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A model for self-organization of receptive fields and orientation-selective columns in the striate cortex

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SUMMARY

In area 17 of the cat visual cortex, simple cells form a hypercolumn in which the optimum orientation from one column to the next gradually changes, composing a complete set of orientation-selective columns (orientation column). This article proposes a model for the development of the bar-shape receptive field of a simple cell and the self-organization of orientation columns. The receptive field of an immature cell in area 17 is assumed to be composed of a circular center and surrounding regions whose synaptic modification rules are different. The synaptic modifications also differ depending on whether the response of a cell is locally maximal or not. The modification of the efficacy of both excitatory and inhibitory synapses is determined according to the combination of activities of the visual cortical cell and the lateral geniculate neuron. The simulation of this model shows the development of the bar-shape receptive field and the self-organization of orientation columns of more than one cycle from 0° to 180°. The abnormal presentations of visual stimuli to this model result in the abnormal development of the orientation column. These simulation results are in good agreement with reported experimental results. Possible neural circuits to achieve this model are proposed. The neural circuits for the synaptic modification are built on the assumption that cortical cells release molecules to modify synaptic efficacies. The neural circuits for the detection of the maximally responding cell are composed of two kinds of inhibitory interneurons. The bar-shape receptive field is assumed to be a consequence of the topographic projection of visual afferents, radial branching of dendrites of a simple cell, and the existence of an inhibitory interneuron.

INTRODUCTION

Simple cells in the cat striate cortex selectively respond to a light or dark bar of specific orientation [17,19]. These orientation-selective cells form columns in which the optimum orientation from one column to the next gradually changes, composing a complete set of orientation-selective columns (orientation column) from 0° to 180° [21,22]. A simple cell receives signals monosynaptically from axons of the lateral geniculate nucleus (LGN) [16,38,39]. The receptive field of a simple cell comprises the elongated center region flanked by two antagonistic fields on both sides of the center region [15], while the receptive field of an LGN neuron is composed of a circular center region and a surrounding circular region which is antagonistic to the center region. The organiza-

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tion of the receptive field and the orientation selectivity of a simple cell are suggested to be consequences of the inhibitory intracortical neural connection [1,25,34,35,40].

In kittens, these orientation-selective cells compose only a part of the total cell population before 2–3 postnatal weeks, after which the population of the orientation-selective cells gradually increases when the kittens experience normal vison during a critical period [5,11]. Kittens reared with abnormal visual experience in which only vertical bars are exposed to them only have cells which respond to the vertical direction [4,30]. The population of cells which respond to other orientations is very small. These experimental results indicate that the visual cortex has plasticity to reorganize its structure according to visual experience.

Although extensive studies have focused on the elucidation of neural mechanisms for the emergence of the orientation selectivity and the self-organization of orientation columns, we have not yet reached this objective, and the following questions are left to be answered: (1) Through what neural mechanisms does a simple cell acquire orientation selectivity, starting with the immature organization of the receptive field? (2) Through what neural mechanisms do orientation columns self-organize? (3) What is the role of prenatally existing orientation-selective cells? Are these cells indispensable for the development of orientation columns?

In this report, the development of orientation-selective simple cells is first discussed and, next, a model for the self-organization of orientation columns is proposed. Finally, possible neural circuits for the present model are proposed. Many models for the formation of receptive fields [27,33] and the self-organization of orientation-selective columns [28,42] of simple cells have been proposed. In these models, however, the two kinds of maturation were modeled separately or as a sequential process. The aim of the present study is to create a model for both the development of orientation-selective cells and the self-organization of orientation columns from a single theoretical basis, in which the two kinds of maturation proceed in a parallel process. Although the edge-type receptive fields are also reported in addition to the bar-type receptive fields, the present model is only concerned with the bar-type receptive field.

METHODS

The model is created using the reported results of anatomical and physiological observations from many laboratories together with plausible assumptions, as described below in Parts I–III. The degree of change in the excitatory and inhibitory synaptic efficacies for each stimulus presentation is arbitrarily determined, and the dependency of the simulation results of the model on parameter values is examined.

In the model proposed here, the synaptic modifications are assumed to be determined by a combination of activities (i.e., firing or not firing) of an LGN neuron and a cortical cell, whatever the threshold value of firing of each neuron is. In addition, it is also assumed that only cortical cells whose response is locally maximal can make synaptic modifications to themselves and their neighbors. Therefore, the detailed characteristics of each neuron are not considered in the present model.

The number of columns in an orientation column is 6 because of the limited computational power. There is one simple cell for each column in the calculation. For simplicity, the orientation of a stimulating light bar is selected to be from 0° to 150° with 30° intervals. A light bar is presented with a random sequence of orientation. Within 6 presentations of a light bar, however, all orientations from 0° to 150° appear

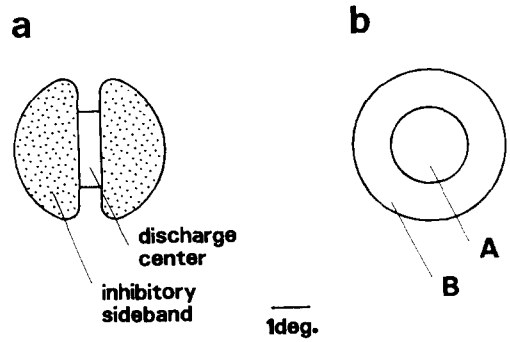

Fig. 1. The receptive field of a mature and an immature cell. (a) The receptive field of a mature simple cell reported to be composed of a rectangular discharge center and two antagonistic inhibitory sidebands flanking it. (b) The receptive field of an immature cell is assumed to be circular. In the early stage of postnatal life, the center (A) and surrounding (B) regions respond to stimuli equally to all orientations. However, the synaptic modification rule for these two regions is assumed to be different, and the receptive field gradually develops to a mature organization. The scale bar shows the visual angle of 1°.

in order to stimulate cells to all orientations with equal probability. The simulation was performed on a 32-bit personal computer.

Part I. Development of receptive fields of simple cells

Model creation

1. Neural connections from retina to striate cortex The optic nerve fibers are relayed by LGN neurons on the way to the striate cortex. The shape and size of the receptive field of LGN neurons in cats are almost the same as those of retinal ganglion cells [7,12,18]. Therefore, it is assumed in this report that there are one-to-one mappings in the LGN from optic nerve fibers to LGN neurons.

The second assumption in the present model is the topographic mapping: i.e., the two neighboring ganglion cells in the retina project their axons to the two neighboring LGN neurons, and these LGN neurons project their axons to the two neighboring cortical cells in the striate cortex. The third assumption is that a simple cell receives both excitatory and inhibitory input from one LGN neuron. Ferster and Lindstrom [9] reported that cortical neurons receive excitation and inhibition from the same type of geniculate afferents. This supports the third assumption.

2. Receptive field The receptive field of a mature simple cell is composed of a rectangular discharge center of on-response flanked by antagonistic inhibitory sidebands on both sides of the discharge center (Fig. 1a) [15]. The orientation selectivity is a consequence of this organization of the receptive field. This mature receptive field of a simple cell develops from an immature non-orientation-selective organization. When a cat is in the early stage of postnatal period or reared in the dark, visual cortical cells respond almost equally to two different orientations 90° apart from each other [5,41]. This suggests that the shape of the receptive field of immature cells is circular. Furthermore, if cortical cells in the newborn cat can develop into a simple cell which responds to any orientation, the initial shape of the discharge center should be circular. Therefore, the shape of the receptive field of immature cortical cells is assumed to be circular (Fig. 1b). This circular receptive field is assumed to be divided into two regions: the internal circular region (A) and the surrounding region (B). A part of region A develops into the

rectangular discharge center, and the remaining part of this region develops into the inhibitory sidebands. Region B develops into the inhibitory sidebands.

3. Synaptic modification The receptive field of a cortical cell develops from the immature structure to mature organization according to different synaptic modification rules from regions A and B. Although there is no direct evidence for the plasticity of the inhibitory synapse in the visual cortex, the inhibitory synaptic efficacy in addition to the excitatory synaptic efficacy is assumed to be modified during development. Sillito et al. [35] suggested that the orientation selectivity was caused by the intracortical inhibitory mechanism, not by a reduced summation of the excitatory synapses converging on a cell, because when the GABA antagonist *N*-methylbicuculline was applied to the visual cortex, simple cells responded to a stimuli of orthogonal orientation with the same discharge rate as to the optimum orientation. Therefore, immature cortical cells respond to a stimulus of orthogonal orientation without application of the GABA antagonist [5,9]. Therefore, it is likely that the inhibitory synaptic efficacy is modified during development, and finally, simple cells acquire orientation selectivity. The following rules are applied for synaptic modification in the center region A:

Rule 1 When a cortical neuron is not firing, the efficacy of the excitatory and inhibitory synapses is not modified even though the LGN neuron elicits firing. That is, only when a cortical cell is firing, synapses on it are modified.
Rule 2 When a cortical neuron is firing and an LGN neuron is not firing, the efficacy of the excitatory synapse is decreased and that of the inhibitory synapse is increased. This rule decreases the strength of synaptic input of a firing cortical cell when an afferent axon is silent.
Rule 3 When both a cortical neuron and an LGN neuron are firing, the efficacy of the excitatory synapse is increased (Hebbian rule) and that of the inhibitory synapse is decreased.

