ pool was based on a first-order diffusion model and each opening of the Ca channels contributes Ca** to the pool.

#### 3. Simulation

Simulations were performed using SNNAP, the Simulator for Neural Networks and Action Potentials (http://snnap.uth.tmc.edu/), on a PC running Linux or Windows operating system (see Abbreviations for 23.5.0).

To measure the excitability and changes produced by one-trial conditioning, we first held the membrane potential at -60 mV by injecting hyperpolarizing current, then applied a 0.2 nA depolarizing current. The excitability changes were calculated as the increase in spike elicited by the current.

### Results

#### Spontaneous and light-elicited responses of the model

The model consists of a somatic compartment, three axonal compartments, one terminal compartment, and one microvillar compartment. The first one (axon A) represents the section up to the axon hillock and is responsible for the generation of spikes.

### Discussion

1. We developed a model that is able to simulate dark-adapted spikes and responses to illumination.

2. We used the model to study current changes underlying changes in excitability produced by one-trial conditioning.

3. $I_{K,A}$ does not play a major role in the enhanced excitability produced by one-trial conditioning.

4. Modulation of $I_{Ca,S}$ leads to a decrease in mean spike frequency, but this effect is compensated by modulations of other currents.

Acknowledgments

This work was supported by P01 NS38310.

Fig. 2. Schematic representation of the multi-compartment model. The somatic compartment is in red, the two axonal compartments are in green, and the terminal compartment is in blue. The blue line represents the membrane potential of the type-B photoreceptor and the red line represents the activity of a single action potential.

Fig. 3. Currents underlying an action potential. A. The action potential in the soma and somatic axon. The somatic spike is small and brief. B. Changes in currents in the soma and somatic axon. C. Changes in currents in the somatic axon. D. Changes in currents in axon B. E. Changes in currents in axon C. F. Changes in currents in the terminal compartment. G. Changes in currents in the terminal compartment. H. Changes in currents in the terminal compartment. I. Changes in currents in the terminal compartment. J. Changes in currents in the terminal compartment.

Fig. 4. Summary of changes in mean spike frequency after several currents were modulated simultaneously. The number on top of each bar indicates the degree to which the conductance was changed. These combinations produced increases in spike frequency from 60% to 100%, similar to what was observed experimentally.

Fig. 5. Effects of $I_{Ca,S}$ and $I_{K,Ca}$ modulation on mean spike frequency.

Fig. 6. Summary of changes in mean spike frequency after individual currents were modulated. The number on top of each bar indicates the degree to which the conductance was changed. These combinations produced increases in spike frequency from 60% to 100%, similar to what was observed experimentally.

Fig. 7. Effects of $I_{Ca,S}$ and $I_{K,Ca}$ modulation on mean spike frequency. The $E_{Ca}$ is reduced by 25% and $g_{Ca,S}$ by 50%. In each panel, the $I_{Ca,S}$ was varied from left to right. The panels are: A. Control; B. $I_{Ca,S}$ reduced by 25%; C. $I_{Ca,S}$ reduced by 50%; D. $I_{Ca,S}$ reduced by 50%.