Photoresponses from Cells in the Fly's Eye which are not Visual Cells

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It is shown that light elicits distinct responses from cells in the fly's eye which are different from photoreceptors. These cells are designated as "slow cells" because of their sluggish response characteristics. Like the visual cells the slow cells depolarize upon light stimulation, but the time course of their responses is clearly different from that of the photoreceptors. Furthermore, the intensity necessary to evoke a given response amplitude is considerably higher in slow cells than in photoreceptors. Several lines of evidence indicate that the slow cell response is caused by light absorption through the visual pigment rhodopsin in the peripheral photoreceptors R1–6. The slow cells are electrically coupled among each other, as demonstrated by application of local light stimuli and injection of the fluorescent dye Lucifer Yellow. The identity of the slow cells and the mechanism of response generation are discussed.

Introduction

The complex eyes of flies are composed of numerous small functional units, the ommatidia. A single ommatidium consists of a cornea lens, a crystalline cone that is secreted by four crystalline cone cells (Semper cells), two primary pigments cells surrounding the distal part of the ommatidium, six peripheral (R1–6) and two central (R7/8) photoreceptor cells, and one tracheole adjacent to receptors R7/8. The whole ommatidium is enveloped by secondary pigment cells shared also by the neighbouring ommatidia [1–3]. Most of the components in an ommatidium have optical, mechanical and/or metabolic functions. The visual cells are the only ommatidial elements in which light stimulation has been found to elicit a physiologically significant electric response. The electric response of fly photoreceptors has been subject of numerous studies (see [4] for review).

In this article it is shown that light also elicits distinct electric responses in cells which are different from the visual cells. The identity of the cells and the mechanism of response generation are discussed.

Materials and Methods

The experiments were carried out on male white-eyed blowflies (*Calliphora erythrocephala* Meig., chalky mutant). Most experiments were made on animals which were reared on carotinoid-rich liver in order to maximize the rhodopsin content in the photoreceptors of the adult flies (R^+-flies; see [5, 6]). For comparison, some experiments were made on flies bred on heart meat which caused the photoreceptors to be rhodopsin-deficient (R^--flies).

The preparation of the flies and the experimental set-up have been described in detail [7]. In short, a fly was fixed in a holder and its head horizontally hemisected near the eye equator. The holder was fitted to a perfusion chamber in which the head was steadily superfused with a saline of the following composition: 130 mM NaCl, 0.1 mM $CaCl_2$, 10 mM Hepes (N-2-hydroxyethylpiperazine-N′-ethanesulfonic acid), pH 7.0. In this medium the light responses of the photoreceptors were virtually the same as those in the dark adapted intact eye [7].

Perfusion chamber and manipulator for the microelectrode were mounted on the object table of a microscope by means of which the electrode was positioned in the preparation. Stimulation of the preparation was performed *via* the microscope objective. The orientation of the preparation was such that the ommatidia were illuminated perpendicular to their long axes, which guaranteed homogeneous light absorption over the entire lenght of the ommatidia.

The preparation of the fly was performed under white light and the positioning of the electrode under red light (RG 570; Schott, Mainz). The white light led to a relative rhodopsin content in the photoequilibrium between rhodopsin and metarhodopsin of 0.78 as measured photometrically. The photoequilibrium was further shifted towards the rhodopsin side by the red light so that most of the visual

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pigment was in the rhodopsin state at the beginning of the experiment.

Two light sources were used for stimulation: a 2 ms xenon photoflash and a high pressure xenon arc lamp. The light of the xenon flash was either filtered through a pair of cut-off filters (GG 400 and KG 1; Schott, Mainz) to exclude UV- and IR-radiation (white flash) or through a 504 nm interference filter (green flash). The wavelength of the green flash was near to the isosbestic wavelength (506 nm) of fly's rhodopsin/metarhodopsin system [8]. The light of the arc lamp was always filtered through 4 KG 1 filters and a water bath to eliminate IR-radiation. When spectral sensitivity was recorded the light was filtered through interference filters whose maximum transmission ranged between 375 and 604 nm (16 different wavelengths). The light intensities at the 16 wavelengths were adjusted by neutral density filters to obtain equal quantum fluxes. Quantum flux was measured in the position of the preparation by means of a calibrated thermopile (model M 2, Dexter Research Center Inc.). In chromatic adaptation experiments the light of the arc lamp was filtered through a BG 12 or RG 570 filter (Schott, Mainz) to obtain blue or red light of high intensity. Using these filters the relative rhodopsin content in the photoreceptors could be varied between 0.3 and about 1, as measured spectrophotometrically.

