The Neuronal Basis of Direction Selectivity in Lobula Plate Tangential Cells

Timothy Melano and Charles M. Higgins

Biomedical Engineering Program / ARL Division of Neurobiology
The University of Arizona, Tucson

Abstract

Using a neuronally-based computational model of the fly's visual elementary motion detection (EMD) system, the effects of picrotoxin, a GABA receptor antagonist, were modeled to investigate the role of various GABAergic cells in direction selectivity. By comparing the results of our simulation of an anatomically correct model to previously published electrophysiological results, this study supports the hypothesis that EMD outputs integrated into tangential cells are weakly directional, although the tangential cells themselves respond to moving stimuli in a strongly directional manner.

Key words: dipteran Visual System, Elementary Motion Detection

1 Introduction

The Hassenstein-Reichardt (HR) correlation model, published in 1956 [1], has contributed greatly to the understanding of the optomotor response in insects. Many years after the development of the HR model, cells were discovered in the

Corresponding author: Timothy Melano, *tmelano@email.arizona.edu*.

lobula plate of the fly that were sensitive to wide-field motion stimuli and the electrical activity of which was well described by the HR model. Although the HR model mathematically describes the responses of these so-called *tangential cells*, it leaves the neural basis of this system open for investigation. Based on anatomical and electrophysiological studies, a neuronally-based model of the dipteran visual motion detection system has been developed [2]. This model, an attempt to understand the neural basis of elementary motion detection, computes small-field visual motion in two stages, a non-directional stage and a directional stage seen in the response of T5 bushy T-cells (see Figure 1a).

An important question that arises in motion detection is the method in which output from elementary motion detectors (EMDs) is integrated into lobula plate tangential cells (LPTCs) and whether direction selectivity arises presynaptically or within the dendrites of LPTCs. In 1996, a study conducted by Single *et al.* addressed this question by injecting picrotoxin (PTX) into the hemolymph after punctuating the lobula plate [3]. Picrotoxin has been shown to block inhibitory input to LPTCs and is an antagonist to GABA receptors [4]. The investigators conducted electrophysiological experiments and computer modeling studies and the results of the two were compared.

In the electrophysiological experiments, the activity of several tangential cells was recorded before and 10 minutes after the application of PTX (see Figure 2). Prior to the application of PTX, the LPTC depolarized when the visual stimulus moved in the preferred direction and hyperpolarized in the null direction. The change in cellular input resistance was negative and equal for both directions. After the application of PTX the LPTCs lost their directionality: the cells depolarized for both preferred and null directions. The change in cell input resistance was also different after the application of PTX and was greater in the preferred direction than in the null direction.

In the computer modeling, two types of EMDs were tested: weakly directional

EMDs and strongly directional EMDs. Weakly directional EMDs exhibited a strong response to visual motion only in the preferred direction while strongly directional EMDs exhibited a strong response of opposite polarity to motion in preferred and null directions. These two types of EMDs were altered in systematic ways to simulate the different possible effects of PTX on the motion response and membrane resistance of LPTCs. When the inhibitory synapses of weakly directional EMDs were blocked, the motion response and the change in membrane resistance were similar to the electrophysiological results after PTX was applied. Based on this the authors concluded that physiological EMDs are weakly directional and that directional selectivity is calculated largely in lobula plate tangential cells.

In this study, we simulated the possible effects of PTX on the neuronally based EMD model [2] in order to determine how the output of T5 cells are integrated at the level of LPTCs.

2 Methods

Simulations of the computational model were conducted using the *Matlab* package (The Mathworks, Natick, MA). The image input was 40 x 40 pixels and the visual system was composed of 20 x 20 photoreceptors and optic cartridges. The filters used to model cell activity were first order with time constants of 50 ms for the first high pass and low pass filters and 100 ms for the last low pass filter. A two-dimensional sinusoidal grating was used as visual input to the model. The possible effects of PTX were modeled by manipulating the equations that dictate the activity of cells that may be sensitive to this chemical (below).

The response of the simulated LPTC is represented by the spatial sum:

$$R_{LPTC} = \sum_{allEMDs} (pos(T5_L) - f \cdot pos(T5_R))$$

where $T5_L$ and $T5_R$ are the membrane potentials of the two T5 cells with the same preferred-null direction axis but with opposite preferred directions (see Figure 1b). The *pos* operator has a rectifying effect, i.e. it only passes the positive values. In control simulations f was set to 1. Inhibition to LPTCs was blocked by setting f to zero.

