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Models describing nonlinear interactions in graded neuron synapses

J.-F. Hu[1], **Y. Liu**[1], **P.-J. Liang**[2]

[1] Shanghai Institutes for Biological Sciences, Chinese Academy of Sciences, 320 Yue-Yang Road, Shanghai 200031, China
[2] Department of Biomedical Engineering, Shanghai Jiao Tong University, 1954 Hua-Shan Road, Shanghai 200030, China

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Abstract. An intracellular recording made from the retinal luminosity horizontal cell (LHC) demonstrated that repetitive red flashes enhanced the cell's responsiveness to red stimulus and depressed its responsiveness to green stimulus and that repetitive green flashes suppressed the cell's red response but produced little change in its green response. Based on the idea that the spectral plasticity of LHCs may reflect some synaptic efficacy changes between the LHC and various cones, a self-organizing system is proposed to investigate the possible manner of information processing and storage within the synapses. The results of model analysis suggest that the stimulus-pattern-related spectral plasticity is attributable to the excitatory interaction within the same kinds of synapses and the inhibitory interaction between different kinds of synapses. This system is able to encode and store the history of signal transmission in a graded and cumulative fashion.

1 Introduction

It has been widely accepted that activity-dependent modification in the strength of synaptic connection (synaptic weight) is fundamental to information processing and storage in the nervous system (for a review see Abraham and Bear 1996; Miller 1996). It has been suggested that a stimulus pattern generates an activation pattern across the neural structure, and both stimulus and activation patterns contribute to the changes in neuron response properties. Theoretical work via modeling approaches has been carried out to explain the dynamics of synaptic modifications observed from spiking neurons of various parts of the central nervous system (Kohonen 1982; Fukai 1995; Reynolds et al. 1999).

Correspondence to: Pei-Ji Liang
(e-mail: pjliang@sjtu.edu.cn,
Tel./Fax: (86) 2164070495)

In the carp retina, the luminosity-type cone-driven horizontal cell (LHC, a type of nonspiking neuron) receives inputs from both red-sensitive and green-sensitive cone photoreceptors, and the LHC's activity is dependent on the past and present activity status of the relevant neurons. Previous morphological work has revealed that the synapses between cones and LHCs were plastic and highly stimulus-pattern-related (Wagner and Djamgoz 1993; Haamedi and Djamgoz 1996). In addition, it has been frequently reported that the retinal horizontal cell's spectral sensitivity can be effectively regulated, with the modification effect closely related to the spectral and temporal characteristics of conditioning light (Yang et al. 1983; Kamermans et al. 1989; Djamgoz et al. 1996), which seems to suggest that the synaptic efficacy between LHCs and various kinds of cone cells is not fixed but rather status-dependent. This idea was recently confirmed by the observations made in our laboratory (Hu et al. 2000). It was found that repetitive red flashes enhanced the LHC's response to red light and depressed its response to green light; on the other hand, repetitive green flashes inhibited the cell's response to red stimulus, although it caused a trivial change in the cell's green response. It was thus assumed that the efficacy of some cone–LHC synapses could be enhanced, given a certain stimulation condition, while at the same time the efficacy of the remaining ones would be inhibited (Hu et al. 2000).

In the present study, theoretical models were investigated to explore the possible underlying mechanism of the observed spectral plasticity of the LHC. Using a simple group synapse model, we looked into the possible means of weight change in the relevant synapses. The results suggested that stimulus-pattern-related spectral plasticity is attributable to the excitatory interaction within each synapse group and the inhibitory interaction between the groups, which in turn contributes to the processing and storage of information within the synapses. This simple model was further extended into a two-dimensional structure, which allows for the inspection of the spatial interaction between the synapses during the stimulus-pattern-induced modification.