Local stimulation of the preparation was performed by projecting a small spot of blue light (BG 12) of about 20 μm diameter onto the preparation. The stimulus had a duration of 200 ms and was applied every 8 s. Between the stimuli the site of illumination was systematically changed by moving the preparation under microscopic control. This adjustment was carried out under dim dark-red light (RG 675), which is not absorbed by rhodopsin. The local stimulus did not significantly change the photoequilibrium between rhodopsin and metarhodopsin, since the responses of the impaled cells to repetitive stimulation at the same position were constant.

In staining experiments electrodes were filled with a 5% aqueous solution of Lucifer Yellow (Li salt; Sigma). Blue light (BG 12) was used for excitation of the Lucifer fluorescence; the emitted fluorescent light was filtered through a 540 nm cut-off filter.

Results

The electric responses to light described in this article were derived from the zone of the fly's eye in which photoreceptors and dioptric apparatus contact each other. When the tip of the electrode was pushed into this zone loci of pronounced negative potential were found, suggesting that the electrode was inside a cell. Upon light stimulation distinct changes in membrane potential were recorded. Since a definitive identification of these cells was not possible, the cells were preliminarily designated as "slow cells" due to their sluggish response characteristics.

Resting potential and response characteristics

The resting potential of the slow cells varied between −50 and −70 mV. The average value was -57.3 ± 7.1 mV ($\pm$S.D., $n = 17$ cells). Upon flash stimulation the slow cells transiently depolarized (Fig. 1a). Depolarization started after a distinct latency period of about 10 ms that was almost independent of stimulus intensity (Fig. 1b). At low intensity (log $I = -3.30$) the peak of the response was reached about 50 ms after the flash. Time-to-peak increased with stimulus intensity and at the highest intensity (log $I = 0$) it ranged between 200 and 400 ms. The decay of the responses was much slower than the rise and lasted more than 10 s at high intensities (Fig. 1a).

The time course of the responses derived from the slow cells clearly differed from that recorded in the photoreceptors (Fig. 1c, d). Furthermore, in contrast to the slow cells, latency and time-to-peak of the photoreceptor responses were shortened with increasing intensity. Thus, in photoreceptors latency was 10 ms at log $I = -3.30$ and 2 ms at log $I = 0$; the corresponding values for the peak time were 30 ms or, respectively, 10 ms. Moreover, the resting potential of the photoreceptor cells was about 10 mV smaller than that of the slow cells. The average value obtained from 62 photoreceptors was -47.5 ± 3.1 mV.

The differences between the response characteristics of slow cells and photoreceptors became even more evident when long lasting light stimuli were used (Fig. 2a, b). In slow cells the response maximum was reached 1 to 2 s after stimulus onset and within less than 30 ms in photoreceptors. Also, in photoreceptors illumination led to a considerable increase in membrane noise, whereas membrane noise appeared unchanged in slow cells. After cessation of the stimulus the slow cells repolarized almost exponentially to the resting potential, whereby the time course of the repolarization process was hardly de-