The synaptic efficacy at $t = t_0$ is changed to a new value at $t = t_1$ according to the above rules. These synaptic modification rules are described by the following equations:

$$ {}_{c}E_{jk}(t_1) = {}_{c}E_{jk}(t_0) + \epsilon f\big(S_j(t_0),\ L_k(t_0)\big), \tag{1} $$

$$ {}_{c}I_{jk}(t_1) = {}_{c}I_{jk}(t_0) + \iota g\big(S_j(t_0),\ L_k(t_0)\big), \tag{2} $$

where ${}_{c}E_{jk}$ and ${}_{c}I_{jk}$ designate the excitatory and inhibitory synaptic efficacies, respectively, in the center region A; ϵ and ι are the increments of the excitatory and inhibitory synaptic efficacies, respectively; $f(S_j(t_0), L_k(t_0))$ and $g(S_j(t_0), L_k(t_0))$ are the determinants of the modification, which are functions of activities of the jth cortical neuron $S_j(t_0)$ and kth LGN neuron $L_k(t_0)$.

$f(S_j(t_0), L_k(t_0))$ and $g(S_j(t_0), L_k(t_0))$ take values of -1, 0 or 1 according to the activities of the cortical neuron and the LGN neuron:

$$
\begin{aligned}
f = \ & 0 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 0 \ (\textit{Rule 1}) \\
& 0 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 1 \ (\textit{Rule 1}) \\
& -1 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 0 \ (\textit{Rule 2}) \\
& 1 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 1 \ (\textit{Rule 3}) \\
g = \ & 0 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 0 \ (\textit{Rule 1}) \\
& 0 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 1 \ (\textit{Rule 1}) \\
& 1 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 0 \ (\textit{Rule 2}) \\
& -1 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 1 \ (\textit{Rule 3}).
\end{aligned}
$$

$S_j = 1$ ($L_k = 1$) and $S_j = 0$ ($L_k = 0$) indicate that a cortical cell (LGN neuron) is active and not active, respectively.

For the surrounding region B, the following rules are assumed:

Rule 4 When the cortical neuron is not firing, the modification rule is the same as that for the center region.

Rule 5 When the cortical neuron is firing and an LGN neuron is not firing, the modification rule is the same as that for the center region.

Rule 6 When both a cortical neuron and an LGN neuron are firing, the efficacy of the excitatory synapse is not changed, and that of the inhibitory synapse is decreased. With this rule, the discharge center develops within region A of an immature cortical cell, even though the stimulus bar length is longer than the diameter of region B.

These synaptic modification rules are described by the following equations:

$$ {}_sE_{jk}(t_1) = {}_sE_{jk}(t_0) + \epsilon f\big(S_j(t_0),\, L_k(t_0)\big), \tag{3} $$

$$ {}_sI_{jk}(t_1) = {}_sI_{jk}(t_0) + \iota g\big(S_j(t_0),\, L_k(t_0)\big), \tag{4} $$

where ${}_sE_{jk}$ and ${}_sI_{jk}$ designate the excitatory and inhibitory synaptic efficacies, respectively, in the surrounding region B. The values of $f(S_j(t_0), L_k(t_0))$ and $g(S_j(t_0), L_k(t_0))$ are determined as follows:

$$
\begin{aligned}
f = \;& 0 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 0 \text{ (\textit{Rule 4})} \\
& 0 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 1 \text{ (\textit{Rule 4})} \\
& -1 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 0 \text{ (\textit{Rule 5})} \\
& 0 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 1 \text{ (\textit{Rule 6}),} \\
g = \;& 0 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 0 \text{ (\textit{Rule 4})} \\
& 0 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 1 \text{ (\textit{Rule 4})} \\
& 1 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 0 \text{ (\textit{Rule 5})} \\
& -1 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 1 \text{ (\textit{Rule 6}).}
\end{aligned}
$$

The important difference between Eqs. (1)–(2) and (3)–(4) is that f and g functions have different values even if the cortical neuron and the LGN neuron are in the same activity states.

Parameters used in the simulation

The diameter of the center region of on-center ganglion cells is reported to be from 0.5° to 3.2° in the cat [12,18]. Almost the same size is reported for LGN neurons [18]. On the other hand, the size of a receptive field of a simple cell is reported to be from 0.25° to 16°[2] [6,13,24]. Receptive fields of both LGN neurons and simple cells become large as eccentricity increases [20]. In this simulation, the receptive field diameter of 2° for the center region A of an immature cortical neuron and $0.6 \times 2°^2$ for the discharge center of a simple cell are used. These values correspond to the fovea in the cat. The diameter of the antagonistic sidebands is reported to be from 1.7 to 9.5° [3]. The diameter of 4° for the sidebands is used in the calculation. The density of ganglion cells in the cat retina is assumed to be $6000/\text{mm}^2$ [14,31]. These values lead to the calculation that one simple cell receives inputs from 575 retinal ganglion cells, assuming the distance from the second nodal point to the surface of the retina to be 5 mm in the cat. The values for

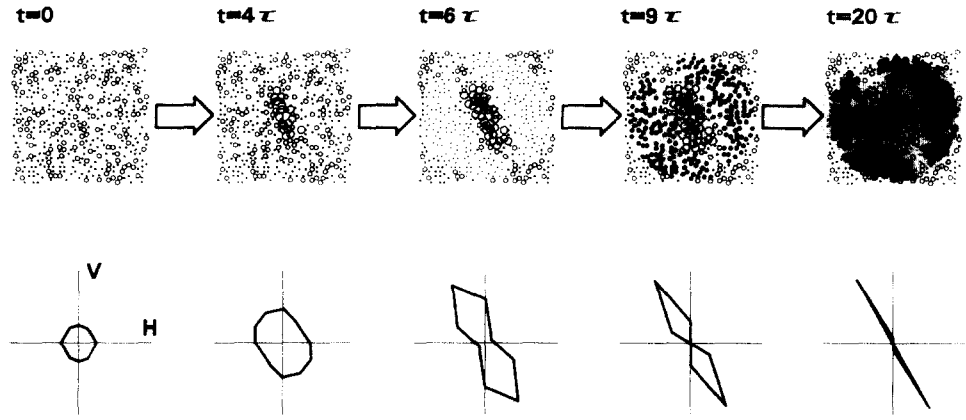

Fig. 2. A simulation of the development of an orientation-selective cell. The upper row shows the development of the receptive field. The large open or closed circles indicate the excitatory or inhibitory input to a cell whose strength is more than 60% of the maximum. The small open or closed circles indicate the excitatory or inhibitory input whose strength is larger than 20% but smaller or equal to 60% of the maximum. The input strength of dots is equal to or smaller than 20% of the maximum. The lower row shows a plot of the orientation selectivity in the polar coordinate. No orientation selectivity is seen at $t = 0$. At $t = 4_\tau$, the orientation selectivity begins to emerge, and at $t = 20_\tau$, the cell is distinctly tuned to one orientation. τ is a time period in which stimuli of all orientations are presented at 30° intervals.

the excitatory and inhibitory synaptic modifications are chosen to be one-tenth of the maximum value of the excitatory synapse for every step of stimulus presentations.

Simulation results

Figure 2 shows the simulation result for the development of a receptive field of a simple cell. Large open and closed circles are the excitatory and inhibitory inputs whose strengths are more than 60% of the maximum. Small open or closed circles have input strength of more than 20% but equal to or smaller than 60% of the maximum, respectively. Dots are equal to or smaller than 20% of the maximum for both excitatory and inhibitory inputs. The stimulus is a light bar of $0.6 \times 6°^2$. As shown in the upper row of Figure 2, the synaptic efficacy gradually changes, and finally the receptive field develops into the mature structure reported by Henry and Bishop [15]. The lower row in Figure 2 is the plot of the orientation selectivity of the simulated simple cell in the polar coordinate. It is clearly seen that the orientation selectivity gradually develops and finally reaches the mature level.

Part II. Self-organization of orientation columns

Model creation

1. Intracortical connections For the self-organization of a complete set of orientation-selective columns of 180° range, the mechanism for the intercolumnar interaction in addition to the development of an orientation-selective column is necessary. This hypothetical mechanism is created taking into account the reported anatomical observation. It was reported that when a recording electrode was penetrated tangentially into the cortical surface, the orderly change in the optimum orientation was seen with the cycle of 239–434°/mm, depending on the penetration direction, in the tree shrew [23] and 180°/mm in the macaque monkey [29]. This structure is also reported in the cat [36]. On the other hand, the arborization of afferent axons of LGN neurons is reported to

extend tangentially to the cortical surface as far as 500–2000 μm in the cat [6,8,37]. Therefore, it is likely that all cortical neurons in an orientation column receive the same input from an afferent axon. This expanded connection between an afferent axon of the LGN neuron and a cortical neuron is assumed in this model.

The following rule is assumed in order to avoid the development of two adjacent columns having the same optimum orientation.

Rule 7 Only the column whose response is maximum within an orientation column can modify synaptic efficacies of cells in this maximally responding column and in the neighboring columns. That is, there is a neural circuit which detects the maximum response within an orientation column.