2 Construction of models

2.1 Group synapses model

A simple hypothetical model describing the use-dependent synaptic connections in the outer retina was developed based on the following factors and assumptions: (1) according to the spectral sensitivity curves of various cone photoreceptors of teleost fish retina (Palacios et al. 1998), the ratio of the inputs to red-cone and green-cone systems was assigned to be 0.9:0.1 for 703 nm light stimulus and 0.4:0.6 for 501 nm light stimulus; (2) cone-horizontal cell synapses were grouped into two sets, mediating red-cone and green-cone signals fed forward onto LHC (Kamermans et al. 1989); (3) the excitatory change of one group of synapses was accompanied by the inhibition of another (Miller 1996); (4) for simplicity, in this model the feedback signals from the LHC to the cones were ignored; (5) lateral coupling effect between the LHCs was minimized because the full field light stimulation was applied. The model construction can thus be equated as follows (Fig. 1a):

red-cone synapses:

$$\frac{dx_r(t)}{dt} = -\frac{1}{\tau_r}(x_r(t) - x_r(0)) + (a_r - x_r(t))E_r x_k I_k(t) - (x_r(t) - b_r)S_r x_{\bar{k}} I_{\bar{k}}(t) \quad (1a)$$

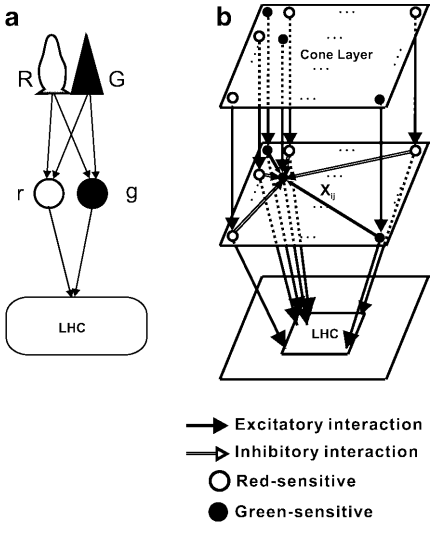


Fig. 1. Models for describing the synaptic connections between cones and LHCs. **a** Group synapse model. R and G, red and green cones, respectively; r and g, synapse connecting red and green cones to LHC, respectively. **b** Spatiotemporal model. *Open circles*: red-sensitive cones and relevant synapses. *Filled circles*: green-sensitive cones and synapses. *Solid lines*: excitatory interactions within same synapse population. *Open lines*: inhibitory interactions between different synapse populations

green-cone synapses:

$$\frac{dx_g(t)}{dt} = -\frac{1}{\tau_g}(x_g(t) - x_g(0)) + (a_g - x_g(t))E_g x_{\bar{k}} I_{\bar{k}}(t) - (x_g(t) - b_g)S_g x_k I_k(t) \quad (1b)$$

where $k, \bar{k} = r, g; x(t)$ is the strength of the relevant synapse at time t. Constants a and b denote, respectively, the maximum and minimum possible values of synaptic strength. Therefore, $(a - x(t))$ and $((x(t) - b)$ serve as nonlinear compression terms for, respectively, the excitatory and inhibitory interactions among the synapses. τ is the passive decay time constant of synapses. $I(t)$ is the input to synapse at time t, which is the intensity of light applied to relevant cone systems. The positive constants E and S are coefficients of, respectively, excitatory and suppressive interactions between the synapse populations (Ellias and Grossberg 1975).

The model thus has the following properties: (1) the synaptic strength has a static state value $x(0)$ when no stimulation is applied; if any synaptic-strength change is driven by a certain stimulus, the synaptic weight will return to its static value following a first-order dynamics with a time constant τ after the stimulation has ceased; (2) the synaptic excitation is weighted, as well as restricted, by $(a - x(t))$; (3) the synaptic inhibition is weighted and restricted by $(x(t) - b)$; (4) the output signal of a synapse population is determined by both the input signal $I(t)$ and the synaptic activity $x(t)$ following a sigmoid function:

$$f(u) = A\left(1 - e^{-u/\alpha}\right) \quad (2)$$

(5) the postsynaptic activity is, therefore, determined by a simple summation of the output of the two signal pathways (Kohonen 1982):

$$V(t) = \sum_k f(x_k(t)\, I_k(t)) \quad k = r, g \quad (3)$$

where $V(t)$ reflects the response amplitude of the postsynaptic LHC, with the synaptic weight $x(t)$ varying between 0 and 1. For simplicity, we set a with a maximum value of 1 and b with a minimum value of 0. All the parameters were optimized following the least-square-errors criterion, as listed in Table 1.

