Lateral Feedback from Monophasic Horizontal Cells to Cones in Carp Retina

I. Experiments

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ABSTRACT The spatial and color coding of the monophasic horizontal cells were studied in light- and dark-adapted retinae. Slit displacement experiments revealed differences in integration area for the different cone inputs of the monophasic horizontal cells. The integration area measured with a 670-nm stimulus was larger than that measured with a 570-nm stimulus. Experiments in which the diameter of the test spot was varied, however, revealed at high stimulus intensities a larger summation area for 520-nm stimuli than for 670-nm stimuli. The reverse was found for low stimulus intensities. To investigate whether these differences were due to interaction between the various cone inputs to the monophasic horizontal cell, adaptation experiments were performed. It was found that the various cone inputs were not independent. Finally, some mechanisms for the spatial and color coding will be discussed.

INTRODUCTION

Monophasic horizontal cells (MHCs) in carp retina receive predominantly hyperpolarizing input from the red-sensitive cone (R-cone) system (Norton et al., 1968; Spekreijse and Norton, 1970; Stell and Lightfoot, 1975; Stell et al., 1975). However, ~50% of the MHCs have additional input from the green-sensitive cone system (G-cone) (Yang et al., 1982; Tauchi et al., 1984; Yang et al., 1983; van Dijk, 1985). Those MHCs that receive R-cone and G-cone input are the subject of the present study. They are called nonunivariant MHCs (van Dijk, 1985). The effect of the interaction of the two cone inputs on the response properties and receptive field sizes of the MHC will be the central part of this paper.

The synaptic connections of horizontal cells (HCs) with the photoreceptors in fish have been described extensively (Scholes, 1975; Stell et al., 1975; Stell and Light-

foot, 1975). Stell proposed a feedback model to describe the responses of the various types of HCs in goldfish. This model consists of direct cone input to the HCs and a feedback from HCs to cones (Stell et al., 1975; Stell and Lightfoot, 1975). Since his model treats the various cone inputs as independent and since the model is not extended spatially, it cannot account for the differences in receptive field sizes of the different cone inputs to the nonunivariant MHCs reported by van Dijk (1985).

Receptive field properties of MHCs are influenced by numerous factors. For instance, horizontal cells have been shown to feed back on photoreceptors. This pathway underlies the surround of the photoreceptors in turtle (Baylor et al., 1971; Gerschenfeld and Piccolino, 1980; Lasansky, 1981), and presumably also of bipolar and ganglion cells (Baylor et al., 1971; Naka and Witkovsky, 1972; Kaneko, 1973; Burkhardt, 1974). In the turtle and salamander retinae, it is reported that HCs have a center/surround structure (Lamb, 1976; Lasansky, 1981; Piccolino et al., 1981; Itzhaki and Perlman, 1984). In the carp retina the various cone inputs feeding into the MHCs have different summation areas but do not show a true center/surround structure (van Dijk, 1985).

HCs of equal type are electrically coupled through gap-junctions (Kaneko, 1971). Because of this coupling, the receptive fields of HCs are larger than their dendritic fields (Kaneko, 1971). Modification of the gap-junctions induced by light adaptation has been reported (Wolburg and Kurz-Isler, 1985; Kurz-Isler and Wolburg, 1986). According to these studies, light adaptation makes the gap-junctions less dense and results in an increase of the gap-junction resistance. The resistance of the gap-junctions can be modulated by dopamine, cAMP, GABA, and light (van Buskirk and Dowling, 1981; Piccolino et al., 1982; Negishi et al., 1983; Teranishi et al., 1983; Mangel and Dowling, 1985; O'Connor et al., 1986). These modifications of gap-junction resistance are reported to be rather slow (0.5–2 h). The modulation of the gap-junctions has major implications for the size and the shape of the receptive fields of HCs (Teranishi et al., 1984).

Not only the gap-junction resistance but also the membrane resistance determines the coupling between the HCs. Synaptic input from the cones to the HCs decreases the membrane resistance (Trifonov, 1968; Werblin, 1975). As a result the coupling between the HCs will decrease. Moreover, a nonlinear I-V relation of the HC membrane resistance (Trifonov et al., 1974; Werblin, 1975; Byzov et al., 1977; Byzov and Trifonov, 1981; Tachibana, 1981) and voltage-dependent changes in coupling between HCs (Itzhaki and Perlman, 1987) have been reported. Due to such mechanisms, the coupling will be intensity dependent.