It was reported that a basket cell, which is thought to be an inhibitory interneuron, extended its axon tangentially to the cortical surface as far as 1000 μm in the cat [37]. Therefore, it is likely that all simple cells in an orientation column receive the same inhibitory input from a basket cell. The basket cell is the most probable candidate for the constituents of the neural circuit of maximum response detection (see Part III).

2. Synaptic modification The synaptic modification in the maximally responding column is performed by the same rules as noted before (*Rules 1–6* above). For the columns neighboring the maximally responding column, the synaptic efficacies are modified according to the combination of activities of the cortical cells and the LGN neuron, as noted in Part I. However, the increment of this synaptic modification is assumed to be a function of the discharge rate of the maximally responding column and the distance from it:

Rule 8 The strength of the synaptic modification for the non-maximally responding cell is decreased as the distance from the maximally responding cell increases. The synaptic modification is also a function of the discharge rate of the maximally responding cell, and the synaptic modification of neighboring cells is strong when the response of the maximally responding cell is large. Therefore, ϵ and ι are functions of the distance r from the maximally responding cell and the discharge rate R of the maximally responding cell.

The synaptic modification rules for the center and surrounding regions of the immature cell are assumed to be different, as noted before. For the center region, the following rules are assumed:

Rule 9 Only when a cortical cell is not firing and an LGN neuron is firing, the efficacy of the excitatory synapse is increased and that of the inhibitory synapse is decreased.

Rule 10 In the other cases of the activity of a cortical neuron and an LGN neuron from *Rule 9*, the synaptic efficacy is not changed.

With *Rules 9 and 10*, the input strength of the adjacent column is increased for the same direction as the maximally responding cell. These synaptic modification rules are described by the following equations:

$$ {}_{c}e_{jk}(t_1) = {}_{c}e_{jk}(t_0) + \epsilon(r, R) f\big(S_j(t_0), L_k(t_0)\big), \tag{5} $$

$$ {}_{c}j_{jk}(t_1) = {}_{c}i_{jk}(t_0) + \iota(r, R) g\big(S_j(t_0), L_k(t_0)\big), \tag{6} $$

where ${}_{c}e_{jk}$ and ${}_{c}i_{jk}$ designate the excitatory and inhibitory synaptic efficacies in the center region A of the non-maximally responding cell, respectively. $f(S_j(t_0), L_k(t_0))$ and $g(S_j(t_0), L_k(t_0))$ are determined as follows:

$$
\begin{aligned}
f = \;& 0 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 0 \;(\textit{Rule 10}) \\
& 1 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 1 \;(\textit{Rule 9}) \\
& 0 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 0 \;(\textit{Rule 10}) \\
& 0 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 1 \;(\textit{Rule 10}) \\
g = \;& 0 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 0 \;(\textit{Rule 10}) \\
& -1 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 1 \;(\textit{Rule 9}) \\
& 0 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 0 \;(\textit{Rule 10}) \\
& 0 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 1 \;(\textit{Rule 10}).
\end{aligned}
$$

For the surrounding region, the following rules are assumed:

Rule 11 Only when a cortical cell is not firing and an LGN neuron is firing, the efficacy of the excitatory synapse is decreased and that of the inhibitory synapse is increased.

Rule 12 In the other cases, the synaptic efficacy is not changed.

With *Rules 11 and 12*, the inhibition in region B of the adjacent cell is increased in order to keep the response of this cell from becoming larger than that of the maximally responding cell. These synaptic modification rules are described by the following equations:

$$
{}_{s}e_{jk}(t_1) = {}_{s}e_{jk}(t_0) + \epsilon(r, R) f\big(S_j(t_0), L_k(t_0)\big), \tag{7}
$$

$$
{}_{s}i_{jk}(t_1) = {}_{s}i_{jk}(t_0) + \iota(r, R) g\big(S_j(t_0), L_k(t_0)\big), \tag{8}
$$

$$
\begin{aligned}
f = \;& 0 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 0 \;(\textit{Rule 12}) \\
& -1 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 1 \;(\textit{Rule 11}) \\
& 0 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 0 \;(\textit{Rule 12}) \\
& 0 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 1 \;(\textit{Rule 12}), \\
g = \;& 0 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 0 \;(\textit{Rule 12}) \\
& 1 \quad \text{when } S_j(t_0) = 0 \text{ and } L_k(t_0) = 1 \;(\textit{Rule 11}) \\
& 0 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 0 \;(\textit{Rule 12}) \\
& 0 \quad \text{when } S_j(t_0) = 1 \text{ and } L_k(t_0) = 1 \;(\textit{Rule 12}).
\end{aligned}
$$

In Table I, these modification rules of excitatory and inhibitory synaptic efficacies are summarized. In this table, upward and downward arrows designate the increase and decrease of the synaptic efficacy, respectively. Right-facing arrows designate no modification.

There is a small fraction of orientation-selective cells at the time of birth in the kitten[5]. These cells are thought to be guides for the development of the orientation-selective columns. It is also assumed in this model that the innately mature cell in an orientation column guides the self-organization of the orientation columns.

In order to further clarify the proposed hypothetical mechanisms, a schematical representation of the self-organization of the orientation column is shown in Figure 3. At the initial state ($t = 0$), no column, except column C_n, has orientation selectivity. Column C_n corresponds to the innately mature cell in the kitten. Dots in the circular

TABLE I

MODIFICATION RULES FOR EXCITATORY AND INHIBITORY SYNAPSES IN THE MODEL

Receptive field area	Activity		Synaptic modification			
	Simple cell	LGN axon	For R_{max} col.		For adj. col.	
			Exc	Inh	Exc	Inh
Center	non-act.	non-act.	→	→	→	→
(region A)	non-act.	act.	→	→	↑	↓
	act.	non-act.	↓	↑	→	→
	act.	act.	↑	↓	→	→
Surrounding	non-act.	non-act.	→	→	→	→
(region B)	non-act.	act.	→	→	↓	↑
	act.	non-act.	↓	↑	→	→
	act.	act.	→	↓	→	→

The synaptic modification is different with different combinations of the activities of the visual afferent axon and the simple cell. In addition, the modification rule is different for the center and the surrounding region of the receptive field of a simple cell. It is also different for cells in the maximally responding column and the non-maximally responding columns. The upward and downward arrows indicate the increase and decrease of the efficacy of both excitatory and inhibitory synapses, respectively. The right-facing arrow indicates no modification in the efficacy.

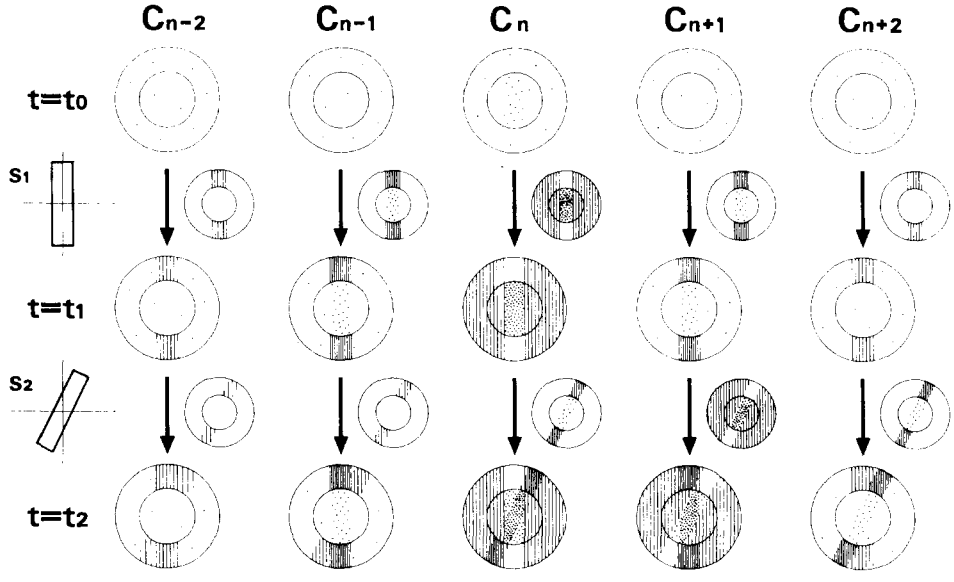

Fig. 3. A schematic drawing of the development of orientation-selective columns according to the synaptic modification rules proposed in this report. C_n is the innately mature column in this drawing. At the initial stage ($t = t_0$), all columns from C_{n-2} to C_{n+2} except C_n have no orientation selectivity and they respond slightly to all orientations. This is indicated by dots of low density. When the stimulus of S_1 is presented, the synapse of simple cells in these columns is modified according to the rules. The modification is shown by the small circles drawn at the right-hand side of the arrows. In this example, column C_n maximally responds to S_1, and the receptive fields of C_{n-2} to C_{n+2} result in the synaptic organization shown at $t = t_1$. The hatched area indicates the inhibitory modification or the connection of visual afferent axons to a simple cell. The more heavily the dots or hatches are drawn, the stronger the excitatory or inhibitory action to a simple cell. When the second stimulus S_2 is presented, the maximally responding column is C_{n+1} and the synapses are modified according to the rule. Thus, the sequentially ordered orientation column develops. The presented stimuli (S_1 and S_2) are sequentially ordered in this figure for ease of explanation and understanding. However, the stimulus orientation is random in the actual simulation.

receptive fields indicate the excitatory input to the cell. In this example, the optimum orientation of column C_n is vertical. When a vertical light bar is presented at $t = 0$, the maximally responding column is C_n. This column modifies synaptic efficacy in itself and its neighboring columns according to the hypothetical mechanisms described above, resulting in the next state of synaptic efficacy ($t = t_1$). The small circular receptive fields beside downward arrows show the synaptic modifications of cells in orientation-selective columns. Dots and hatching in these small receptive fields indicate the increase of the excitatory and inhibitory inputs to cells, respectively. The stronger the synaptic modification is, the more heavily are the dots and hatchings drawn in this figure. At $t = t_2$, when a stimulus S_2 of slightly different orientation to S_1 is presented, a cell in the column adjacent to the previously maximally responded one (C_n) will be the maximally responding cell.