2.2 Spatiotemporal model

In the retina, each single LHC actually contacts a number of cone photoreceptors via densely populated synapses. Following the results of numerical analysis based on the simple group synapse model, the model structure was further extended into a spatiotemporal form with self-excitation and cross inhibition among the synapse population to describe the use-dependent synaptic connection in the outer retina. In this extended model, the synapses were arranged in a two-dimensional topological array ($N \times N$), with each synapse between

Table 1. Optimized parameter values for different models

Parameters	Group synapse model		Spatiotemporal model
	I	II	
E_r	0.114	0.127	0.020
E_g	0.314	0.091	0.030
S_r	0.171	0.073	0.015
S_g	0.314	0.109	0.010
a_r	1.0	1.0	0.9
a_g	1.0	1.0	0.9
b_r	0.0	0.0	0.0
b_g	0.0	0.0	0.0
τ_k	5.0 s	5.0 s	5.0 s
x_{k0}	0.5	0.5	0.5
N	/	/	10
σ_E	/	/	3.0
σ_s	/	/	5.0
β	/	/	0.05
σ_ε	/	/	1

the red cone and LHC being sandwiched by two synapses connecting the green cones and the LHC, in both abscissa and ordinate directions, and vice versa (Fig. 1b).

The corresponding equations are as follows:

for red cone–LHC synapses:

$$\begin{aligned}\frac{\mathrm{d}x_r(t,i,j)}{\mathrm{d}t} &= -\frac{1}{\tau_r}(x_r(t,i,j)-x^*_{r0})+\sum_{\substack{m\neq i\\ n\neq j}}(a_r-x_r(t,i,j))x_r(t,m,n)\\ &\quad\times E_r(i,j,m,n)I_r(t,m,n)-\sum_{\substack{m\neq i\\ n\neq j}}(x_r(t,i,j)-b_r)x_g(t,m,n)\\ &\quad\times S_g(i,j,m,n)I_g(t,m,n) \qquad (4a)\end{aligned}$$

for green cone–LHC synapses:

$$\begin{aligned}\frac{\mathrm{d}x_g(t,i,j)}{\mathrm{d}t} &= -\frac{1}{\tau_g}(x_g(t,i,j)-x^*_{g0})\\ &\quad+\sum_{\substack{m\neq i\\ n\neq j}}(a_g-x_g(t,i,j))x_g(t,m,n)\\ &\quad\times E_g(i,j,m,n)I_g(t,m,n)\\ &\quad-\sum_{\substack{m\neq i\\ n\neq j}}(x_g(t,i,j)-b_g)x_r(t,m,n)\\ &\quad\times S_r(i,j,m,n)I_r(t,m,n) \qquad (4b)\end{aligned}$$

where

$$x^*_{r0}=x_{r0}+\beta\varepsilon(t,i,j);\ x^*_{g0}=x_{g0}+\beta\varepsilon(t,i,j);$$
$$t=1,2,\ldots,40;\ i=1,2,\ldots,N;\ j=1,2,\ldots,N;$$
$$m=1,2,\ldots,N;\ n=1,2,\ldots,N;$$
$$i+j=\begin{cases}\text{even, for red synapses}\\ \text{odd, for green synapses}\end{cases}$$