The aim of the present study is to describe the spatial response characteristics of the nonunivariant MHC as a function of stimulus wavelength, intensity, and of light and dark adaptation. To investigate the spatial and color coding of the MHCs, the receptive field size is measured in two different ways i.e., with spots of different sizes and with a slit positioned on various places in the receptive field. To investigate the interaction of the color processes feeding into the MHC, the response properties to flashes during chromatic backgrounds of various intensities and wavelengths were studied. The conclusions drawn from these experimental findings lead to a quantitative model for the MHC network, its cone inputs and feedback properties. This model is presented in the accompanying paper (Kamermans et al., 1989).

MATERIAL AND METHODS

Preparation

Eyes of grass carp (*Ctenopharyngodon idella*), weighing 700–1,200 g, were enucleated and the retinae were isolated under dim red light. Dark-adapted fish were not exposed to light for at least 1 h before the experiment and the light-adapted fish were exposed for at least 1 h before the experiment to 1,000 lx incandescent light. Morphological verification confirmed the state of adaptation (Zweypfenning et al., 1987). A total of 33 cells from 14 light-adapted and 36 cells from 22 dark-adapted carp were studied. The retina was placed with the receptor side up in a chamber that was continuously perfused with oxygenated (97.5% O_2 + 2.5% CO_2) Ringer's solution containing 102.0 mM NaCl, 2.0 mM KCl, 1.0 mM $MgCl_2$, 1.0 mM $CaCl_2$, 5.0 mM glucose, and 28 mM $NaHCO_3$. The temperature was kept at 17.5°C. The pH was kept at 7.8. Light stimuli were projected from below.

The Optical Stimulator

The experiments were performed on the setup described by van Dijk (van Dijk and Spekreijse, 1984; van Dijk, 1985). Briefly, the setup consists of two stimulus beams. The two beams, from a 450-W Xenon source, (Heinzinger, Rosenheim, FRG) were used to project spots, slits, and annuli of various sizes. In one stimulus channel the wavelength was controlled by a monochromator (Ebert, Waltman, USA), in the second stimulus channel it was controlled by interference filters (Ealing IRI filters, Watford, UK). The intensity in each of the channels was controlled by a pair of circular neutral density filters (Barr and Strout, CND_3, Glasgow, UK) ranging over 6 log units. Throughout the paper intensity values will be expressed in relative log units. Zero log intensity is equal to $9 \cdot 10^{16}$ quanta s^{-1} m^{-2}.

Recording

The microelectrodes were pulled on a Narashige puller (PA-81; Narashige Scientific Laboratory, Tokyo, Japan) and had a resistance of ~60 MΩ when filled with 3 M KCl and measured in Ringer's solution. The tip of the recording electrode was filled with a solution of 5% HRP (horseradish peroxidase type VI; Sigma Chemical Co., St. Louis, MO) in 500 mM KCl in a 0.05 M Tris/HCl buffer at pH 7.4. The stem of the electrode was filled with 500 mM KCl. The HRP electrodes had a resistance of 2 GΩ or more when measured in Ringer's solution. Intracellular responses were amplified using a World Precision Instruments, Inc. (WPI) S7000A with electrometer module S7071A (New Haven, CT). Data were recorded on magnetic tape (Ampex Corp., Redwood City, CA) and on chartpaper (Graphtec Linearcorder; Tokyo, Japan).

Classification

Cells were classified by standard criteria: spectral sensitivity and dependence on test spot diameter (Kaneko, 1970; Mitarai et al., 1974; Hashimoto et al., 1976; Kaneko and Stuart, 1980). If an MHC was found it was tested for G-cone input. For this test two spots of equal dimensions were superposed, one with a wavelength of 670 nm and the other with a wavelength of 520 nm. These spots were presented in alternation while the intensity of one of the spots was varied. The cell was classified as univariant if an intensity could be found for which the response could be nulled (Estévez and Spekreijse, 1982).

Stimuli

Data from three different sets of experiments will be reported.

