Lateral Inhibition in the Compound Eye of the Fly, *Musca*

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Lateral Inhibition, Compound Eye, Fly

Lateral inhibition is a common phenomenon in the eyes of vertebrates as well as of invertebrates. Indications of lateral inhibitory interactions in dipteran eyes have been found by means of electrophysiological techniques [1, 2]. It was not possible, however, to correlate the sources of inhibitory activity to one of the known types of receptors nor to an exact orientation in the hexagonal array of ommatidia.

The compound eye of the fly, a "neural superposition eye" with unfused rhabdomeres, allows stimulation of single photoreceptors in the intact animal and measurement of optomotor turning reactions in open loop conditions. Stimulation f. i. of receptors 1 and 6 (inset Fig. 3) of one ommatidium with phase shifted periodic stimuli (frequency 2 Hz, phase angle 90°) leads to measurable turning reactions [3, 4]: These two receptors apparently are the inputs of a movement detector.

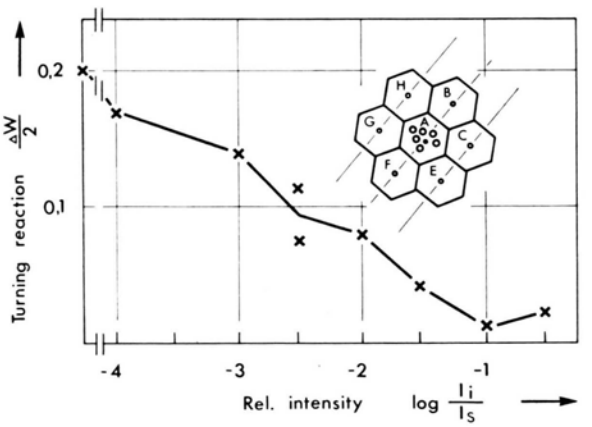

Fig. 1. Turning reactions in units of $\Delta W/2$ as a function of the intensity I_i at the rhabdomer 7 in ommatidium called C (see inset), relative to the intensity I_s at the receptors 1+6 in ommatidium called A (movement detector type β). The reactions have been measured by means of a y-maze globe [13]. $\Delta W/2$ is defined as one half of the difference between the turning tendencies induced by movement stimuli to the right and to the left. Linearly polarized light, $\varphi = 0$ (see Fig. 3). Inset: arrangement of ommatidia in the investigated upper front region of the eye.

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These reactions can be reduced by illumination of rhabdomeres No. 7 and 8 in other ommatidia. Ommatidia called A to H have been tested (inset Fig. 1). Two different types of interacting connections are realized: Receptor 7 and/or 8 of either the ommatidia B, E and G or those of ommatidia C, F and H inhibit the turning reaction produced by stimulation of receptors 1 + 6 in ommatidium A (Fig. 2), the corresponding movement detectors are

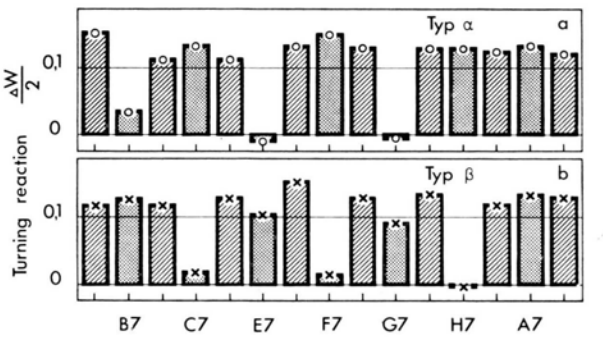

Fig. 2. Turning reactions induced by stimulation of receptors 1+6 in ommatidium called A (inset Fig. 1). Either no rhabdomer No. 7 (control, hatched, no symbol at the abscissa) or one rhabdomer No. 7 in the ommatidia indicated at the abscissa were stimulated (stippled). I_i/I_s was 0.03. Data of a movement-detector type α and β are shown respectively.

called type α or type β respectively. If the receptors 1 + 6 in ommatidium A are the input to a movement detector of type α, all receptors 1 + 6 in the ommatidia along the ommatidial row $-F-A-B-$ are also input to movement detectors of type α. Receptors 1 + 6 of the ommatidial rows $-G-H-$ and $-E-C-$ however are then input to movement detectors of type β: The movement detectors α and β are in the upper front region of the eye arranged in alternating rows as indicated by the lines in the inset Fig. 1. Stimulation of the rhabdomeres 7 and 8 with linearly polarized light and varying E-vector orientation (Fig. 3) shows that inhibition of the two types of movement detectors apparently is correlated to the two types of receptors, 7 and 8, since those have their microvillar- and probably their analyzer-orientations for linearly polarized light perpendicular to each other [5]. It can not yet be decided however, if only one of the two receptors, 7 or 8, is responsible for the inhibitory effect on each of the two detector types respectively, or if signals

combined in different ways from both receptors are the source of inhibition. It is remarkable that the maximal change in the inhibitory effect corresponds to an equivalent change in intensity by a factor of approximately 1:100 which is rather high (compare Figs 1 and 3).