In Eq. 4, $x_k(t,i,j)$ $(k=r,g)$ represents the strength of the synapse (i,j) at time t, with x_{k0} being the static value when no stimulation is applied. Constants a and b denote, respectively, the maximum and minimum possible values of the relevant synaptic weight. Therefore, the nonnegative terms $(a-x(t,i,j))$ and $((x(t,i,j)-b)$ form nonlinear compressions for both self-excitatory and cross-inhibitory processes among the synapse populations, respectively. τ represents the passive decay time constant of the synaptic strength. $I(t)$ is the intensity of light input to relevant signal pathway at time t. $\varepsilon(t,i,j)$ is a white noise process following the distribution:

$$\varepsilon\sim N\,(0,\sigma^2_\varepsilon) \qquad (5)$$

The nonnegative terms $E(i,j,m,n)$ and $S(i,j,m,n)$ are excitatory and inhibitory interaction functions within and between the synapse populations, respectively (Ellias and Grossberg 1975), with their spatial decay following the forms:

$$E(i,j,m,n)=E\times e^{-((i-m)^2+(j-n)^2)/\sigma^2_e} \qquad (6a)$$

$$S(i,j,m,n)=S\times e^{-((i-m)^2+(j-n)^2)/\sigma^2_s} \qquad (6b)$$

The postsynaptic activity, i.e., the response amplitude of LHC at time t, is therefore determined by a simple summation of the input stimulus $I(t)$ multiplied by the synaptic weight x (Kohonen 1982):

$$Y(t)=\sum_{i,j}f(x_k(t,i,j)I_k(t,i,j)) \qquad (7)$$

where the synaptic weight $x(t,i,j)$ varies between 0 and 1, with the initial value following a normal distribution:

$$x^*_{k0}\sim N(x_{k0},\beta^2) \qquad (8)$$

both spatially and temporally. All the parameters were determined via the least-square-errors algorithm, as listed in Table 1.

3 Results

3.1 Experimental data

In the carp retina, LHC receives signals from both red-cone and green-cone pathways. The signal-transmission efficacy between LHC and each cone system is plastic, which is dependent on both the history and activity of the signal pathways. Our previous data seemed to suggest that the strengthening of a certain signal pathway is accompanied by the weakening of the other (Hu et al. 2000). Such stimulus-related response enhancement and depression effects caused by repetitive monowavelength light flashes are plotted in Fig. 2, which gives a typical LHC's response to red and green test flashes before, during, and after a sequence of repetitive red or green light flashes measured intracellularly from the cell (solid lines in the left and right panels, respectively). It is shown that a repetitive red (703 nm) flash sequence (1 Hz, light-on duration 500 ms, 20 repeats) progressively enhanced the LHC's response to