Slit displacement experiments. In these experiments the receptive field profile of a MHC was plotted with a 250-μm wide and 3,300-μm long slit, flashed on for 0.5 s, and off for at

least 4.5 s without background illumination. The response amplitude was measured as a function of the position of the slit. The position was varied in steps of 800 μm perpendicular to the slit. Experiments were repeated at different stimulus intensities, in steps of 0.5 log units and for two wavelengths of 520 and 670 nm, respectively. In our setup these wavelengths activate the R-cone system approximately equally while the G-cone system is about 100 times more sensitive to the 520-nm stimulus.

Spot size experiments. In this set of experiments the receptive field profile of a MHC was measured by flashing spots of different diameter centered in the receptive field. As in the slit displacement experiments, the duty cycle was 0.5 s on and minimally 4.5 s off. The response amplitude was measured as a function of intensity, while the test spot diameter varied from 200 to 3,300 μm. Experiments were repeated for 520- and 670-nm test wavelengths. No background illumination was used.

Chromatic adaptation. In this set of experiments the influence of a steady chromatic background was recorded. Test flashes were on for 0.5 s and off for at least 1.5 s. Both test flashes and backgrounds covered the entire retina. The response amplitude was recorded as a function of background intensity, which was varied in 0.5-log unit steps. Responses obtained on 500-and 694-nm backgrounds were compared while the intensities of these backgrounds were matched for the R-cone system. Experiments were repeated for 520- and 670-nm test flashes at two different intensity levels.

RESULTS

Slit Displacement Experiments

Fig. 1 a shows the responses of a MHC in a light-adapted retina to 670-nm test flashes of the slit at different positions. Responses to slits at increasing distances from the center of the receptive field (left to right) and increasing intensity (top to bottom) are shown in Fig. 1 a for 670 nm, and in Fig. 1 b for 520 nm. The maximum response amplitude (at position 0 and intensity 0 log), was larger for 520 nm than for 670 nm. Comparison of the responses recorded at a displacement of 3,200 μm yields the opposite result; the responses to 670-nm flashes were larger than the responses to the 520-nm flashes. Note that there was no major change in the response waveform with position and intensity for either wavelength.

Fig. 2 shows the receptive field response amplitude profile for the two intensities and two test wavelengths depicted in Fig. 1. These curves are typical of all MHCs studied. Maximum hyperpolarization is taken as response amplitude. For the highest intensity, the receptive field profile measured with a 520-nm slit was steeper and the maximum response amplitude was larger than for the 670-nm slit. For the lowest intensity, however, the profiles were similar, while the responses to the 520-nm test slit were consistently smaller than or equal to those of the 670-nm test slit.

The difference of the receptive field profile for 670- and 520-nm test flashes cannot be due to stray light because such an effect would not depend on intensity.

The receptive field size became larger when the retina was dark adapted. At the highest intensity of the 520-nm stimuli, the diameter at half maximum was 1.1 ± 0.3 mm (n = 12) for light-adapted retinae, and 1.7 ± 0.5 mm (n = 18) for dark-adapted retinae.

Spot Size Experiments

Fig. 3 a shows the responses to 670-nm test flashes for a light-adapted MHC. Responses to flashes of spots with increasing spot diameter are shown from left to
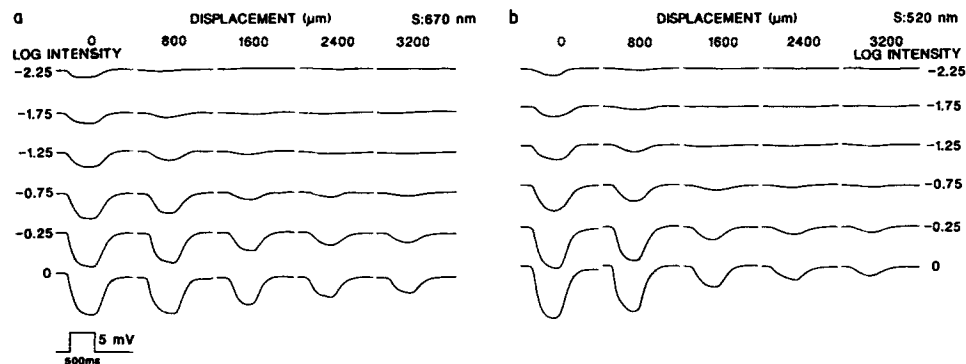
FIGURE 1. MHC responses to flashing slits of different wavelengths and positions within the receptive field. The dimensions of the slit are 250 μm wide and 3,000 μm long. Stimulus wavelength for *a* was 670 nm and for *b*, 520 nm.

right and to flashes with increasing intensity from top to bottom. The data are from the same cell as presented in Fig. 1. Fig. 3 *b* shows the responses for the 520-nm test flashes. Comparison of the responses to the largest spots shows that the peak amplitude was considerably higher for the 520-nm than for the 670-nm flash, and that the wave forms differ. A considerable repolarizing phase was present in the 520-nm responses but not in the response to 670-nm flashes. This repolarizing phase does not correlate with the response amplitude.