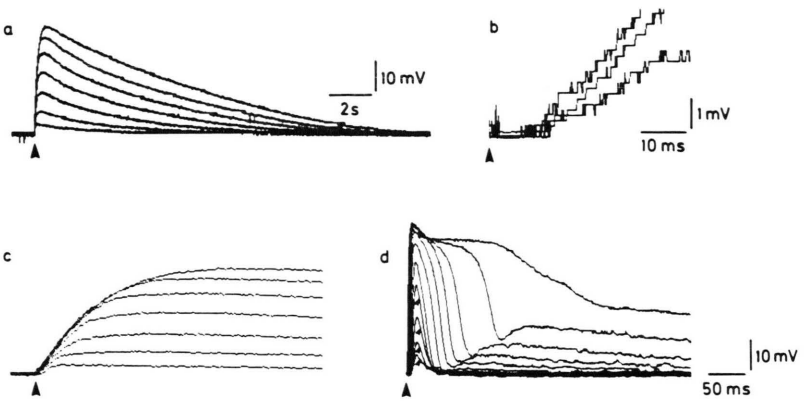

Fig. 1. Superimposed responses of slow cells (**a**, **b**, **c**) and a photoreceptor cell (**d**) to white xenon flashes of increasing intensity. Arrowheads mark the moment at which the stimulus was given. Stimulus interval was 1 min. Slow cell responses in **a** and **c** are the same, responses in **b** were derived from a different cell.
a. Complete slow cell responses to flashes of relative intensities log $I = 0$, -0.55, -1.10 ... -3.30.
b. Initial phases of slow cell responses (log $I = 0$, -1.10, -2.20) at magnified scaling.
c. Slow cell responses, and (**d**) responses of a photoreceptor cell presented in the same scaling, for comparison. Upper 7 responses in (**d**) were evoked by stimuli of the same intensity as used in (**c**) and (**a**). Stimulus intensities in (**d**) corresponding to the 4 lower traces (marked by small arrowheads) were log $I = -3.85$, -4.40, -4.95 and -5.50.

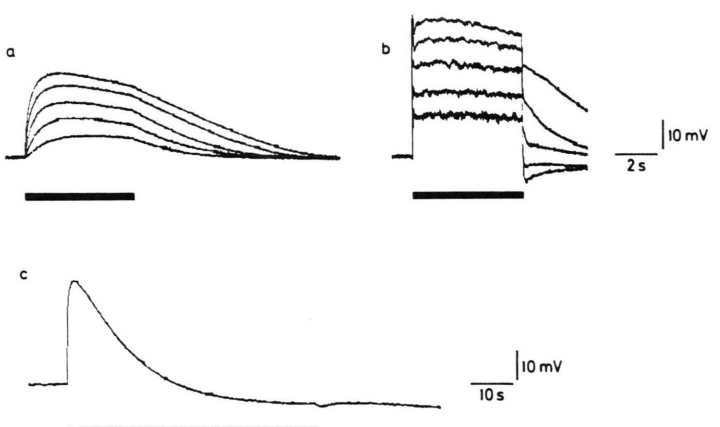

Fig. 2. Responses of slow cells (**a**, **c**) and a photoreceptor cell (**b**) to long lasting light stimuli (marked by bars beneath the traces).
a. Superimposed responses of a slow cell to 5 s stimuli of monochromatic light (513 nm). Relative intensities were log $I = 0$, -0.55, -1.10 ... -2.20. Stimulus interval was 1 min.
b. Superimposed responses of a photoreceptor cell to same stimuli as in **a**.
c. Response of a slow cell to a 60 s blue stimulus (BG 12). Note that during stimulation no steady state depolarization was established. Instead the cell repolarized even below the resting potential, and almost no change in membrane potential was observed upon cessation of the stimulus.

pendent on stimulus intensity (Fig. 2a). In contrast, the repolarization of photoreceptors strongly depended on stimulus intensity and proceeded in a more complex way (Fig. 2b).

The traces in Fig. 2 a show that at higher intensities the slow cells reached no steady state of depolarization during the 5 s stimulus. When the duration of the stimulus was increased the cells completely repolarized, and even hyperpolarized, and at the end of the stimulus only a slight fluctuation in membrane potential was detectable (Fig. 2c). The restoration of the response took several minutes to complete (see Fig. 5). In contrast, photoreceptors maintain a steady depolarization during long lasting stimulation and always respond to the cessation of the stimulus with a sudden increase in membrane potential.

