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The neuronal basis of direction selectivity in lobula plate tangential cells

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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.
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Keywords: Diptera; Visual motion; Elementary motion detection; Tangential cells

1. Introduction

The Hassenstein–Reichardt (HR) correlation model, published in 1956 [4], 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 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 [5]. Although the HR model mathematically describes the responses of these so-called *tangential cells*, which are

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believed to integrate the outputs of many small-field elementary motion detectors (EMDs), it leaves the neural basis of this system open for investigation.

An important question that arises in motion detection is the method by which output from 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 puncturing the lobula plate [7]. Picrotoxin has been shown to block inhibitory input to LPTCs and is an antagonist to GABA receptors [1]. The investigators conducted electrophysiological experiments and computer modeling studies and the results of the two were compared.

In Single et al.'s electrophysiological experiments, the activity of several tangential cells was recorded before and 10 min after the application of PTX (see Fig. 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. Also, the change in cellular input resistance was greater in the preferred direction than in the null direction.

Directional selectivity can take two forms: weak and strong. Weakly directional EMDs exhibit an excitatory response to visual motion in the preferred direction and little response in the null, while strongly directional EMDs exhibit an excitatory response in the preferred direction and an inhibitory response in the null. In Single et al.'s computer modeling, weakly and strongly directional EMDs were tested. 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 1995, Douglass and Strausfeld recorded from T5 bushy T-cells, which are presynaptic to LPTCs, and described their activity as strongly directional [2]. This observation is in contradiction with one of the conclusions of Single et al. which states that strong direction selectivity first arises at the level of LPTCs and that the inputs to LPTCs are weakly directional. These recordings, along with other anatomical and electrophysiological evidence, led to the development of a neuronally based model of dipteran elementary motion detection (see Fig. 1a) [6]. In the present study, we simulated the possible effects of PTX on a tangential cell using the neuronally based EMD model in order to determine how the output of T5 cells are integrated at the level of LPTCs and propose interpretations that reconcile the above-mentioned studies. The present study also considers the possibility that, in the experiment by Single et al., PTX also affected inhibition in the lobula, since the dipteran lobula plate is very thin and T5 dendrites are located nearby in the outermost stratum of the lobula [3].

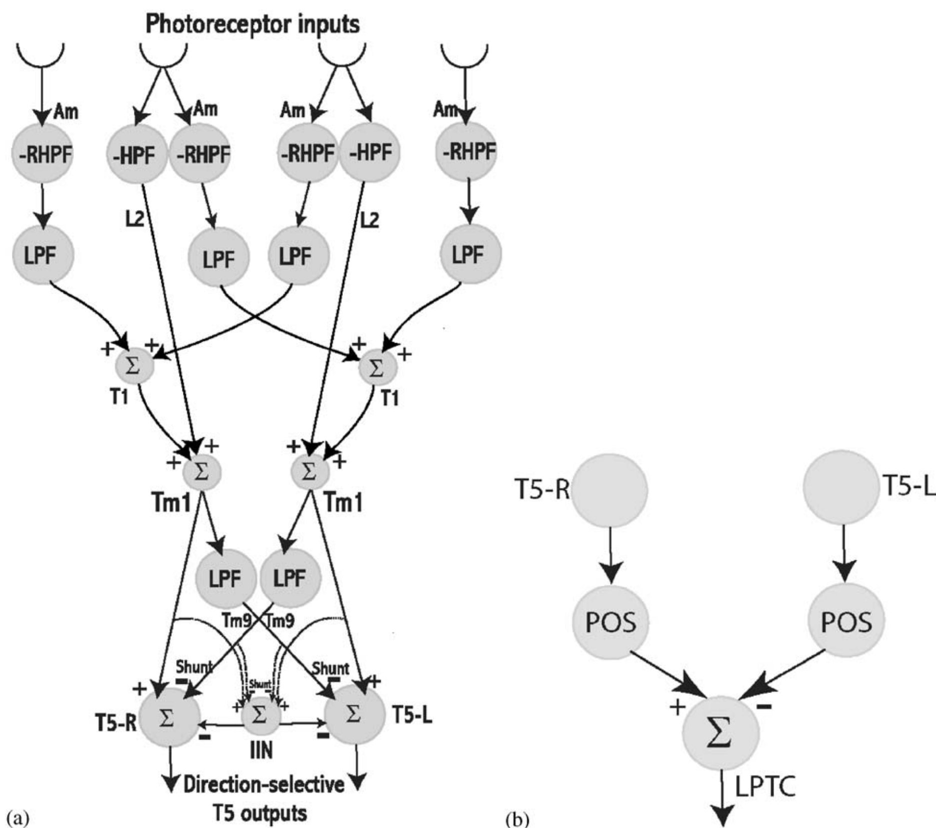

Fig. 1. (a) Neuronally based model of EMDs in dipteran insects. This model is composed of photoreceptors, amacrine (Am) cells, lamina monopolar (L2) cells, basket T-cells (T1), Tm1 and Tm9 transmedullary cells, an inhibitory interneuron (IIN), and T5 bushy T-cells (T5-L and T5-R). RHPF denotes a relaxed high-pass filter (allows a small component of a sustained output), HPF denotes a high-pass filter, LPF a low-pass filter, and Σ 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).