It is obvious that the width at half maximum amplitude of the response does not correlate with the response amplitude. For instance, the response to a stimulus of 670 nm, 0 log, and 3,300 μm in diameter has a maximum amplitude of 15.5 mV and a width of 585 ms, whereas a response to a stimulus of 520 nm, −0.25 log, and 1,830 μm in diameter has a maximum amplitude of 16 mV and a width of 510 ms.
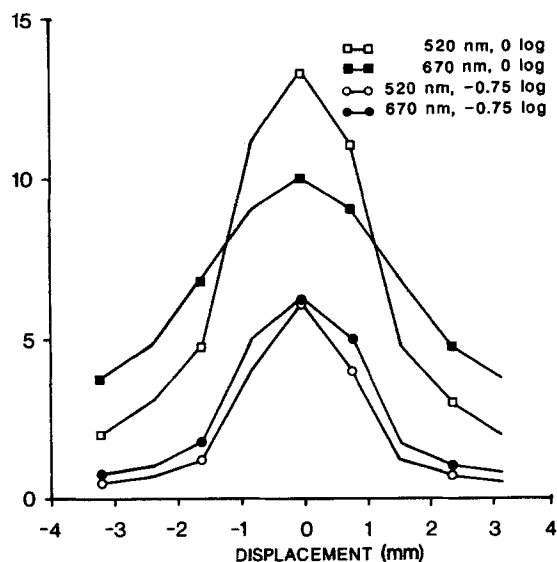

FIGURE 2. Receptive field response amplitude profiles for the experiment of Fig. 1. The squares indicate the receptive field response amplitude profile for a stimulus intensity of 0 log (filled symbols = 670 nm and open symbols = 520 nm). The circles indicate the receptive field response amplitude profiles for −0.75 log.
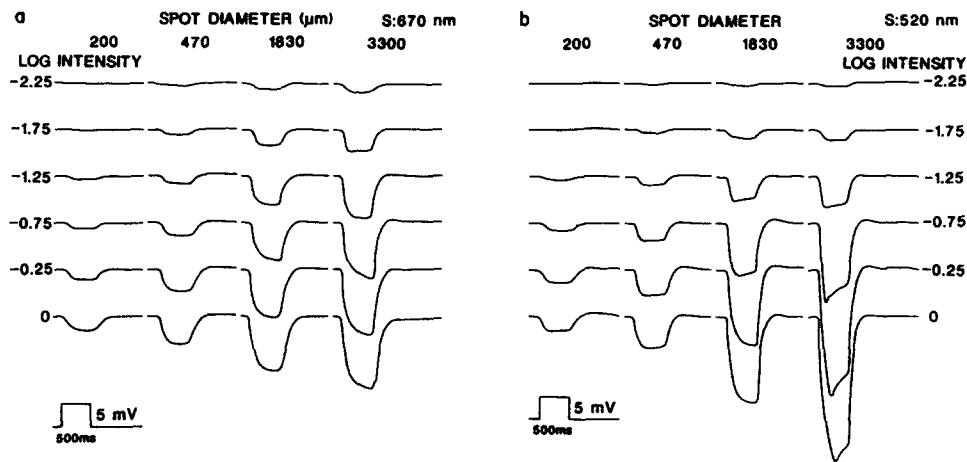
FIGURE 3.   MHC responses to test spots of different diameters and wavelengths. The stimulus intensity is varied vertically. Stimulus wavelengths for *a* were 670 nm and for *b*, 520 nm.