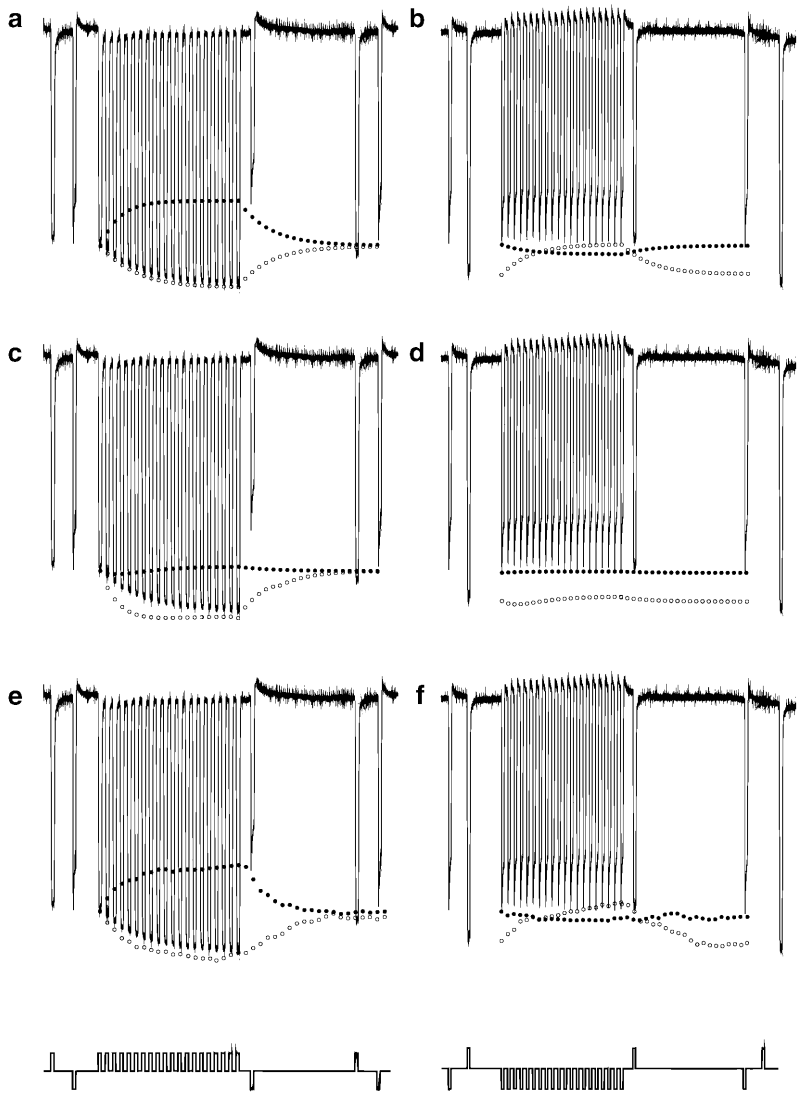


Fig. 2. Stimulus-pattern-related LHC spectral plasticity and model description. *Solid lines* in the *left* and *right panels* are the experimental observations for the response changes caused by repetitive red and green flash sequences, respectively. *Open* and *filled circles* are the model predictions for the cell's response to red and green test light during each condition. **a, b** group synapse model I; **c, d** group synapse model II; **e, f** spatiotemporal model. The *lower panels* give the light stimulus patterns applied

the red stimulus and depressed the cell's response to the green (501 nm) test flash. On the other hand, repetitive green flashes (1 Hz, light-on duration 500 ms, 20 repeats) remarkably depressed the LHC's red response, although it caused little change in the cell's green response. All the response amplitude change caused by repetitive monowavelength light flashes could be recovered within a short period (about 10 s) after the flickering light had ceased.

3.2 Model analysis

The group synapse model provides a number of combinations of the synaptic weight changes. However, according to the experimental recordings, only two possibilities are acceptable: (1) stimulating one signal pathway enhances its efficacy but inhibits the unstimulated pathway ($k = r, \overline{k} = g$ in Eq. 1) and (2) stimulating one signal pathway attenuates its connection efficacy but enhances the strength of the unstimulated one ($k = g, \overline{k} = r$ in Eq. 1). The model was fitted to the experimental data with the parameter values confined according to these two conditions. The results are plotted in Fig. 2. It is shown that when the model parameters were chosen to follow the assumption of self-enhancement/cross inhibition among the synapses, the model output was able to follow the experimental recordings that showed that during repetitive red flashes the cell's response to red light was gradually enhanced (Fig. 2a, open circles) while its response to green test light was correspondingly decreased; the recordings additionally showed that such changes could be recovered after the red flickering had ceased (Fig. 2a, filled circles). Similarly, the cell's behavior during green flickering light indicates that its response to green light was barely affected, while its response to red flash was remarkably depressed. The cell's response amplitude recovered to its initial values within a short period after the green flickering stimulation had ceased (Fig. 2b). The parameters were also optimized to the model using the self-attenuation/cross-enhancement assumption. With such constraints the model failed to describe the cell's response changes induced by the monowavelength repetitive flashes (Fig. 2c and d).

These results suggest that the stimulus-pattern-related spectral plasticity is attributed to the excitatory interaction within the same kinds of synapses and the inhibitory interaction between different kinds of synapses. The model structure was then extended into a spatiotemporal form to explore the changes of different synaptic weights, as described in the previous session, since each LHC actually contacts a number of cone photoreceptors via relevant synapse populations. The model output was compared to the experimental data. The cell's behavior was well described by the model as illustrated in Fig. 2e and f, which is similar to that given by Fig. 2a and b. However, with the presence of an intrinsic noise, the model output shows some fluctuation in response amplitude.