The intensities of light flashes necessary to evoke responses of a given amplitude were much higher in slow cells than in photoreceptors (see Fig. 1). The relation between response amplitude (A) and stimulus intensity (I; A-log I function) is presented in Fig. 3 for both slow cells and photoreceptors. The figure shows that in slow cells the intensity necessary to evoke a criterion response of 10 mV was almost three orders of magnitude larger than in the photoreceptors: In rhodopsin-rich (R^+-) flies the intensity had to be increased by a factor 680 and almost the same value (770) was found in rhodopsin-poor (R^--) flies.

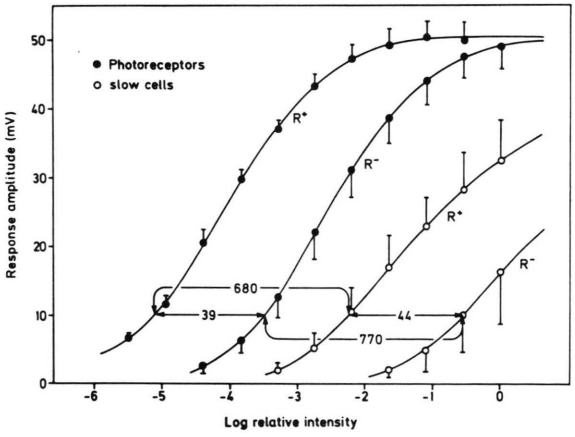

Fig. 3. Relation of response amplitude (A) on stimulus intensity (I; A-log I function) in photoreceptors and slow cells. A-log I functions were recorded in rhodopsin-rich (R^+-) or in rhodopsin-deficient (R^--) flies. Data points are the mean of 3 to 6 experiments. Vertical bars give standard deviation of the mean. Note that in R^--flies A-log I functions of both photoreceptors and slow cells are shifted to higher intensities as compared to the corresponding functions in R^+-flies, and that in both cases the shift was almost the same. The numbers give the factors by which the intensities had to be changed in order to evoke a response amplitude of 10 mV.

It is emphasized that this relationship is valid only for short light stimuli. If stimulus durations of more than 1 s were used, the difference between the intensities necessary to evoke a criterion response was considerably smaller (see Fig. 2a, b). This indicates that the "summation time", *i.e.*, the time span within which an increase in stimulus duration leads to an increase in response amplitude, is distinctly longer in slow cells than in photoreceptors.

Significance of carotinoid content in the larval food

The sensitivity of blowfly photoreceptors is proportional to the amount of rhodopsin in the rhabdomeres, which has been found to depend strongly on the supply of carotinoides during the larval stage [5, 6, 9]. Consequently, in R^--flies the A-log I function of the photoreceptors is shifted to higher stimulus intensities as compared to that of R^+-flies [5]. The shift by a factor 39 seen in Fig. 3 corresponds to a decrease in sensitivity to $1/39 = 0.026$. A very similar shift in the A-log I functions, by a factor 44, corresponding to a decrease in sensitivity to 0.022, was found in slow cells of R^+- or R^--flies. The result shows that the carotinoid supply during the larval stage affects the sensitivity of the slow cells in the same way as in photoreceptors, which suggests that the response of the slow cells is evoked by light absorption through the visual pigment rhodopsin. This presumption was strongly supported by the spectral sensitivity of the slow cells as well as by chromatic adaptation experiments.

Spectral sensitivity

Fig. 4a shows the responses of a slow cell to 5 s light stimuli the wavelength of which was varied between 375 and 604 nm (action spectrum). The maximum response amplitudes were elicited by light stimuli with wavelengths slightly below 500 nm. This range closely corresponds to the absorption maximum of the visual pigment rhodopsin in the photoreceptors R1–6 (see [6, 8]). Moreover, the spectral sensitivity of the slow cells determined from action spectrum (Fig. 4a) and A-log I function (Fig. 4b)