Fig. 4 shows the response amplitude vs. log intensity curves for the spotsize experiment to the two test wavelengths of 520 and 670 nm. As is evident from Fig. 3, the time to peak varied considerably throughout the set of responses. We determined the response amplitude 560 ms after stimulus onset, just before the response to light offset. The curves recorded with 670-nm flashes (open symbols) have been shifted along the log intensity axis such that they overlap. The curves recorded with 520-nm flashes (filled symbols) were shifted over the same distance as were the 670-nm spot data; for clarity they are plotted 1 log unit away from the 670-nm data. As seen, the curves for the 670-nm spots overlap, whereas the curves for the 520-nm spots become steeper with increasing spot diameter.

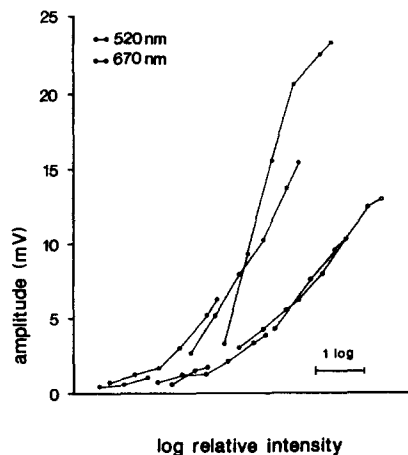No significant differences were found between cells in light- and dark-adapted retinae.

FIGURE 4.   Response amplitude vs. relative log intensity curves for four spot diameters are depicted: 200, 470, 1,830, and 3,300 µm, respectively, for spots of 670- and 520-nm wavelengths (filled symbols: 520 nm; open symbols: 670 nm). The 670-nm curves are shifted along the log intensity axis until they coincided. The 520-nm curves are shifted over the same distance as the 670-nm curves and for clarity over 1 log unit in addition.

Chromatic Adaptation

Fig. 5 shows the responses of a dark-adapted MHC to various combinations of stimulus and background wavelength as a function of background intensity. The data of Fig. 5 *a* were obtained with a stimulus intensity of −1 log, and those of Fig. 5 *b*, with a stimulus intensity of −0.25 log. In both figures, the first column shows the response to a 670-nm test spot for various intensities of a 500-nm background. The second column shows responses to a 670-nm test spot and 694-nm backgrounds, the last two columns show similar data but for a test spot of 520 nm. Only when both the stimulus and background were red (694 and 670 nm) the response waveform was invariant with the intensity (second row). In all other conditions the response waveform changed with the intensity of the background.

Fig. 6 depicts the amplitude of the flash responses of MHCs from dark-adapted preparations as a function of background intensity. The data points represent the
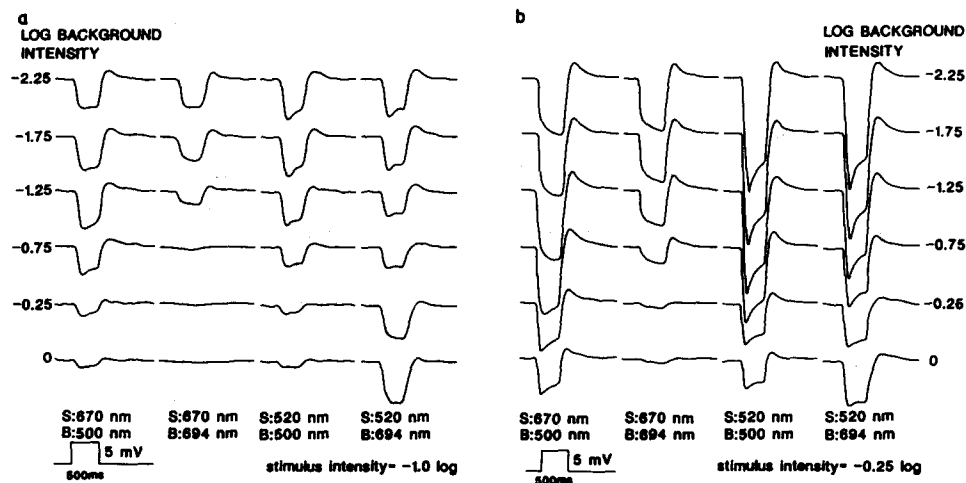

FIGURE 5.   MHC responses to full field test flashes on backgrounds of different wavelengths and intensities. Stimulus and background wavelengths and intensities are mentioned in the figure. (*S*, stimulus wavelengths; *B*, background wavelengths).

mean values and standard deviations for the number of cells indicated. Response amplitudes measured with a 694-nm background are depicted by a dashed line, and those obtained with a 500-nm background, by a solid line. The intensities of these backgrounds were matched for the R-cone mechanism. In the figure, the response amplitude is plotted at 560 ms after stimulus onset, which was just before the offset response. The results obtained with 670-nm test flashes are depicted in Fig. 6 *a* and the results obtained with 520-nm test flashes are shown in Fig. 6 *b*. All flashes had an intensity of −1 log.