3.3 Synaptic weight changes

The changes in synaptic weight are crucial to the changes in the cell's light response as well as the information storage in the network. We thus examined the spatiotemporal changes of synaptic weights between LHCs and various cone systems during various stimulation conditions. The calculated values of synaptic weight ($x(t,i,j)$ in the model) are plotted in Fig. 3. Figure 3a illustrates the calculated synaptic weight during a 20-s static period when there was no intense stimulation to either red-cone or green-cone pathways. The synaptic weights were kept near their static values, with a random fluctuation due to the intrinsic noise $\varepsilon(t,i,j)$. Figure 3b gives the synaptic-weight changes during the 20-s repetitive red flashes and the after-flickering recovery predicted by the model. It is shown that the synaptic weights between red-cone and LHC were strengthened during the time interval, whereas the synapses between green-cone and LHC weakened, with the changes in the central area affected to a greater extent. According to Palacios et al. (1998), red stimulus predominantly activates red cones, whereas green stimulus activates not only green cones but also, to a certain extent, red cones. The cell's red response enhancement during repetitive red flashes is attributed to the strengthening of synapses between red cone and LHC. And the cell's green response depression is the result of a major weakening in green cone–LHC synapses vs. a minor strengthening in red cone–LHC synapses. It is likely that with the green flickering the weights between red cone and LHC were slightly decreased, while those connecting green cone and LHC were moderately strengthened. The decrease in red cone–LHC synaptic weight led to the inhibition of LHC's response to red test light, whereas the balance in green-cone signal increase vs. red-cone signal decrease resulted in a trivial change in the LHC's green response.