3 Results

T5 endings are almost certainly presynaptic to lobula plate tangential cells [5]. Each optic cartridge contains four strongly directional T5 cells, two of which are proposed to have opposite preferred-null directions along each of the two axes of the compound eye [6]. It seems likely that both T5 cells with the same orientation (but with opposite preferred directions) are integrated into an LPTC since T5 cells, located in the outer lobula, are believed to project only to the lobula plate. If both of the two T5 cells with the same orientation are integrated into an LPTC, one must be excitatory and the other inhibitory (perhaps through the action of an inhibitory interneuron) since otherwise their activity would cancel. Single *et al.* demonstrated that strong directionality in LPTCs is lost when inhibitory inputs to LPTCs are blocked (see Figure 2b). The only way to produce this effect is if the synapses of T5 cells onto LPTCs are rectifying (see Figure 1b).

It is possible that the application of PTX affected inhibitory synapses in the outer lobula as well as the nearby lobula plate. To investigate the possibility that inhibitory synapses inside the neuronally-based EMD model were modified in the study by Single *et al.*, we blocked inhibition in the computational model, all of which occurs at the same level in the outer lobula. If this inhibition is removed, the model responds the same to stimuli moving in any direction, which is inconsistent with the results of Single *et al.*, and thus we conclude that only inhibition at the level of the lobula plate was affected.

Using the model shown in Figure 1b, we simulated the integration of T5 cells into an LPTC. In Figure 3a, we show the response to preferred and null direction stimuli of an LPTC before simulated application of PTX, comparable to the electrophysiological results in Figure 2a. After inhibition in the model at the level of the lobula plate is removed, simulating the effect of PTX, the response shown in Figure 3b is obtained, in qualitative agreement with the electrophysiological results.

In addition, since changes in cellular input resistance are proportional to changes in channel conductivity, it is likely that the input resistance data of Single *et al.* are also qualitatively supported by the model. After removal of simulated inhibition, the only conductance change in the simulated LPTC is due to the excitatory input from a single T5 cell which responds more strongly in the preferred direction than in the null. Thus input resistance changes are larger in the preferred direction.

4 Discussion

In this study, the neuronal basis of signal integration into LPTCs was investigated. A model of T5 cell integration into LPTCs was proposed, and the effects of PTX on inhibition in the model were simulated. In the control case, where all synapses were unmodified, the mean response of the LPTC (Figure 3a) was qualitatively similar to the electrophysiological data in the study by Single *et al.* (Figure 2a). The cases where inhibition from T5 cells was blocked (Figure 3b) provided results similar to the electrophysiological results of Single *et al.* after PTX was introduced into the lobula plate (Figure 2b).

Electrophysiological data collected by Douglass and Strausfeld [7] shows that direction selectivity is computed presynaptic to LPTCs. Although the activity of T5 cells is strongly directional (i.e. depolarized in the preferred direction and hyperpolarized in the null) the rectification of the output of T5 cells in the model (Figure 1b) transforms the signal into a weakly directional one, in agreement with Single *et al.* The results of our study thus support both that strong directionality arises at the level of T5 cells, and that weakly directional activity (the rectified output of $T5$ cells) is integrated at the level of the lobula plate.

Acknowledgements

This work was supported by the Biomedical Engineering Program at the University of Arizona, and the University of Arizona Program in Applied Mathematics NSF IGERT for Biology, Mathematics and Physics Initiative.

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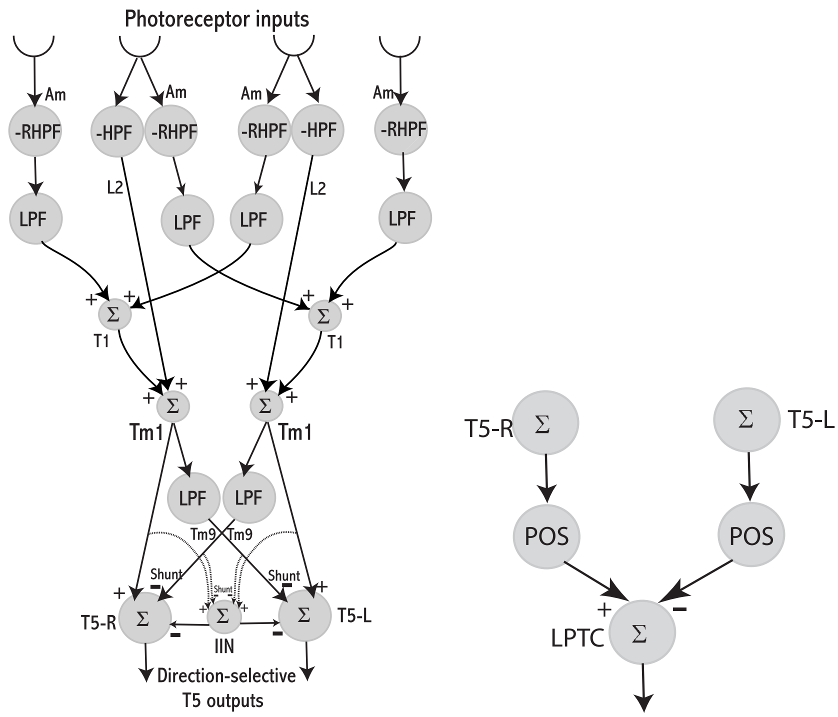
Figure captions