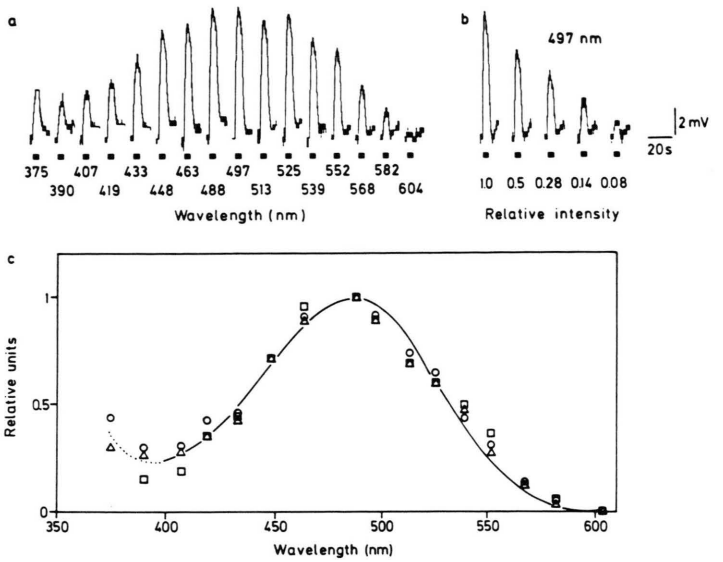

Fig. 4. Spectral sensitivity of slow cells.
a. Slow cell responses to 5 s stimuli of different wavelength as indicated. Stimulus interval was 1 min.
b. Responses to stimuli of relative intensities 1 to 0.08 at wavelength 497 nm. Stimulus interval as in **a**.
c. Relative spectral sensitivity of slow cells, as determined by relating the response amplitudes at the various wavelengths (**a**) to the *A*-log *I* function recorded at wavelength 497 nm (**b**). Data corrected with regard to intensity measured in the position of the preparation. Data collected from three experiments marked by different symbols. Circles represent data from the experiment shown in **a** and **b**. Solid line represents the absorption spectrum of rhodopsin normalized to maximum absorption at 490 nm.

closely matched the absorbance spectrum of the rhodopsin in the peripheral photoreceptors R1–6 (Fig. 4c).

Chromatic adaptation

The effect of chromatic adaptation on the response of a slow cell is presented in Fig. 5. The figure shows that after adaptation by blue light the response amplitudes were considerably smaller than after red adaptation. In the red adapted state the intensity of the test flash had to be reduced from 1 to about 0.28 in order to evoke the same response amplitude as after blue adaptation. This result indicates, that in the blue adapted state the sensitivity was more than

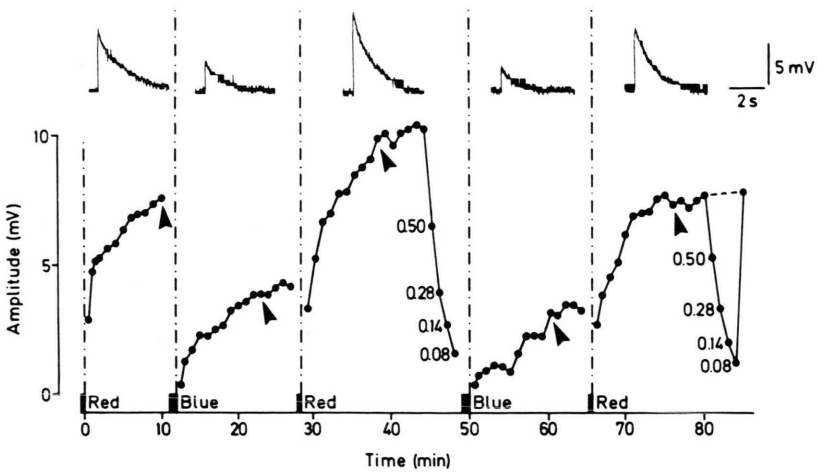

Fig. 5. Dependence of slow cell sensitivity on chromatic adaptation to blue or red light. Sensitivity was tested by means of green test flashes (504 nm) of moderate intensity given at different times after cessation of the adapting light. Following red adaptation the intensity of the test stimulus was reduced in steps from 1 to 0.08 as indicated (45–48 min and 81–84 min) in order to estimate the loss in sensitivity caused by blue adaptation. Traces show responses recorded 10 min after cessation of the adapting light (corresponding data points marked by arrowheads).

three times smaller than after red adaptation. This shift in sensitivity closely corresponds to the change in the photoequilibrium between rhodopsin and metarhodopsin that is induced by the red and blue adapting light [5, 6, 8]. The chromatic adaptation had no significant effect on the time course of the responses, as is evident from the traces shown at the top of Fig. 5.