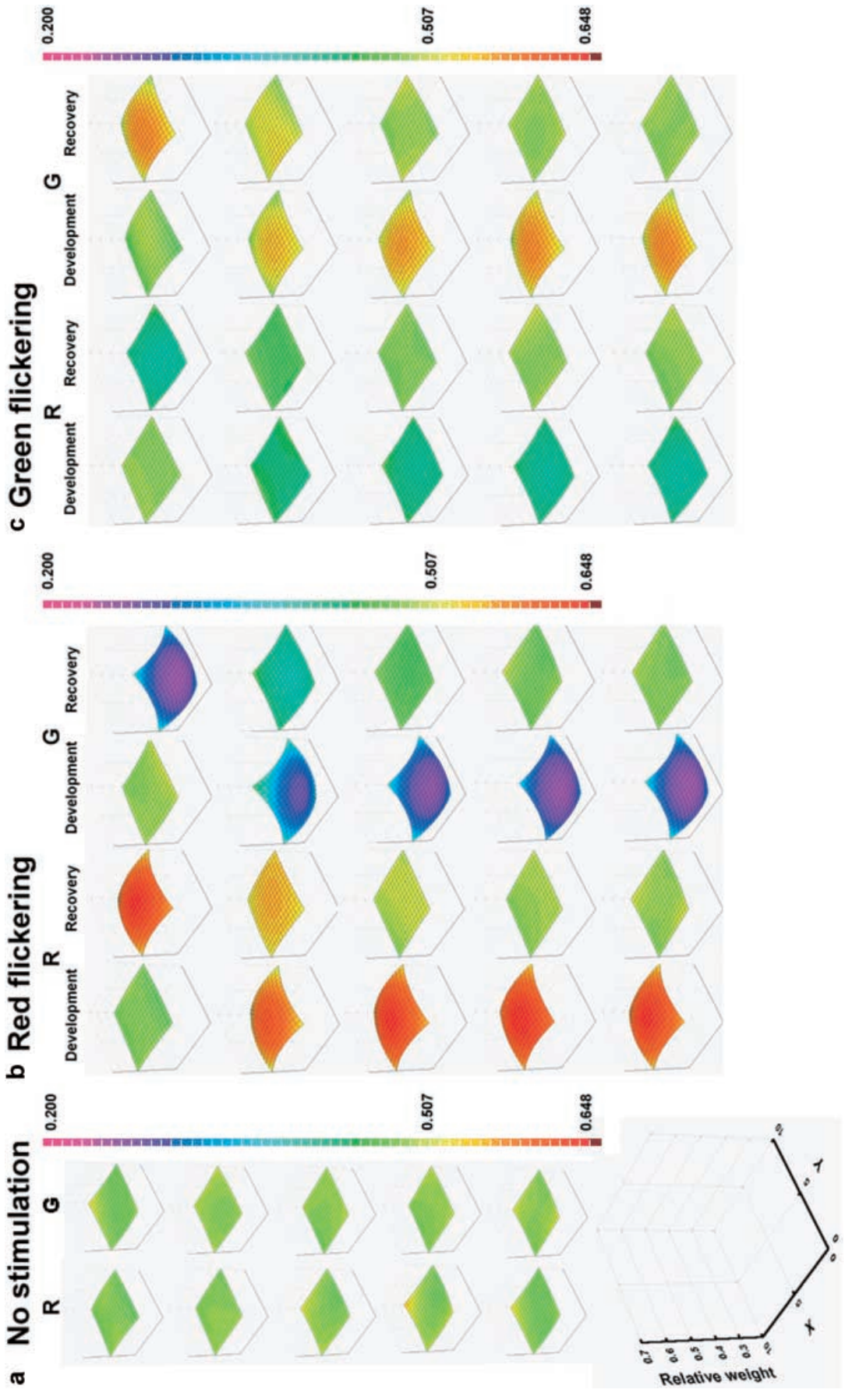
4 Discussion

The modification of synaptic efficacy, or its "self-organizing" property, has been widely studied over the decades. Theoretical work has provided a number of hypotheses. An early model proposed by von der Malsburg (1973) assumed that synapses could be modified following the Hebbian rule and that these processes were competitive so that some synapses were strengthened at the expense of the others. In such a model, the summed value of synaptic strength related to each postsynaptic neuron was restrained as a constant. Grossberg and colleagues later introduced a new form of enhancement and suppression interactions into the network. They simulated neuronal signal processing and storage in networks where each population excited itself and inhibited the others (Ellias and Grossberg 1975; Grossberg 1976). A more attractive structure was formulated by Bienenstock et al. (1982) in which the ability of synaptic modification varied as a nonlinear function of corresponding input signal as well as postsynaptic activity. The direction and degree of changes in synaptic strength were governed by both the intensity of input signal and the activity of postsynaptic neuron (x and y, respectively). The Hebbian rule was modified following the relationship $\Delta w_i \propto (x-\theta_x)(y-\theta_y)$ (for a review see Miller 1996), where θ_x and θ_y represent the modification threshold of presynaptic input and postsynaptic output, respectively. In this model, potentiation occurs only when both x and y are above their thresholds, whereas depression should be induced when one is below its threshold and another above. An additional, but important, assumption of the Bienenstock model is that the threshold values are not fixed but rather fluctuate according to the history and previous activity of the presynaptic and postsynaptic neurons. Such a property of synaptic connection has been termed "metaplasticity," which means that the modifiability of the synaptic strength is changeable (Miller 1996). Thus, as a result of stimulation-induced increase, θ may shift to promote synaptic depression, and vice versa.

Extending Bienenstock's model, which describes the metaplasticity in a single synapse, we developed a more complicated nonlinear structure to describe the multi-signal interactions in the outer plexiform layer of carp retina (Eq. 4). Self-organizing processes were proposed. The strength change of a certain population of synapses was determined by the combination of two major factors. Instead of a "serial-additive" structure proposed by Miller, the concept of "metaplasticity" is presented in