In general, stimuli are not necessarily presented in a sequential order. Therefore, in the simulation described later, the stimuli are presented in random sequence (see 'Methods'). In Figure 3, however, the orientation at $t = t_2$ is set to be slightly different from that at $t = 0$ for ease of explanation and understanding.

Cells in two columns, C_{n+1} and C_{n-1}, can respond greatly to this second stimulus. However, there is only one maximally responding column. In Figure 3, the maximally responding column to S_2 is assumed to be C_{n+1}. When the next stimulus, which is more inclined than S_2 from the vertical, is presented, column C_{n+2} will be the maximally responding column. Thus, orderly sequenced orientation-selective columns develop.

Function of $\epsilon(r, R)$ and $\iota(r, R)$ and parameters used in the simulation

As noted before, the synaptic modification strengths (ϵ and ι) for non-maximally responding cells are a function of the distance from the maximally responding column and the discharge rate of the maximally responding cell. In the present simulation, ϵ and ι are assumed to be expressed as:

$$\epsilon = (\epsilon_0/r^m)(R/R_{MAX}), \quad \iota = (\iota_0/r^n)(R/R_{MAX}) \quad (m, n = 1, 2, 3, \ldots) \tag{9}$$

$$r = N_{MAX} - N,$$

where N_{MAX} and N are the column number of the maximally responding column and that of the adjacent column, respectively, and R_{MAX} is the largest possible response of cells. Since there are no experimental data on how fast ϵ and ι decrease as the distance from the maximally responding column increases, the dependence of column formation on the values of n and m was simulated. Figure 4 shows this result for one cycle of an orientation column. As m increases, the fraction of complete column formation increases (Fig. 4a). This indicates that the fraction becomes large if the excitatory synaptic modification by the maximally responding column is localized around it. Therefore, the excitatory synaptic modification by the maximally responding column is performed only on the adjacent two columns on both sides in the present simulation. On the other hand, the fraction decreases as n increases (Fig. 4b). This indicates that the fraction becomes large if the inhibitory synaptic modification by the maximally responding column is delocalized. If $n = 0$, however, the inhibitory synaptic modification is performed on the far column with the same strength as on the one adjacent to the maximally responding column. This will not be the case, because this will deteriorate the formation of orientation columns of more than one cycle. From Figure 4b, the fraction is almost saturated when $n = 4$. Thus an n-value of 4 is used in this simulation.

The maximum increment values of the excitatory and inhibitory synapses for the maximally responding cell are equal to those used in the simulation of the development

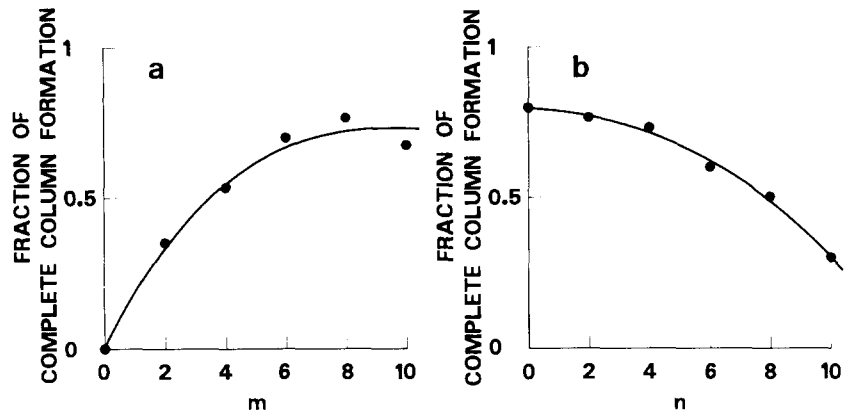

Fig. 4. The dependence of the formation of the orientation column on m and n. (a) As m increases, the fraction of complete column formation increases. This indicates that column formation is stabilized when the synaptic modification by the maximally responding cell is localized. (b) As n increases, the fraction decreases. This indicates that column formation is unstable when the synaptic modification by the maximally responding cell is localized.

of receptive fields of simple cells. The maximum increment values for the non-maximally responding cell are set at 20 times that of the maximally responding cell for the excitatory synapse and 0.1 times for the inhibitory synapse. The maximum value for the inhibitory synaptic efficacy is set at half that of the excitatory synapse.

Simulation results

Figure 5 shows a simulation result of the self-organization of the orientation selectivity for 6 columns. Column 3 is selected as the innately mature column. All other columns have random values of excitatory synaptic efficacy from 0% to 30% of the maximum at $t = 0$. The inhibitory synaptic efficacy is zero at $t = 0$ in the simulation. τ is the simulation time period in which all orientations of a stimulating bar are presented to the model. At $t = 3\tau$, orientation selectivity begins to emerge and, at the same time, the

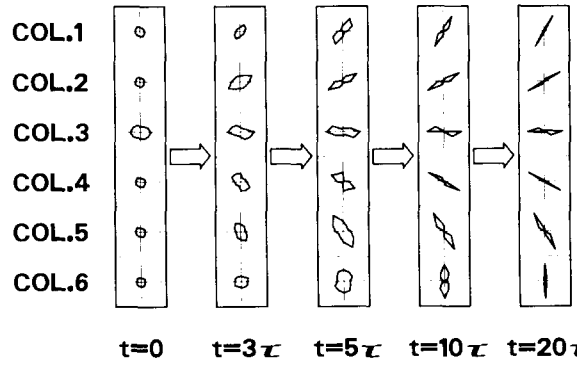

Fig. 5. Simulation results of the self-organization of orientation-selective columns. The stimuli are randomly presented in 30° intervals to 6 columns. However, the presentation frequencies were set equal in all orientations. All orientations are detected by the 6 columns. Column 3 includes an innately mature cell. At $t = 3\tau$, orientation selectivity already emerges in all columns. However, the maximally responding orientation of Column 6 at this stage is different from that in the developed stage of $t = 20\tau$. The meaning of τ is the same as in Fig. 2.

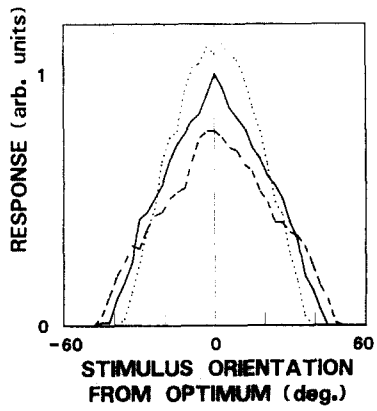

Fig. 6. A simulated orientation tuning curve. The shape of the orientation tuning curve is more triangular than bell-shaped. The cell response approaches the basal level when the stimulus orientation is rotated more than 40° from the optimum. The continuous line shows the tuning curve for the stimulus whose width is the same as that of the discharge center of the receptive field. If the width of the stimuli is widened, the orientation tuning curve becomes sharp (dotted line), while if it is narrowed, the tuning curve becomes broad (broken line).

responses become large. The tendency of the organization of orientation columns is already seen at this time. However, the orientation of maximum response in Column 6 is different from that of the mature one at $t = 20\tau$. At $t = 5\tau$, the orientation of the maximum response for each column becomes the same as that at the mature state. However, the orientation selectivity is still incomplete. At $t = 20\tau$, all columns become mature and have developed orientation selectivity with clockwise rotation in this simulation. Thus, the complete set of orientation-selective columns develops.

Figure 6 shows the simulated orientation tuning curve of a cell. The response is represented in arbitrary units, and the zero response corresponds to a resting level of the cell activity. Cells with spontaneous activity will still discharge even when the response in this figure is zero. The response decreases as the stimulus orientation is rotated from the optimal one. When the stimulus orientation is rotated more than 40° from the optimum orientation, the cell response becomes almost the same as the resting level. The continuous line is the response for the stimulus width of 0.4°, which is the same as the width of the excitatory discharge center of the receptive field. The dotted and the broken lines are the orientation tuning curve for the stimulus widths of 0.8° and 0.2°, respectively. As the stimulus is widened, the orientation tuning curve becomes narrower. The shape of the tuning curve is triangular rather than bell-shaped. This corresponds to the experimental results by Henry and Bishop [15].