Local illumination

Spectral sensitivity, the effect of chromatic adaptation, and the dependence of the sensitivity on the carotinoid supply indicate that the response of the slow cells is due to light absorption through the visual pigment rhodopsin. The question then arises whether the response is elicited by light absorption within the slow cells themselves or caused by some interaction with the photoreceptors. To answer this question the efficiency of local light stimuli at different positions within the preparation was determined for both cell types. As seen in Fig. 6a, when the position of the stimulus was shifted along the axis of the ommatidium in which the cell had been impaled, the efficiency profiles obtained for photoreceptors and slow cells were virtually the same. Both efficiency profiles rather well agreed with that expected for the photoreceptor cells calculated from the dimensions of the rhabdomere ([10], dotted area in Fig. 6a). That stimulus efficiency was not zero when the stimulus was outside the rhabdomere is most probably due to light scattering. The results indicate that the response of the slow cells is caused by light absorption in the photoreceptors.

Contrastingly, when the stimulus was shifted perpendicularly to the ommatidia, the profiles obtained for slow cells and photoreceptors clearly differed (Fig. 6b). In case of photoreceptors stimulus efficiency dropped considerably sharper with increasing distance from the impaled ommatidium as compared to slow cells. For example, 80 μm away from the impaled ommatidium stimulus efficiency in photoreceptors was reduced to 0.2 whereas in slow cells it dropped only to 0.8, and at a distance of 200 μm the efficiency in slow cells was more than 10 times higher than in photoreceptors (0.35 vs. 0.03). The results show that, in contrast to photoreceptors, light absorption in remote regions of the eye contributes to the response of the slow cells. This strongly suggests that the slow cells are electrically coupled among each other.

Dye injection

In order to identify the slow cells with one of the known cells types of the fly's eye, attempts were made to mark the cells by iontophoretic injection of

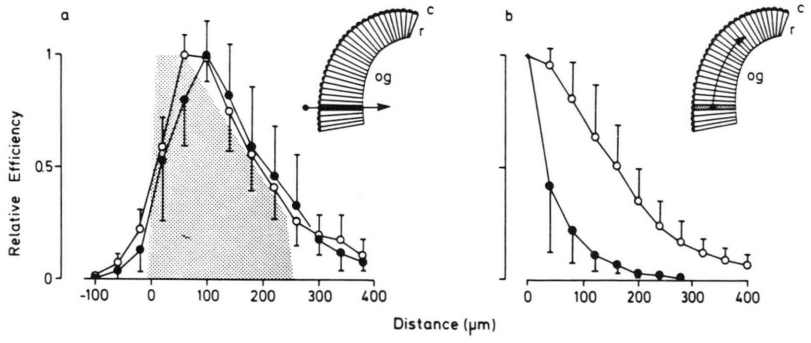

Fig. 6. Relative efficiency of a local light stimulus in slow cells (open circles) and photoreceptors (closed circles). Stimulus duration was 200 ms and the diameter of the illuminated area was about 20 μm. Stimulus efficiency was determined by relating the response amplitudes measured at the various positions to the A-log I function recorded at the position of maximum efficiency.

a. Stimulus shifted stepwise along the axis of the impaled ommatidium (axial efficiency profile). The zero point of the scale corresponds to the distal ends of the photoreceptors. Dotted area indicates the efficiency profile expected for the photoreceptor cells, according to the dimensions of the rhabdomere given by Boschek [10].

b. Stimulus shifted perpendicularly to the ommatidia, 150 to 200 μm proximally to the outer corneal surface (vertical efficiency profile).