As can be seen, all responses recorded with a 694-nm background and 670-nm test spots were smaller than the responses recorded with a 500-nm background of equal intensity. For low intensity 500-nm backgrounds and 670-nm test spots, the response amplitude increased with background intensity. At higher background
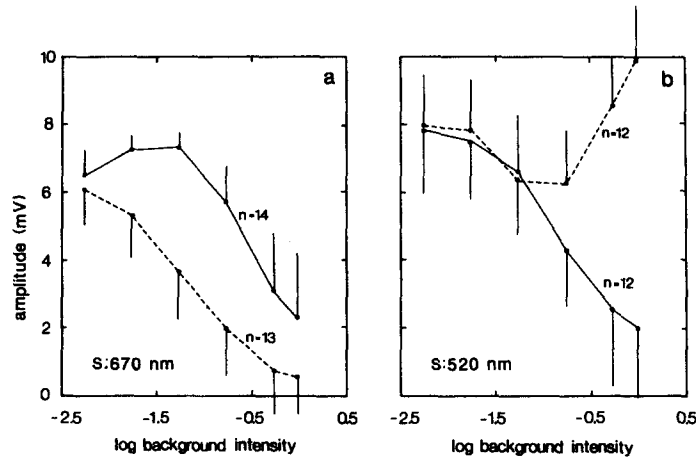
FIGURE 6. Mean response amplitudes are plotted against background intensity. The bars indicate standard deviations. The solid line represents data for a 500-nm background wavelength and the dashed line data for a 694-nm background wavelength. The stimulus intensity is −1 log. The response amplitude is measured at 560 ms after stimulus onset. n is the number of experiments.

intensities the response amplitude decreased for both background wavelengths as expected. When 520-nm test spots were used on a 694-nm background, a rather complex relation between amplitude, background, and stimulus intensity was found.

In Fig. 7 the change in the resting membrane potential induced by the background illumination is plotted as a function of background intensity. Note that at high intensities the slope for the 694-nm background curve (dashed line) was less steep than for the 500-nm background curve (solid line).
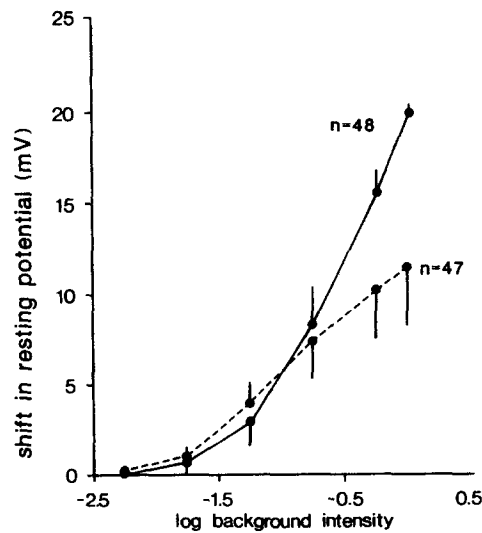

FIGURE 7. The shift in resting membrane potential induced by the background illumination as a function of the intensity of the background. Solid line, 500-nm background; dashed line, 694-nm background. The bars indicate the standard deviation. n is the number of experiments.

No significant differences were found when comparing light- and dark-adapted retinae.

DISCUSSION

Light Adaptation

Results for the slit displacement, the spot size, and the background experiments are not significantly different in light- and dark-adapted retinae. For the background experiments this is expected, because full field stimulation was used and, therefore, changes in the gap-junction resistance, induced by the light adaptation, will not have a major influence on the response amplitude. For the slit displacements and the spot size experiments, only quantitative differences in estimated receptive field sizes were expected. The slit displacement experiments revealed a smaller receptive field in light-adapted retinae compared with the dark-adapted retinae, which is in agreement with an increase of the gap-junction resistance during light adaptation. However, due to the variation in the receptive field size, these differences were not significant.