The proposed algorithm of the self-organization of orientation columns can be expanded to the formation of columns which have more than one cycle of orientation selectivity. Figure 7 shows the simulation result of the self-organization of 3 cycles of orientation columns. The orientation selectivity develops from Column 4, which was selected as the innately mature column, to both sides of it. The response of Column 18 begins to grow earlier than Column 17. This is because the synaptic modification of Column 18 was performed by Column 1, and vice versa, Column 1 was modified by Column 18 in this simulation. The initial strength of synaptic efficacy was set at zero except in the innately mature column. In this simulation, two columns adjacent to the

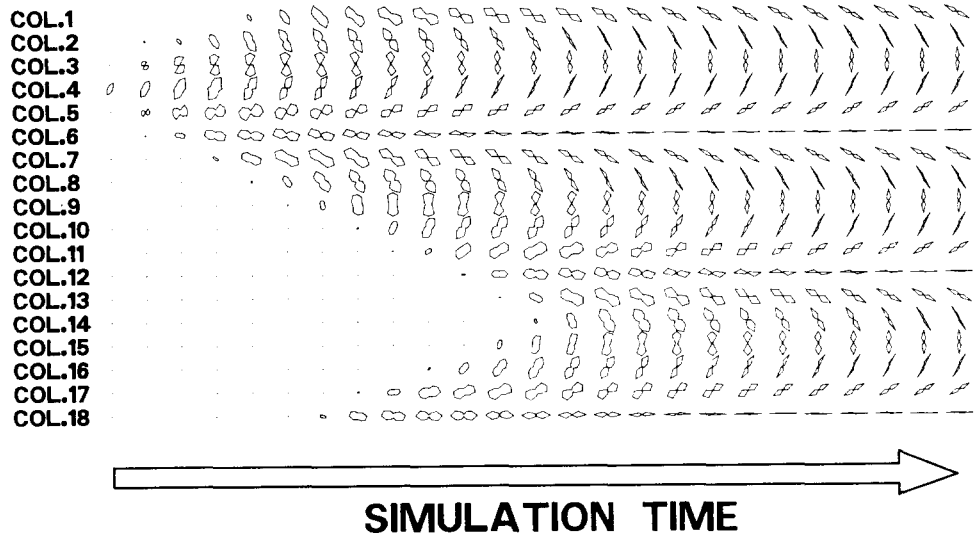

Fig. 7. The development of orientation-selective columns in 3 cycles of orientation columns. There was only one innately mature column (Column 4) in this simulation. It is seen that the orientation selectivity of an ordered sequence develops as the simulation time increases.

innately mature one (Columns 3 and 5) have inhibitory input in the surrounding region B of the immature receptive field at the initial stage (Fig. 1a).

It is reported that there are considerable amounts of visually responsive and non-orientation-selective cells at the time of birth in cats [5]. Figure 8 shows a simulation result for this case where there is one innately mature column (Column 16), and the others are visually responsive and non-orientation-selective cells at the initial stage of the simulation. The orientation selectivity for columns near Column 16 develops to a mature level with clockwise rotation (closed bar on the right-hand side of Fig. 8) as in Figure 7. However, columns located far from the innately mature one (Columns 2–11) begin to develop without the influence of sequentially developing neighboring columns, and result in irregular orientation selectivity. Columns 2–7 develop to a mature level with counterclockwise rotation (open bar), which is the opposite rotation from that of the orientation column near Column 16, and there is an abrupt change of 90° in orientation selectivity between Columns 1 and 2 (arrow). Most columns from Columns 8–13 do not develop to a mature level and their orientation selectivities stay at the immature level. Columns 8 and 11 still respond strongly to all orientations (circles). The orientation selectivity of Column 13 is interesting. It responds to two different orientations which are almost orthogonal to each other (asterisk), showing cross-shaped orientation selectivity. Thus, the orientation selectivity of columns in the developed state is very irregularly organized in this simulation if there are many innately visually responsive cells in columns.

If there is no innately mature column in the simulation, the development of orientation-selective columns will be much more irregular than that in Figure 8. It is obvious that no orientation-selective column develops when the initial value of the excitatory synaptic efficacy is zero. On the contrary, in the case where each cell is visually responsive but not orientation-selective at the initial stage, orientation selectivity develops (left row of Fig. 9b). At the end of the simulation (right row), one orientation column (a closed bar) develops in this simulation. However, there are many immature

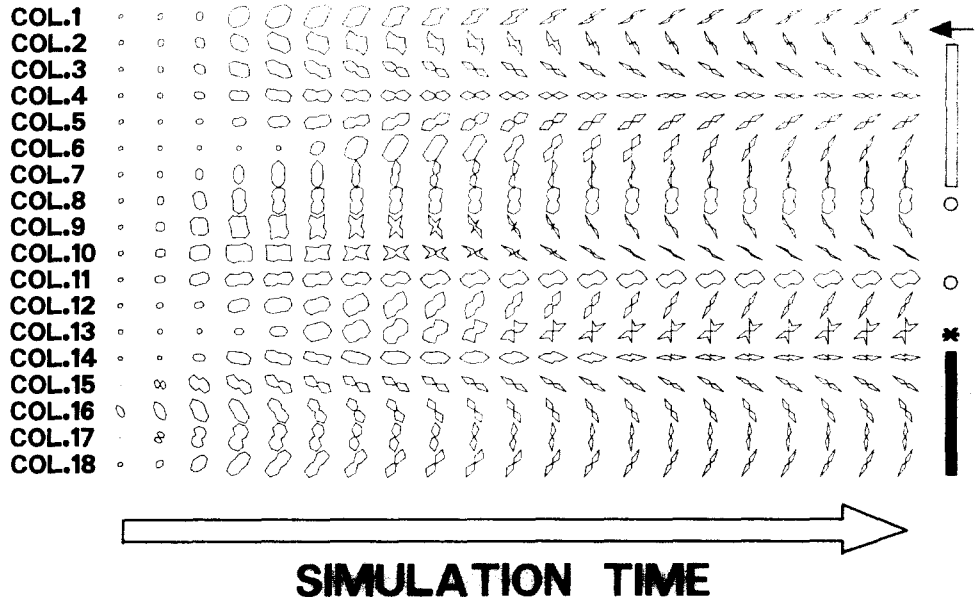

Fig. 8. The development of orientation-selective columns in 3 cycles of orientation columns with many innately visually responsive cells. Some orientation-selective columns develop as the simulation time increases. However, there are two cycles of orientation columns which have clockwise (black bar) and counterclockwise (white bar) sequences of orientation selectivity. There is a 90° difference in the optimum orientation between two adjacent columns at the position indicated by an arrow. The column which is indicated by an asterisk has orientation selectivity to two different orientations. The orientation selectivity of the columns indicated by open circles is not developed to a mature level.

cells (circles) and cells of cross-shaped orientation selectivity (asterisks). Thus, the orientation selectivity is irregular in this situation.

It is reported that the orientation selectivity of kittens with abnormal visual experience was irregular in comparison to that of normally reared ones [4,30]. Figures 9c and 9d are the simulation results when only two stimulating orientations of 90° differences were presented. No visually responsive cell, except in Column 15, was set at the initial stage in Figure 9c. On the contrary, many initially visually responsive cells were set in Figure 9d. For both cases, some cells develop to the mature level. However, many cells are immature (circles) or not developed. In addition to this, there are many cells with cross-shaped orientation selectivity (asterisks).

In the case in which only the stimulus with vertical orientation is presented, the column development is also abnormal. If there is no visually responsive cell except an innately mature one, only a few cells near the innately mature cell develops, being responsive to the vertical direction (Fig. 9e). The rest of the columns do not develop from the initial stage. Even if there are visually responsive cells in the initial stage (Fig. 9f), the result in the development of orientation columns is almost the same as in Fig. 9e. In both cases no cell which responds to orientation other than vertical can be found.