Data points represent average of 5 or 6 experiments; vertical bars indicate standard deviation of the mean. The insets illustrate the movement of the stimulus in the preparation (c: cornea, r: retina, og: optic ganglia).

the fluorescent dye Lucifer Yellow [11]. In all 19 injection experiments the Lucifer staining was not restricted to a single ommatidium but spread laterally over several adjacent ommatidia (Fig. 7a, b). The fluorescence was most prominent in a narrow zone located proximally to the tips of the crystalline cones. From this zone fine fluorescent branches ran into the more proximal portions of the retina. Inspection of some fixed preparations at high magnification revealed that these branches extended to the basement membrane which separates the retina from the optic ganglia. Furthermore, the boundaries of the crystalline cones adjacent to the impaled cell were stained, particularly in the more distal regions (Fig. 7b). Neither the crystalline cones themselves nor the wedge shaped space between the cone tips appeared to be stained.

The staining of the slow cells was not stable, but faded rather quickly within less than 10 min after switching-off the injection current. The staining could be preserved by rapid fixation of the preparation. In contrast, photoreceptors retained the injected dye without fixation for at least 1 h. Furthermore, the staining of the receptor cells was always restricted to a single ommatidium (Fig. 7a, 11 experiments).

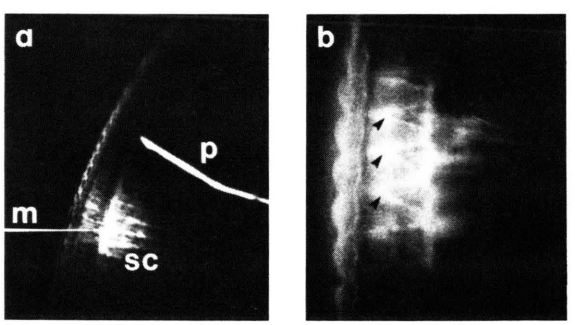

Fig. 7. Injection of Lucifer Yellow into slow cells and photoreceptors.
a. Fluorescence observed upon iontophoretic injection of Lucifer Yellow into a slow cell (sc) or photoreceptor (p) by means of the microelectrode (m). In case of the slow cell injection current was on while the photograph was taken. Injection into the photoreceptor was made about 15 min earlier. Magnification × 60.
b. Fluorescence in the region of the dioptric apparatus at increased magnification (× 160) after dye injection into a slow cell. Preparation immediately fixed in 4% formaldehyde after the injection. Note that the boundaries of the crystalline cones are distinctly stained, particularly in the more distal regions (arrowheads).

The rapid fading of the Lucifer staining in the slow cells could be interpreted to indicate that the observed staining pattern is at least partly due to dye migration within the extracellular space. This interpretation seems improbable, however, because dye injection into the extracellular space never led to a visible staining.

Discussion

This study demonstrates the existence of cells in the fly's eye which are different from the photoreceptors but respond to light with a distinct change in membrane potential. These cells have been designated as "slow cells" due to their sluggish response characteristics. As in photoreceptors a short light flash leads to a transient depolarization of the slow cells. The time course of the response, however, was clearly different from that of the photoreceptors (Fig. 1, 2), and the flash intensities necessary to evoke responses of an amplitude equal to that of the photoreceptors were almost three orders of magnitude larger (Fig. 3). Furthermore, in contrast to photoreceptors, long lasting illumination did not induce a steady state depolarization in slow cells: only a transient depolarization occurred at the beginning of the stimulus, but subsequently the cells repolarized and even hyperpolarized (Fig. 2c).