Figure 1: (a) Neuronally based model of EMDs in dipteran insects. This model is composed of photoreceptors, amacrine (Am) cells, lamina monopolar (L2) cells, basket T-cells (T1), Tm1 and Tm9 transmedullary cells, an inhibitory interneuron (IIN), and T5 bushy T-cells (T5-L and T5-R). RHPF denotes a relaxed high pass filter (allows a small component of a sustained output), HPF denotes a high pass filter, LPF a low pass filter, and $\sum$ a sum. (b) An illustration of the computational model of EMD integration into an LPTC. POS indicates a rectification (allows only the positive component of the signal to pass).

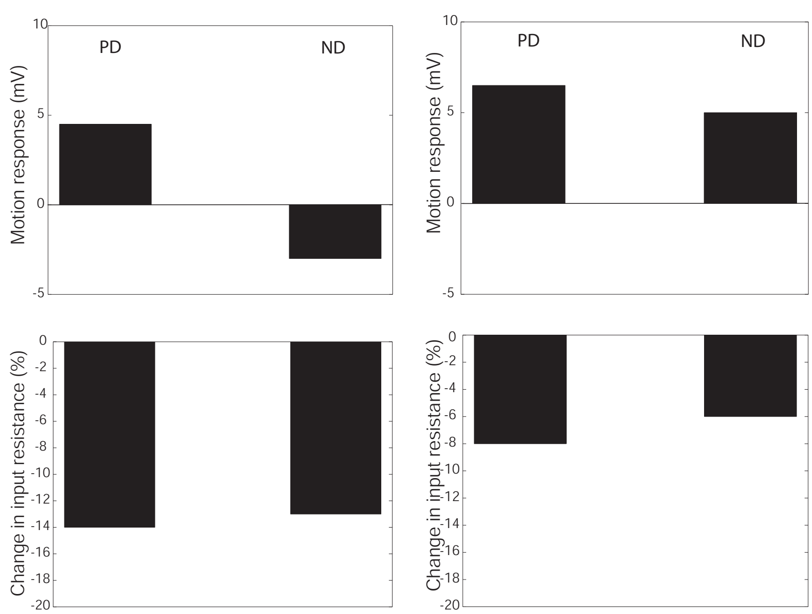
Figure 2: Experimental data from Single *et al.* The first row shows the mean response of LPTCs to moving visual stimuli. The second row shows the percentage change in LPTC input resistance. PD indicates motion in the preferred direction, ND in the null direction. (a) Data from LPTCs before application of PTX. (b) Data from LPTCs ten minutes after application of PTX.

Figure 3: Simulation results. The mean response of simulated LPTCs is shown to preferred (PD) and null (ND) direction stimuli. These results are qualitatively similar to the electrophysiological results of Single *et al.* (see Figure 2). (a) Control case: Inhibition to LPTCs is unblocked. (b) Inhibition to LPTCs is blocked.



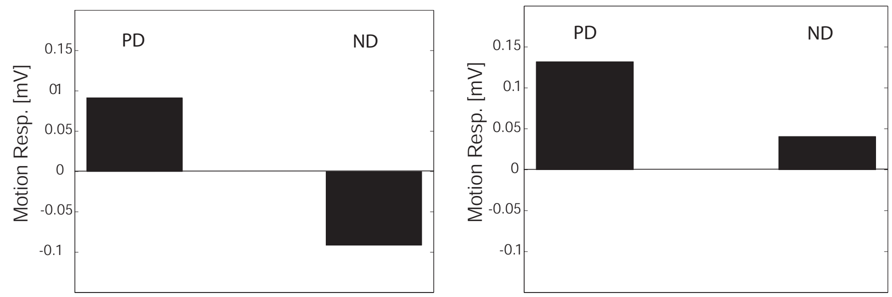
(a) (b)

Fig. 1.



(a) (b)

Fig. 2.



(a) (b)

Fig. 3.

Timothy Melano received his B.S. in Mechanical Engineering from the University of California at Berkeley in 2001. He is presently pursuing a Ph.D. in the Biomedical Engineering Program at the University of Arizona. His research interests are in Computational Neuroscience and Neuroprosthetics.

Charles M. Higgins received the Ph.D. in Electrical Engineering from the California Institute of Technology in 1993. He holds joint appointments in Electrical/Computer Engineering and Neurobiology at the University of Arizona, where his research focuses on investigation of neurobiological computational and control architectures.