From the assumed organization of the receptive field of immature cells, as shown in Figure 1b, it is easily surmised that the self-organization of the sequentially ordered orientation-selective columns is strongly dependent on the width of the stimulating light bar and the radius of region A in Figure 1b. Figure 10a shows the dependence of the column formation on the width of the stimulating light bar for 3 cycles of orientation

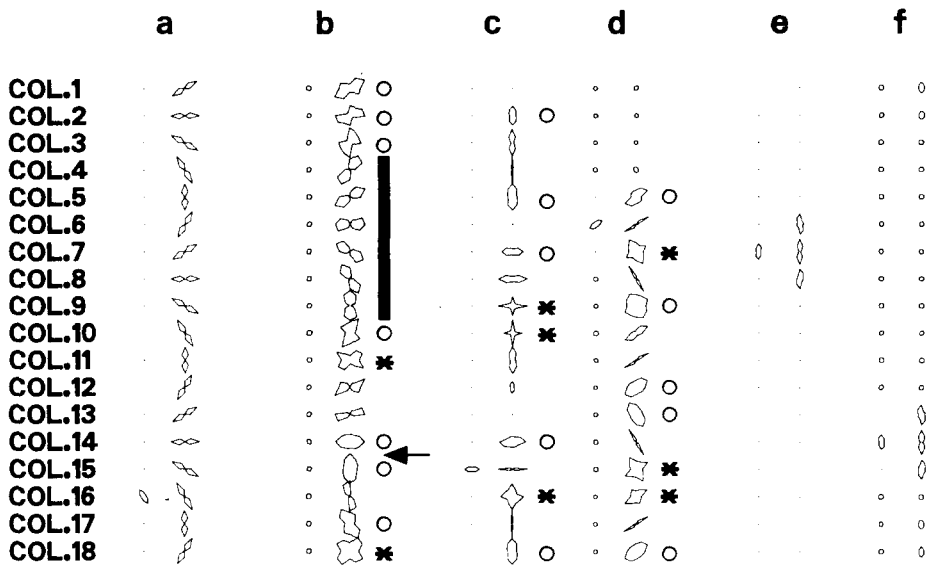

Fig. 9. The development of orientation-selective columns in the abnormal condition. (a) The development of orientation-selective columns in the normal condition for reference. The orientation selectivity is broad in the developed stage (right row) in this simulation because of the different simulation parameter from that in Fig. 7. (b) The development of orientation-selective columns without an innately mature cell. At the developed stage, there are portions within columns where orientation selectivity is well organized (black bar). However, the other columns develop abnormally with broadly tuned cells (open circles) and cells responding to two different stimulus orientations. There is also a 90° difference in the optimum orientation between two adjacent columns (an arrow). (c) The development of orientation-selective columns when the stimuli are only horizontal and vertical. The orientation selectivity of mature columns is either horizontal or vertical. There are columns whose orientation selectivity is cross-shaped. (d) The development of orientation-selective columns with many innately visually responsive cells when the stimuli are only horizontal or vertical. (e) The development of orientation-selective columns when the stimulus is only vertical. Only a few columns near the innately mature one develop. Moreover, all of them respond to the same vertical orientation, and their response is immature. (f) The development of orientation-selective columns with many innately visual responsive cells when the stimulus is only vertical.

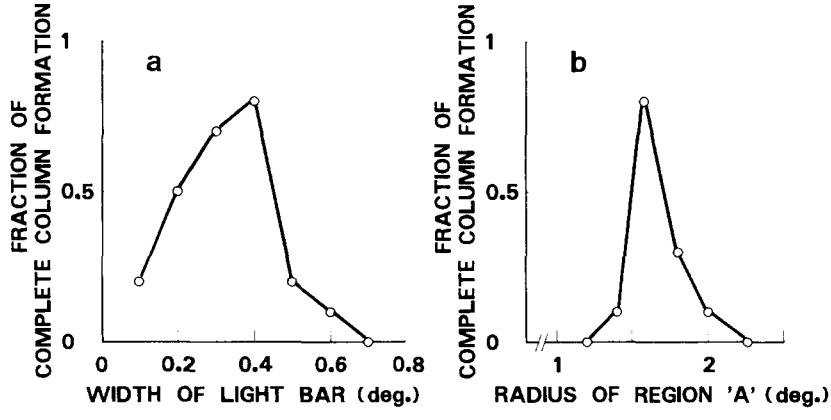

Fig. 10. The dependency of the development of orientation-selective columns on the width of the stimulating light bar and the radius of region A in Fig. 1. (a) The fraction of the development of the complete orientation-selective columns is maximum when the width of the stimuli is the same as that of the discharge center. When the width of the stimuli is widened or narrowed from the optimum one, the fraction decreases steeply. (b) The fraction is also steeply decreased when the radius of region A is increased or decreased from the optimum one.

columns. The column formation was classified as incomplete when there was any column which was not properly orientation-selective. When the bar width is wider than 0.4°, the fraction of complete column formation abruptly decreases. This is because the area of inhibition in region B (Fig. 1b) of the column adjacent to the maximally responding one is overlapped by the excitation by the stimulus of adjacent orientation. This leads to the result that the adjacent column is not the maximally responding one for the stimulus of adjacent orientation. On the other hand, as the bar width is narrowed, the fraction is also decreased. This is because the overlapping area of the excitation for the adjacent column with the stimulus of adjacent orientation becomes small, and the response of the adjacent column to this stimulus is not necessarily maximum within an orientation column because of the fluctuation of the number of exciting ganglion cells.

The fraction of complete column formation is also strongly dependent on the radius of region A (Fig. 10b). The fraction decreases as the radius increases. This is because the inhibition in area B becomes small as the radius increases, and the column adjacent to the maximally responding one also responds to the same orientation. When the radius is decreased, the fraction is again decreased for the same reason as in the case where the width of the light bar is widened.

Part III. Modeling of cortical circuitry

The neural circuits which are engaged in the development of orientation-selective columns are not yet fully understood. In this section, neural circuits which can realize the proposed model are presented. The neural circuits proposed here are examples taken from among many possibilities. The aim of this section is to show the possible mechanisms for the synaptic modification rules.

Circuitry of maximum response detection

An important assumption in the present model is that the synaptic modification for the maximally responding column is different from that for the non-maximally responding columns. In order to achieve these two different synaptic modifications, a neural circuit which can detect the maximum response is needed. An example of this neural circuit is shown in Figure 11. An intracortical neuron M in column C_n receives input from simple cells of other columns on its dendrite (thick lines in Fig. 11). It also receives inhibitory input on the soma from simple cell S in column C_n via inhibitory interneuron I_m. The axon of neuron M produces inhibitory synapse on the soma of S in the same column. When S in column C_n is not the maximally responding cell, there is a stronger input than the inhibitory input from I_m on the dendrite of M from the maximally responding cell, and M will fire and inhibit S. Thus, S will not fire. On the other hand, when S is the maximally responding cell, strong inhibition will be given to M via I_m, and M will not fire. In this case, the simple cell S does not receive inhibitory input from M and continues to fire. Thus, only the maximally responding cell continues to fire.

The neural circuit in Figure 11 does not coincide with the observation that the simple cell receives monosynaptic excitatory input and disynaptic inhibitory input from axons of LGN neurons. One possible explanation is that intracortical neurons I_m and M function only during the developmental period, and after the cells become mature, these neurons or their synapses do not function.

Circuitry of synaptic modification

Other important assumptions in the present model are rules for the synaptic modification. Only when the simple cell responds maximally do the excitatory synapses at the center region of the receptive field follow the modification of the Hebbian rule

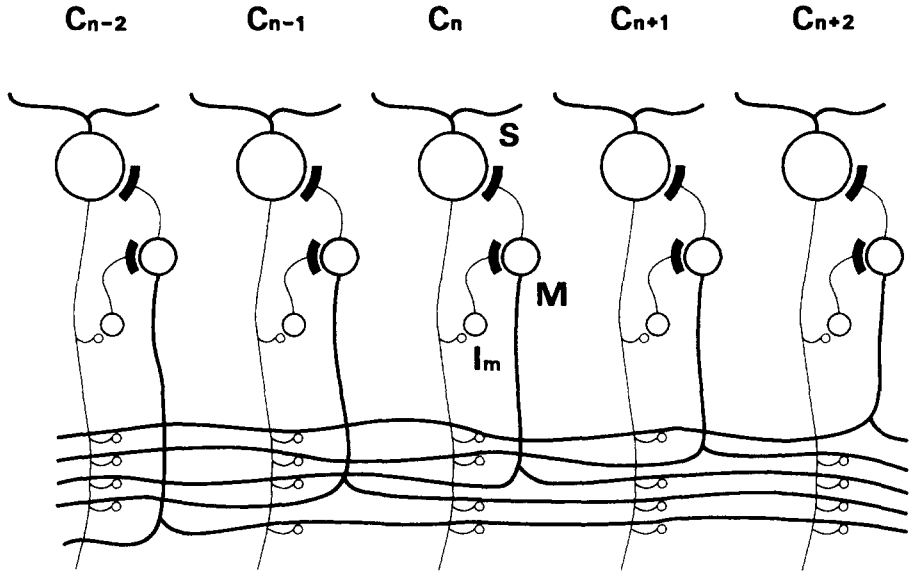

Fig. 11. A neural circuit which detects the maximally responding cell. The interneuron M receives excitatory input from many simple cells in columns different from the one to which M belongs. M also receives an inhibitory input from the simple cell of the same column via inhibitory interneuron I_m. If the simple cell S in column C_n is the maximally responding one, the activity of M is inhibited by I_m and S will continue to fire. On the contrary, if S in C_n is not the maximally responding one, M receives stronger input from other simple cells on its dendrite than the inhibitory input from I_m, and M inhibits S. The firing of S no longer continues. Only the simple cell whose response is maximum can continue to fire in this circuit.

(Table I). The other type of synaptic modification is newly assumed in the present model. Each of these synapses can possibly be related to unknown substances which contribute to the synaptic modification according to the intracellular states both in pre- and postsynaptic cells. On the other hand, such synaptic modification can be made by neural circuits. In this case, the modification agents are released into the synaptic gap from the synaptic terminals of neurons which modify the efficacy. Possible neural circuits for each synaptic modification are shown in Figure 12, where (a) to (c) are circuits for the synaptic modification of the maximally responding cell, and (d) to (g) are circuits for the non-maximally responding cell.