Receptive Field Properties

Comparison of the slit displacement experiment with the spot size experiment yields two different pictures of the receptive field organization. According to the slit displacement experiments, the receptive fields do not differ for low intensity 520- and 670-nm stimuli. This is in contrast to the spot size experiments in which for low intensities a smaller receptive field is found with a 520-nm stimulus than with a 670-nm stimulus. For high intensities, slit displacement experiments give a large receptive field for 670 nm and a smaller one for 520 nm, whereas spot size experiments yield the opposite. So, a stimulus-dependent spatial and color coding is present in the MHCs of carp. To understand how such a coding is accomplished it was necessary to investigate the interaction between the various cone inputs of the MHC.

Mutual Color Enhancement

The experiments with the adapting backgrounds show that the R- and the G-cone inputs are not independent. The 694-nm background can enhance the response to a 520-nm test flash and the 500-nm background can enhance the response to a 694-nm test flash, this is called "mutual color enhancement." This property of MHC responses has been reported by Byzov et al. (1977). Furthermore, a steady 520-nm background can hyperpolarize the MHC further than a steady 694-nm background (Fig. 7).

It is unlikely that a significant rod input was present because intracellular recording from rod-horizontal cells showed that the rods are saturated at the highest intensities used in our experiments. To exclude the possibility that there was a considerable blue-sensitive cone (B-cone) input, we also studied the influence of a 455-nm background on the response amplitude in the same way as already described for the 500- and the 694-nm backgrounds. If the response enhancement seen with the 500-nm background and 670-nm test flashes (Fig. 6) were to originate from B-cones, then this enhancement should have occurred at lower intensities of the 455-nm

background. Instead a shift to a higher intensity by ~1 log unit is found. This is in agreement with the difference in sensitivity for the R- and G-cone system at 455 nm. In conclusion, the R- and G-cones dominate the input to the MHCs strongly.

Models for the Color Coding of the MHC

It is unlikely that the difference in the receptive field diameters found for the R- and G-cone inputs originates through direct connections between R-and G-cones and the MHC. If this were the case, then two or more classes of MHCs with different spatial and spectral receptive field organizations would be expected. Such different classes, however, are not observed; all nonunivariant MHCs have similar response properties (Spekreijse and Norton, 1970; van Dijk, 1985). More importantly, the size of the receptive fields of MHCs is primarily determined by the strength of the coupling with other MHCs. This coupling strength is determined by the ratio of the membrane resistance to the extracellular space and the coupling resistance between the MHCs. Thus, we can conclude that the receptive field differences, as found in this study, originate in the horizontal cell layer itself, which leaves the following possible mechanisms:

(*a*) Both the axon terminals and the cell bodies form coupled networks. These networks have different space constants (Yagi, 1986; Yagi and Kaneko, 1987) and this accounts for receptive fields with different spatial properties for the G-cone and for the R-cone pathway as measured with the slit displacement experiments. However, the spot size experiments cannot be explained by such a model. Furthermore, the organization can be ruled out since the spectral properties of axon terminals and cell bodies in carp are highly similar (Kaneko, 1971).

(*b*) A wavelength- and intensity-dependent change of the gap-junction resistance between MHCs could explain the data of the slit displacement experiments. However, the response enhancement induced by the G-cone modulation of the gap-junction resistance would be most pronounced for small spots while little effect is expected for large spots or full field stimuli because the differences in membrane potential between neighboring MHCs will be very small and so will the current through the gap-junction resistance. The spot size experiments (Fig. 4) give the largest deviation between the responses to 520- and 670-nm test flashes when large spots are used. In the chromatic adaptation experiments only full field stimuli are used. Therefore, no effect of modulation of gap-junction resistance is to be expected. Thus, the response enhancement must have another origin, and direct changes in the gap-junction resistance cannot account for the data presented.