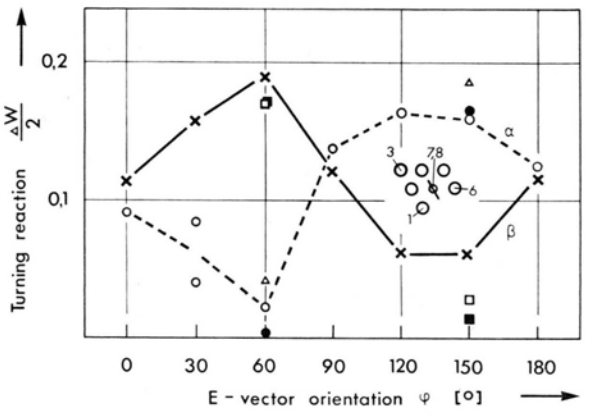

Fig. 3. Turning reactions induced by stimulation of receptors 1+6 in an ommatidium called A. At the same time inhibitory rhabdomeres No. 7 and 8 in neighbouring ommatidia were illuminated with constant, linearly polarized light of intensity I_i. The strength of the inhibition depends upon the E-vector orientation φ ($\varphi=0$: Vertical; $\varphi>0$: Counterclockwise rotation, see inset). Movement detector type α: Rhabdomeres No. 7 and 8 have been stimulated in ommatidium B (○), E (●) and G (△). Movement detector type β: Rhabdomeres No. 7 and 8 have been stimulated in ommatidium C (×), F (□) and H (■). $I_i/I_s=0.003$. The line through rhabdomer No. 7 (inset) indicates approximately the microvillar orientation of receptor No. 7.

The demonstrated inhibition acts onto one of the signal channels *before* the interaction, necessary for movement detection, takes place. This can be shown by experiments in which the input of the movement detector, following the receptors 1+6 in one ommatidium, is not activated directly by stimulation of the receptors 1+6 with periodic, phase shifted, stimuli. Instead, only one of these receptors is stimulated with a periodic stimulus ("ac-light"), the other with a constant light intensity ("dc-light"). This stimulation does not lead to a response. If now a rhabdomere of an inhibiting receptor 7 and/or 8 is stimulated with ac-light with a phase angle of 90° relative to either an ac-stimulated receptor 1 or 6 then no response or a *negative* response occurs. The conclusion is: The negative response shows that the dc-stimulated channel was *inhibited* before interaction for movement detection takes place. The response becomes negative because the phase angle is shifted by 180° due to inhibition. If no response occurs the ac-stimulated channel was the inhibited one. The appearance of a negative response shows furthermore that the inhibition is of the classical subtractive type, and not a multiplicative one which reduces gain in one of the inhibited channels. The negative response indicates also that this inhibitory interaction is a rapid process.

If the previously dc-illuminated receptor is maintained in the dark, no reaction at all can be elicited. This excludes the possibility that the inhibitory channel together with the ac-stimulated receptor 1 or 6 are a *direct* input to a movement detector. The simplest interpretation is that inhibition can only occur if the inhibited channel is not at the dark (= resting) potential.

The inhibitions demonstrated are not only "lateral" ones. Some of the connections act onto the output channel of a receptor that is stimulated from the same area in the optical surround as the inhibiting one (*e.g.* receptors 7 and/or 8 in ommatidium B which inhibit the signals of receptor 1 in ommatidium A receive their information from the same area of the optical surround as receptor 1 in ommatidium A [6]).

The demonstrated inhibition of the receptors 7 and/or 8 onto the system of receptors 1 to 6 might not be active only in movement perception, since it acts on discrete channels *before* information on movement is extracted. It could also be a source for the changes in spectral sensitivity of the phototactic response at different adaptation levels as measured in *Drosophila* [7, 8]: At low intensities the spectral sensitivity of the receptors 1 to 6 is seen according to the concept that these receptors are the input to a system with high absolute sensitivity [9]. At higher light intensities the UV-peak dominates more and more relative to the peak in the visible light. This is to be expected if the receptors 7 and/or 8 have a low sensitivity in the UV [10, 11], and if their efficiency is subtracted from that of receptors 1 to 6.

A detailed functional interpretation of the demonstrated, rapidly acting, inhibitory interactions cannot be given until responses to stimulation of otherwise-arranged ommatidia and of still more ommatidia at the same time are investigated. – Since spectral, angular and polarization sensitivity of the receptor types 1 to 6 and 7/8 respectively are different (*cf.* 12), it is obvious that the inhibitory interactions, beyond their possible role in adaptation of the eye to different average light intensities, may contribute to colour vision, pattern discrimination and/or to vision of polarized light.

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