In Figure 12a, the neural circuit for the excitatory synapse in the center region of the receptive field including the Hebbian rule is shown. The afferent axon from an LGN neuron makes the excitatory synapse on the dendrite (thick lines in this figure) of simple cell S. The hatched neuron I_{S1}, whose presynaptic terminal is indicated by the thin line, is the interneuron which increases the synaptic efficacy of signal transmission from an LGN neuron to S. This increase can possibly be induced by releasing substances which increase the synaptic efficacy into synaptic gaps. The firing threshold of I_{S1} is high and it fires only when the LGN neuron and S fire simultaneously. The efficacy of the synapse of an LGN neuron to S is decreased by the interneuron I_{S2}, whose postsynaptic terminal is indicated by a thick line. I_{S2} receives an inhibitory input from the afferent axon of an LGN neuron via the inhibitory interneuron I_i and an excitatory input from S. When the LGN neuron is active or both the LGN neuron and S are inactive, I_{S2} is not active and does not decrease the synaptic efficacy. In the case where the LGN neuron is inactive and S is active, I_{S2} decreases the synaptic efficacy. This decrease can be

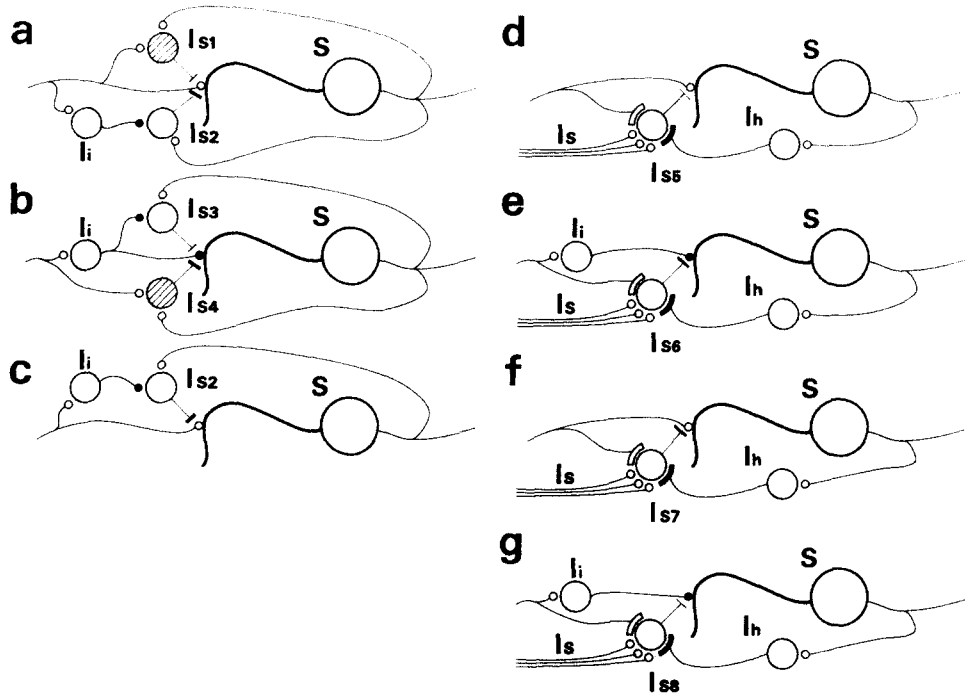

Fig. 12. Examples of neural circuits which modify the synaptic efficacy. The neural circuits for the synaptic modification of the maximally responding cell are shown in (a) to (c). (a) For the modification of the excitatory synapse in the center region of the receptive field. (b) For the modification of the inhibitory synapse both in the center and surrounding region. (c) For the modification of the excitatory synapse in the surrounding region. The hatched interneuron is a high-threshold neuron which becomes active only when two input axons are active simultaneously. The neural circuits for the synaptic modification of the non-maximally responding cell are shown in (d) to (g). (d) For the modification of the excitatory synapse in the center region. (e) For the modification of the inhibitory synapse in the center region. (f) For the modification of the excitatory synapse in the surrounding region. (g) For the modification of the inhibitory synapse in the surrounding region. Interneurons I_{S5}, I_{S6}, I_{S7} and I_{S8} modify the efficacy of these synapses.

induced also by releasing substances which decrease the synaptic efficacy. In Figure 12b, a neural circuit for the modification of the inhibitory synapse in the center and surrounding region is shown. The simple cell S receives inhibitory input from an LGN neuron via the inhibitory interneuron I_i. When both the LGN neuron and S are active, the inhibitory synaptic efficacy is decreased by the high-firing-threshold neuron I_{S4}. The interneuron I_{S3} receives inhibitory input from the LGN neuron and excitatory input from S. When the LGN neuron is inactive and S is active, I_{S3} increases the inhibitory synaptic efficacy. Figure 12c shows the neural circuit for the modification of excitatory synapse in the surrounding region of the receptive field. The interneuron I_{S2} receives inhibitory input from an LGN neuron via I_i and the excitatory synapse from S. In the case where the afferent axon is inactive and S is active, I_{S2} is active and it decreases the synaptic efficacy.

Figure 12d shows a neural circuit for the modification of the excitatory synaptic efficacy in the center region of the receptive field for the non-maximally responding cells. The interneuron I_{S5} receives input I_S from simple cells of different columns. I_{S5} also receives excitatory input from an LGN neuron, and this input acts as a switch to transmit signals on I_S to the output of I_{S5}. Only when the LGN neuron is active, I_{S5}

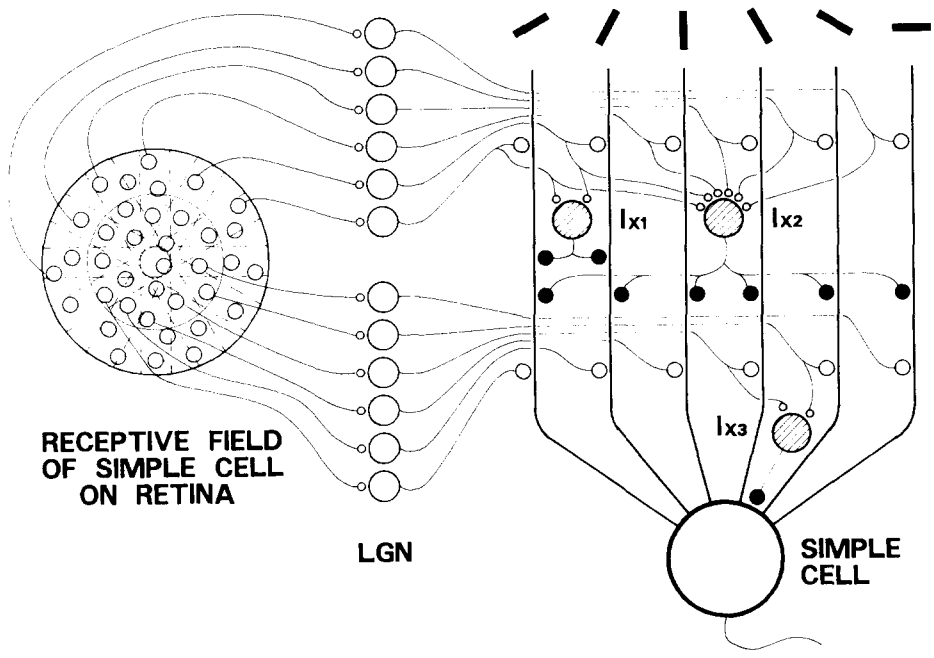

Fig. 13. A putative mapping of the receptive fields in the retina to a simple cell. The mapping is assumed to be topographic, and each dendrite of the simple cell receives LGN input from an elongated field in the retina of one orientation. Interneurons I_{X1}, I_{X2} and I_{X3} act on the simple cell so that it does not respond to more than two orientations simultaneously. The visual afferent axons from the center and the surrounding regions of the receptive field are assumed to produce synapses on a different portion of the dendrite.

transmits signals on I_S and increases the efficacy of the excitatory synapse. However, since I_{S5} also receives strong inhibitory synapse via the inhibitory interneuron I_h from S in the same column, I_{S5} becomes active only when S is not active. Thus, the excitatory synapse increases its efficacy by the signals from the simple cell of different columns only when S is not the maximally responding cell. It has been assumed that the increase of the excitatory synaptic efficacy becomes small as the distance from the maximally responding column increases. This can be explained as follows in this proposed circuits: The number of synapses between I_{S5} and axons of simple cells of different columns decreases as the distance from the maximally responding cell increases. This leads to the decrease in the degree of increase of synaptic efficacy. Figure 12e is a circuit for the decrease in the efficacy of the inhibitory synapse in the center region of the receptive field. The mechanism for the synaptic modification is the same as in Figure 12d, but I_{S6} decreases the inhibitory synaptic efficacy of the visual afferent axon. Figures 12f and 12g are circuits for the decrease and increase of the synaptic efficacy of the excitatory and inhibitory synapses in the surrounding region of the receptive field, respectively. The mechanisms are the same as in Figures 12d and 12e, but I_{S7} decreases the excitatory synaptic efficacy, and I_{S8} increases the inhibitory synaptic efficacy.