2. Methods

Simulations of the computational model were conducted using the *Matlab* package (The Mathworks, Natick, MA). The image input was 40×40 pixels and the visual system was composed of 20×20 photoreceptors with a similar number of optic cartridges. In our model, an optic cartridge consisted of a set of retinotopic cells from the photoreceptors through the T5 cells (see Fig. 1a). The filters used in modeling cellular activity were first order with time constants of 50 ms for the first high- 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

$$R_{\mathrm{LPTC}} = \sum_{All\ EMDs\ i} (pos(\mathrm{T5}_{L,i}) - f\, pos(\mathrm{T5}_{R,i})),$$

where $\mathrm{T5}_{L,i}$ and $\mathrm{T5}_{R,i}$ are the membrane potentials of the two T5 cells in optic cartridge i with the same preferred-null direction axis but with opposite preferred directions (see Fig. 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 [8]. Each optic cartridge contains four T5 cells at the level of LPTC dendrites. T5 cells have been shown to be strongly directional and to project only to the lobula plate [2]. Further, it has been proposed that there are two pairs of T5 cells with 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 are integrated into an LPTC to make maximum use of the redundancy inherent in this pair of opposing motion detectors. If so, one must be excitatory and the other effectively inhibitory 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 Fig. 2b). The only way to produce this effect in our model is if the synapses of T5 cells onto LPTCs are rectifying (see Fig. 1b). Anatomically, we suggest that one T5 neuron directly excites the LPTC, and the other inhibits the LPTC through the action of an inhibitory interneuron. Both synapses onto the LPTC are presumed to be rectifying chemical synapses.

In the study by Single et al., it is possible that the application of PTX-affected inhibitory synapses in the outer lobula as well as the nearby lobula plate. To investigate this possibility, we blocked Tm9 and IIN 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 equally to stimuli moving in any direction, which is inconsistent with the results of Single et al., and thus we conclude that only inhibition in the model at the level of the lobula plate could be affected by the simulated application of PTX.

Using the model shown in Fig. 1b, we simulated the integration of T5 cells into an LPTC. In Fig. 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 Fig. 2a. After inhibition in the model at the level of the lobula plate is removed, simulating the effect of PTX, the response shown in Fig. 3b is obtained, in qualitative agreement with the electrophysiological results.

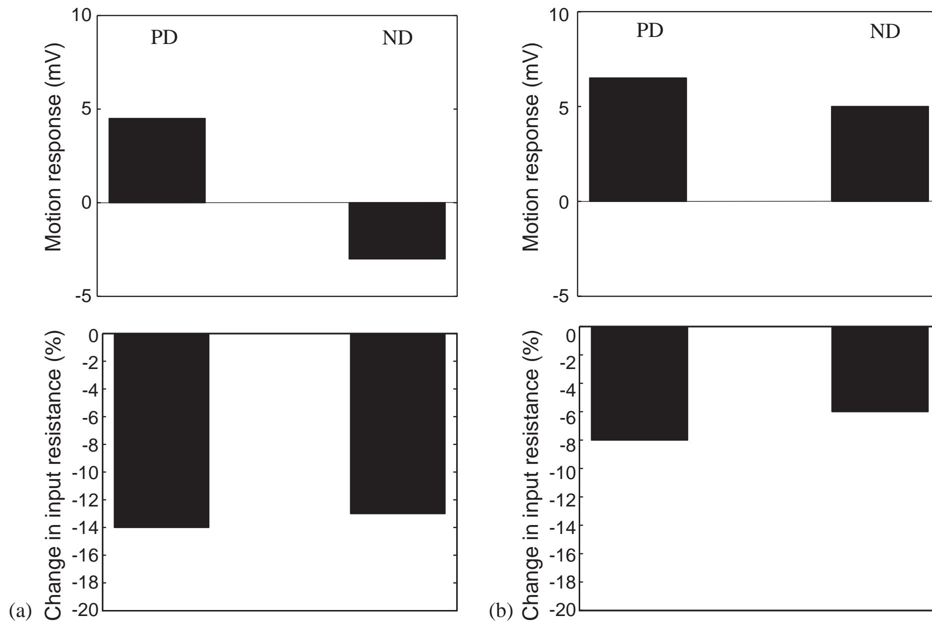

Fig. 2. Experimental data from Single et al. The first row shows the mean response of LPTCs to moving visual stimuli. The second row shows the percentage change in LPTC input resistance. PD indicates motion in the preferred direction, ND in the null direction. (a) Data from LPTCs before application of PTX. (b) Data from LPTCs 10 min after application of PTX.

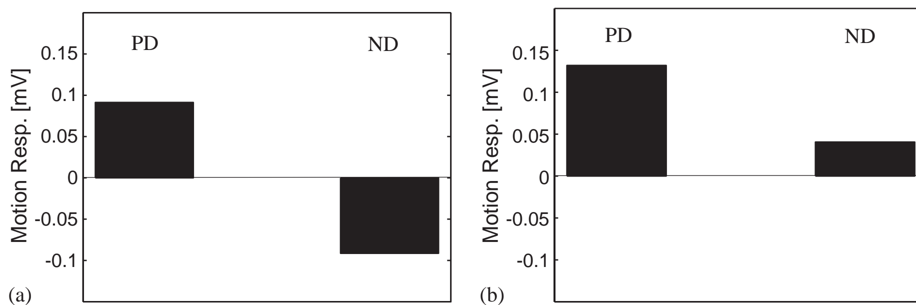

Fig. 3. Simulation results. The mean response of simulated LPTCs is shown to preferred (PD) and null (ND) direction stimuli. These results are qualitatively similar to the electrophysiological results of Single et al. (see Fig. 2). (a) Control case: inhibition to LPTCs is unblocked, (b) inhibition to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

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 excitatory input from T5

cells which respond 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 (Fig. 3a) was qualitatively similar to the electrophysiological data in the study by Single et al. (Fig. 2a). The cases where inhibition from T5 cells was blocked (Fig. 3b) provided results similar to the electrophysiological results of Single et al. after PTX was introduced into the lobula plate (Fig. 2b).

Electrophysiological data collected by Douglass and Strausfeld [2] 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 (Fig. 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.

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