►

Fig. 3. The calculated spatiotemporal changes of synapses connecting different kinds of cones and LHC. **a** When no stimulus is applied, the weights of synapses connecting both red and green cones and LHC (R and G, respectively) are kept near their static values over time (0, 5, 10, 15, 20 s); only a random fluctuation occurs due to the intrinsic noise $\varepsilon(t,i,j)$. **b** During the 20-s repetitive red flashes, the synaptic weights between the red cone and LHC are strengthened over time (0, 5, 10, 15, 20 s, from top to bottom) during red flickering and recover to the initial values (0, 5, 10, 15, 20 s, from top to bottom) after the flickering ceased (*left panels*, R); the synapses between the green cone and LHC were weakened during the flickering and recovered to the control level after flickering (*right panels*, G). **c** During the 20-s repetitive green flashes, the weights between the red cone and LHC are moderately decreased (*left panels*), while those between the green cone and LHC are slightly strengthened (*right panels*)


a No stimulation
b Red flickering
c Green flickering
R
G
Development
Recovery
0.200
0.507
0.648
Relative weight
X
Y

this work in a "parallel-multiplicative" form $\Delta w_i \propto (\phi_a x_k - \phi_b x_{\bar{k}})$.

Basically the relationship between the presynaptic input intensity and the postsynaptic activity x_r (in Eq. 4a, for example) weighted by the excitatory factor $\phi_a = (a - x_r)$ determines the self-excitation within the synapse population; the activation status of the other synapse population x_g, weighted by the inhibitory factor $\phi_b = (x_r - b)$, forms a cross-competition effect between the different populations of synapses. In this model, the changeability of the synaptic strength (the first derivative of x_r as given by the equations) is not a constant but a function defined by the stimulus pattern and the synaptic status over time, with the synaptic weights confined by the maximum and minimum values a and b, respectively. A continuous second derivative of the synaptic weight is available, and the model is thus endowed with the property of "metaplasticity," as discussed by previous authors (Bienenstock et al. 1982; Abraham and Bear 1996; Miller 1996).

Given the model structure, the strength change of a certain synapse (x_r, Eq. 4a) predicted by our model is governed by the following factors: (1) the correlation between the two synapse populations ($x_r x_g$), (2) the status of the inspected synapse population (x_r) and that of the other synapse population (x_g), (3) the squared synaptic weight (x_r^2), and (4) a constant value. The results of model fitting are such that the coefficients related to the correlation between the two populations ($x_r x_g$) are negative, which implies that the interaction between the different populations is competitive; the x_r terms are weighted by negative coefficients, while the coefficients of the x_g terms are positive, which suggests that the energy supporting the strengthening of a certain synapse population is not provided by the environment but rather at the expense of the other synapses; x_r^2 terms also have negative coefficients, which implies that the synaptic-weight changes are limited by the synaptic status.

In a theoretical paper, Lisman explored the possibility that the weight of a synapse could be regulated by Ca^{2+}/Calmodulin Kinase II (CaM-KII), a cytoplasmic structure that directly abuts the receptors within its postsynaptic dendritic spine (Lisman 1989). Recent works have shown that the elevation of Ca^{2+} induced by tetanus stimuli could trigger bidirectional modifications of synaptic weight – potentiation occurs when the postsynaptic Ca^{2+} is enhanced to a high level, whereas a modest Ca^{2+} increase will result in a decrease in synaptic weight (Nishiyama et al. 2000). It is possible that the properties of synapses in the red-sensitive and green-sensitive pathways are different. Thus, when a certain flash stimulus is applied, local Ca^{2+} via Ca^{2+} influx may be enhanced and Ca^{2+} concentration increased. However, during the process there will be regional differences in intracellular calcium concentration. It may be higher in some regions, particularly those that are postsynaptic to a certain group of cone system. This leads to the strengthening of one group of synapses while at the same time to the weakening of another group of synapses.

Taken together, our results suggest that the stimulus-pattern-related horizontal cell spectral plasticity is likely to be attributed to the activity-dependent synaptic-strength modification in the outer plexiform of retina and that the activity-dependent processes are conservative, i.e., the strengthening of the stimulated synapses is compensated by the weakening of the others.

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