The response of the slow cells is caused by light absorption through the visual pigment rhodopsin, as shown by the result that the spectral sensitivity of the slow cells coincides with the absorption spectrum of the rhodopsin in the receptors R1–6 (Fig. 4). In addition, manipulations which have been shown to change the rhodopsin content in the photoreceptors and in consequence their sensitivity, led to equivalent changes in the sensitivity of the slow cells. One procedure to alter the rhodopsin content is to shift the photoequilibrium between rhodopsin and its photoproduct, metarhodopsin, by appropriate chromatic adaptation. Thus, red light shifts the equilibrium almost completely towards the rhodopsin side, whereas blue light reduces the rhodopsin fraction to 0.3 [6, 8, 12]. The reversible changes in the amplitude of the slow cell response observed after red/blue adaptation were in full accordance with the photochemistry of the visual pigment (Fig. 5). Another possibility to manipulate the content of visual pigment in the eye is to vary the amount of carotinoides in the larval food. The rhodopsin con-

tent of flies reared on carotinoid-rich liver (R^+-flies) has been shown to be more than ten times higher than that of flies reared on carotinoid-poor heart meat (R^--flies; [5, 6]), and 200-times higher than in flies bred on special deficient diets [9]. The results that the carotinoid supply during breeding affects the sensitivity of the slow cells in the same way as that of the photoreceptors (Fig. 3) further demonstrates that the slow cell response is caused by light absorption through rhodopsin.

The response of the slow cells most probably is due to light absorption in the photoreceptors, as shown by the result that the efficiency profile of a local light stimulus applied along the ommatidial axis is nearly the same in photoreceptors and slow cells (Fig. 6a). This result suggests that the slow cell response is due to the electric activity of the photoreceptors.

The slow cells are probably electrically coupled among each other, as demonstrated by the finding that stimulation of eye regions remote from the locus of impalement is considerably more efficient in slow cells than in photoreceptors (Fig. 6b). That a coupling among the slow cells exists is supported by the observation that dye injected into a slow cell spreads laterally to neighbouring ommatidia (Fig. 7).

Electric coupling and lateral dye spread both imply that the slow cells form a continuous tissue throughout the eye. This implication obviously excludes the possibility that the slow cells are identical with the Semper cells, since the Semper cells of neighbouring ommatidia are separated from each other by the primary and secondary pigment cells. In addition, the finding that the borders of the crystalline cones were distinctly stained up to the inner corneal surface (Fig. 7b) is not compatible with the position of the Semper cells at the ultimate tips of the crystalline cones [13, 14]. The staining pattern in the region of the crystalline cones is also incompatible with the arrangement of the secondary pigment cells, which mainly fill the space between the proximal parts of the crystalline cones [15]. However, just this space remained unstained upon dye injection (Fig. 7b). Insofar, the primary pigment cells appear to be the only candidates for being the slow cells, and indeed the staining pattern in the region of the crystalline cones is compatible with the fact that the primary pigment cells envelope the crystalline cones [1–3, 14]. Unfortunately, neither the staining in the proximal retinal layers, nor the lateral dye diffusion can unambiguously be attributed to the primary pigment cells, since information concerning the extension of the primary pigment cells to the more proximal parts of the retina is conflicting (compare [2] and [3]), and there is no evidence that the primary pigment cells of adjacent ommatidia contact each other. However, the available histological data do not disprove that the primary pigment cells are identical with the slow cells.

That pigment cells in insect eyes respond to light stimulation with a change in membrane potential has previously been demonstrated in honeybee drones [16]. The pigment ("glial") cells in the drone eye have been shown to be electrically coupled [17], and it is assumed that the response of the pigment cells is due to the light-induced release of potassium from the photoreceptors [16]. By the use of ionselective electrodes it has been shown that most of the potassium released from the photoreceptors during illumination is taken up by the pigment cells after which it is regained by the photoreceptors [16, 17].

A similar potassium "turnover" might also occur in flies. At least, the response of the slow cells to long lasting stimuli is consistent with such a process (Fig. 2c). An initial increase in the extracellular potassium concentration caused by the release of potassium from the photoreceptors (see [18]) could explain the depolarization of the slow cells after stimulus onset. The subsequent repolarization, and even hyperpolarization, may be due to the uptake of the potassium by the slow cells, which would result in a decrease in the extracellular, and in parallel to an increase in the intracellular potassium concentration.

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