Circuitry to form elongated discharge center

In the present model, the neural circuits which give the width of the elongated discharge center of the mature receptive field are not discussed. This can be explained as follows. The topographic mapping of the receptive fields in the retina to the striate cortex is assumed, as noted earlier. If the dendrites of a simple cell extend radially from

its soma, each dendrite receives afferent axons from the elongated field on the retina of specific orientation, organizing the orientation-specific rectangular receptive field of all orientations. This situation is illustrated in Figure 13. However, the dendrites of the simple cell do not extend radially in this figure for the purpose of easier illustration and understanding. The afferent axons from the center and surrounding regions of the receptive field on the retina are assumed to produce synapses on the different portions of the dendrites of a simple cell.

In order for a simple cell to respond to only one orientation at the same time, an additional neural circuit is required. The first example of this circuit is composed of the inhibitory interneuron I_{X1}. I_{X1} receives the excitatory input from two afferent axons of LGN neurons which have adjacent orientation selectivity. I_{X1} is a high-threshold neuron, and it fires and inhibits a simple cell only when both afferent axons of LGN neurons are active simultaneously. Many I_{X1} neurons can exist for each pair of orientation-selective dendrites of a simple cell, organizing the circuits which inhibit a simple cell when stimuli of more than two orientations are presented simultaneously. The second example is shown by the inhibitory interneuron of I_{X2}. There is only one I_{X2} neuron for each simple cell, and its receives excitatory inputs from LGN neurons of all orientations. I_{X2} is also a high-threshold neuron and inhibits the simple cell only when more than two axons of different orientations are active simultaneously. The third

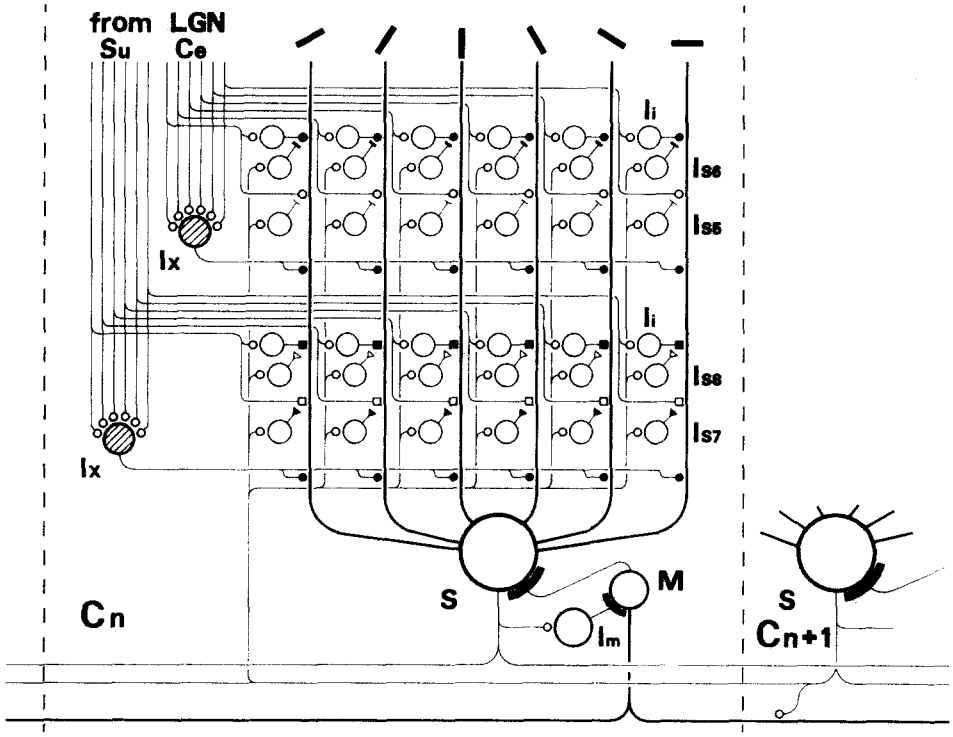

Fig. 14. Summary of the neural circuits which achieve the synaptic modification proposed in this report. These neural circuits summarize those in Figs. 11 through 13. The microcircuits are neglected in this figure for simplicity. The visual afferent axons from the center and the surrounding regions of the receptive field produce synapses in the upper and lower portions of the dendrite of the simple cell. Interneurons I_{S5} and I_{S6} modify the excitatory and inhibitory synapses of the afferent axons from the center region, respectively. Interneurons I_{S7} and I_{S8} modify those synapses of the afferent axons from the surrounding region.

example is the neuron of type I_{X3}. The input to I_{X3} can be type I_{X1} or I_{X2}. The main difference is that I_{X3} produces the inhibitory synapse to the soma of a simple cell. By the mechanisms proposed here, only one orientation of the excitatory inputs is made active at the same time, and the synapses of other orientations are inhibited. If this inhibitory mechanism is blocked, a simple cell will respond to all orientations at the same time.

Figure 14 shows a putative neural network for the self-organization of orientation-selective columns, summarizing Figures 11 through 13. The afferent axons of LGN neurons from the center and surrounding regions of the receptive field produce synapses on the upper and lower portions of the dendrites in this figure. The presynaptic terminals of excitatory and inhibitory synapses from the center region of the receptive field are indicated by open and closed circles, while those from the surrounding region are indicated by open and closed squares. The synaptic modification circuits shown in Figures 12a to 12c as examples are neglected in this figure for simplicity; they are instead indicated by the different symbols for synaptic terminals. The synaptic modification circuits for the center region, when the simple cell in column C_n is not the maximally responding cell, are shown by thin and thick lines of presynaptic terminals of interneurons I_{S5} and I_{S6} for increase and decrease of efficacies, respectively, and those for the surrounding region are shown by open and closed triangles of presynaptic terminals of interneurons I_{S7} and I_{S8} for increase and decrease of efficacies. Only those input axons from column C_{n+1} to these interneurons from simple cells of other columns are shown for simplicity. The excitatory and inhibitory input to these interneurons from visual afferent axons and the simple cell of the same column are also neglected in this figure. These circuits are the simplified drawings of Figures 12d to 12g. The detecting circuit of the maximally responding cell is shown by the interneurons of I_m and M. The interneuron I_X causes the simple cell to fail to respond to more than two orientations at the same time. These neural circuits are one example for achieving the synaptic modification rules proposed in this report. Further experimentation should be carried out to confirm these circuits.

DISCUSSION

In this report, a model for the self-organization of the orientation-selective cells and orientation columns is proposed. The development of the orientation-selective cell is based on the topographic mapping of visual fields in the retina to the striate cortex, circular organization of the receptive field for immature cells, and the different synaptic modification rules for the center and surrounding regions of this receptive field. The self-organization of the orientation columns is based on the synaptic modification by the maximally responding column. The simulation of the present model shows the development of orientation-selective simple cells and orientation columns with a prenatally existing orientation-selective cell. Furthermore, the model shows the abnormal development of orientation selectivity which is seen when the animal is reared in an abnormal visual environment.

It is natural to postulate that the observed organization of the receptive field and the neural connectivity are related to the development of orientation-selective cells and orientation columns. However, this relationship has not been clarified by previously proposed models [2,10,27,28,32,33,42,43]. In the present model, the organization of the receptive field of orientation-selective cells has been related to the development of orientation columns.

It is reported that the response of a simple cell is decreased if the length of the stimulus in the optimum orientation is increased from the one for the maximum response [13]. This is called 'end-inhibition'. In the present model, the receptive field of the surrounding region in its optimum orientation becomes unresponsive to the stimulus. However, if the synaptic modification rule in Table I is so changed for the surrounding inhibitory synapse of the maximally responding cell as not to decrease when both the simple cell and the afferent axon are active, end-inhibition will emerge. This is a possible mechanism for the emergence of end-inhibition.

In the present model, the existence of innately mature orientation-selective cells is crucial for the self-organization of orientation columns. If there is no orientation-selective cell at the initial stage of the simulation, the columns develop into an irregular organization, as shown in Figure 9b. The mechanism for the emergence of the innately mature orientation-selective cells has not been discussed in this report. Recently, it was reported that the orientation-selective cell can develop without any organized stimuli from the retina in the layered neural network [27,28]. This may account for the existence of innately mature orientation-selective cells in the striate cortex. The other possible explanation is that the emergence of the innately mature orientation-selective cells is genetically coded.

Although there is no direct evidence for the plasticity of the inhibitory synapse in the visual cortex, the change in inhibitory synaptic efficacy is assumed in the present model. The orientation columns will emerge in a model which does not assume plasticity of the inhibitory synapse, if instead the selective convergence of visual afferents to a simple cell is assumed. However, such a model fails to explain the development of the receptive field of simple cells. Without the assumption of plasticity of the inhibitory synapse, it is difficult to conceive a model which accounts for both the development of the receptive field and the self-organization of orientation columns. Therefore, the author proposes this assumption as a working hypothesis which will be tested experimentally in the future.

The nervous system is so complicated that the elucidation of their mechanisms only through experiment is quite difficult. This situation is also true for the striate cortex. The synthetic approach of modelling is thought to be very useful for such a complicated system. The model proposed here is created so as to include as many experimental results as possible and can explain many observations, thus clarifying the relationship between the organization of a receptive field and the formation of orientation columns starting from some simple assumptions.

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