(*c*) A nonlinear nonsynaptic membrane resistance (Trifonov et al., 1974; Byzov et al., 1977; Byzov and Trifonov, 1981) would reduce the space constant with increasing response amplitude. The effect of a nonlinear nonsynaptic membrane resistance would be a scaling of the receptive field amplitude profile. The receptive field profile (Fig. 2) measured with a 520-nm stimulus and the profile measured with a 670-nm stimulus cross at high stimulus intensities. If the response enhancement is the effect of the nonlinear nonsynaptic membrane resistance, and thus depends on the membrane potential, no crossing of the curves can occur. Thus, nonlinear nonsynaptic membrane resistance cannot account for the differences in the size of the receptive field as found in this study. Furthermore, if the steepening of the ampli-

tude vs. log intensity curve is induced by a nonlinear nonsynaptic membrane resistance, then no effect from the spot size is expected on the response vs. intensity curves. Again, this does not agree with our data (Fig. 4).

(d) A feedback model, in which presynaptic feedback from MHCs to cones is pooled over a wide array of MHCs, can explain all the data we presented. A schematic view of the proposed model is shown in Fig. 8.

A quantitative description of the model is presented in the model paper (Kamermans et al., 1989); in short, the model consists of the following. Synaptic input to horizontal cells decreases the membrane resistance (Trifonov, 1968; Werblin, 1975). Following Byzov and co-workers, we assume that R- and G-cones modulate separate groups of ion channels in the MHC membrane, Rr and Rg (Trifonov et al., 1974; Byzov et al., 1977; Byzov and Trifonov, 1981). Such an organization is the origin of the mutual color enhancement. Note that the synaptic input is a modula-
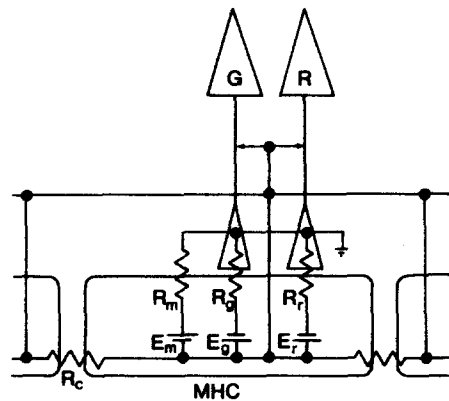

FIGURE 8. A schematic presentation of the proposed model. The following abbreviations have been used in this figure. *MHC*, horizontal cell; *R*, red-sensitive cone; *G*, green-sensitive cone; *Rr*, R-cone–modulated synaptic membrane resistance; *Rg*, G-cone–modulated synaptic membrane resistance; *Rm*, nonsynaptic membrane resistance; *Rc*, coupling resistance; *Em*, equilibrium potential of the ion channels of the nonsynaptic membrane resistance; *Er*, equilibrium potential of the ion channels of the R-cone–modulated synaptic membrane resistance; *Eg*, equilibrium potential of the ion channels of the G-cone–modulated synaptic membrane resistance.

tion of the synaptic membrane resistance and not, as assumed by Usui et al. (1983) and Yagi (1986), a current injection in the resistance network. This is an important difference because the coupling between the MHCs depends on the ratio of the total membrane resistance and the coupling resistance. Therefore, synaptic input changes the effective coupling between MHCs.

We also assume that there is presynaptic feedback from MHCs to all cones; that each cone receives this feedback signal from more than one MHC and that each MHC receives input from more than one cone. In other words, the feedback signal is the pooled signal from all the MHCs surrounding the cone. This assumption is based on the observation that the repolarizing phase of the MHC response, which we assume to originate in the feedback from the MHC to the cones, is more pronounced for large spots than for small spots and is independent of the response amplitude (Fig. 3).

In the model paper (Kamermans et al., 1989) we will show that the proposed model can explain the differences in receptive field amplitude profiles measured with slits of different wavelengths, intensities, and with spots of different sizes. Furthermore, the wavelength-, intensity-, and stimulus size–dependent changes of the dynamics of the MHC responses can be explained in the terms of the model.

In summary, a model of the MHC response should explain all of the following features. (a) Different sizes of the receptive fields measured with 520-nm stimuli compared with 670-nm stimuli. (b) An intensity-dependent shape of the receptive field amplitude profile. (c) A repolarizing phase of the MHC response which is independent of the MHC response amplitude and steady hyperpolarization (Fig. 3). (d) An overshoot of the MHC response that is independent of the MHC response amplitude and steady hyperpolarization (Fig. 5). (e) A width of the response at half maximum response amplitude which is independent of the MHC response amplitude (Fig. 3). (f) Mutual color enhancement.

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