AN ASSOCIATIVE LEARNING MODEL OF THE McCOLLOUGH EFFECT[1]

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Abstract

A computer simulation of a neural network model involving Hebbian synaptic modification between a retinal layer and a cortical layer is proposed as a possible explanation of the McCollough effect, without assumptions about fatigue, inhibitory rebound, or a neutralizing response. Synaptic modification of initially randomly weighted synapses during random presentation of all color-orientation combinations produces a uniform distribution of color-orientation specific units. With additional presentations of alternate red-vertical (R-V) and green-horizontal (G-H) stimuli during synaptic modification, the network responds to black-and-white-horizontal (W-H) and black-and-white-vertical (W-V) stimuli with the McCollough effect. This is because the shift in response of the R-V units to the R-V stimulus causes the depletion of the slightly red-sensitive cells from the W-V population response. Likewise the shift in response of the G-H units to the G-H stimulus produces a depletion of the slightly green-sensitive cells from the W-H population response. The result is a slightly green bias in the W-V response and a slightly red bias in the W-H response.

Synaptic modification affords a more plausible explanation of this effect than fatigue or inhibitory rebound, given its very long durability and its lack of decay during sleep.

Introduction

The McCollough effect [39] is produced by viewing vertical red and black stripes and horizontal green and black stripes alternating every 5 or 10 seconds for about 5 minutes or more. After these adaptation presentations a vertical black and white test grating will appear slightly green while a horizontal black and white test grating will appear pink. The effect can also be induced by red and green vertical gratings of two different spatial frequencies [43], and by horizontal gratings of complementary colors moving in opposite directions [28]. The effect depends on retinal spatial frequency [46] rather than on viewing distance, that may be corrected for by size constancy [22], or on the size of black and white bars [23]. The effect does not depend on retinal fixation necessary for afterimages [26], but only on general retinal location [44]. It is also unlike afterimages in its long duration, sometimes on the order of hours or days depending on the length of adaptation [28, 34], and its lack of decay during sleep [36].

McCollough [39] in 1965 and others [38] have reported that the effect does not transfer interocularly, in fact, opposite effects can be built up in the two eyes [39]. But MacKay and MacKay obtained a binocularly induced effect in 1973 [35]. One eye viewed uniform red and green fields alternately while the other eye viewed black and white gratings tilted 45° left and 45° right of vertical alternately. The red field correlated with one tilt and the green with the other tilt. The normal McCollough effect was induced in the eye that had viewed color while an opposite McCollough effect--the test grating appearing the same color as the adaptation grating--

was induced in the eye that had viewed achromatic gratings. This lack of interocular transfer of the normal effect has lead some researchers to conclude that the McCollough effect occurs in the retina or lateral geniculate [34, 39]. However, the conclusion that the effect occurs in monocularly biased cells in cortex is not incompatible with these results. Both spatial frequency and orientation adaptation have been found to transfer interocularly [2, 14, 38]. And since the McCollough effect is specifically tied to the spatial frequency and the orientation of gratings, the possibility that it occurs cortically cannot be entirely dismissed. In addition, the evidence that it is very long lasting and associated with multiple features points to a more central than peripheral effect.

Another open question concerning the McCollough effect is what possible physiological basis can exist for such a long term effect. Since it is clear that inhibitory rebound or fatigue is not long lasting enough to explain these effects [24, 34, 49], the physiological basis must be some sort of structural modification of the network. Synaptic modification is a good candidate for a long term effect.

Another argument against fatigue is that the effect is always a weak one no matter for how long adaptation occurs. Prolonged exposure merely prolongs the aftereffect rather than increasing its strength. Fatigue as in afterimages would be expected to increase to a maximum and then decay in the same amount of time from that maximum independent of exposure time [49].

Evidence of an Adaptive Metric

An organism's ability to detect features in the visual field is dependent on its exposure to such features in early life [3, 13, 29, 30, 41]. However, the distribution of feature detectors induced by early exposure to a set of stimuli remains plastic even in adult life [7]. Prolonged exposure to a point along some perceptual dimension, such as spatial frequency, warps the perception of neighboring points within a region of about $\pm$ one octave [4, 5, 6, 45]. I believe this to be evidence of a dynamic visual metric, that is the ability of an organism to measure continuous features of the visual input changes with time. Such a metric would have obvious adaptive (in the evolutionary sense) value by more precisely specifying familiar features and suppressing irrelevant features, such as the suppression of colored fringes produced by prism glasses worn by subjects in Kohler's studies [31].

There are at least two kinds of effects here: one positive and one negative. The negative aftereffect is the one most commonly reported in psychophysical studies. All the usual masking and adaptation effects fall into this category. A negative aftereffect lowers the activity or raises the threshold of the adaptation stimulus or raises the activity or lowers the threshold of the complementary stimulus. A positive after-effect raises the activity or lowers the threshold of the adaptation stimulus. DeValois [8] calls positive aftereffects "similitude" effects as opposed to contrast effects. Tuning of receptive fields to line orientations by early experience would be an example of positive effects. There are fewer instances of positive effects in adults reported in the literature but

those instances that have been studied appear to be afterimages originating mainly in the retina [1, 10, 15], although not entirely in the retina [12]. Positive effects have a longer time course and may appear after negative effects [8].

Both effects can be explained by positive synaptic modification. Wilson [49] devised a mathematical model of spatial frequency adaptation in which connection strengths from inhibitors to excitors were increased with the correlation in firing of the presynaptic and postsynaptic cells. The net effect was an increase in the inhibition of excitors sensitive to the adaptation spatial frequency. Positive effects are demonstrated by positive synaptic changes from input cells to excitors. von der Malsburg's neural network model of cortex [48] simulates the organization of cortical cells with initially randomly specified receptive fields into a distribution of cells sensitive to particular line orientations. Receptive fields become tuned to the set of inputs by Hebbian [27] synaptic modification of connections between a retinal layer and a cortical layer after each stimulus presentation. Supposedly, this is the process involved in receptive field organization of young organisms as a result of visual experience. But is this process useful to the adult animal besides? Hebb [27] and Grossberg [19, 20] maintain that this process is the primary mechanism for learning. Grossberg has proven this mechanism capable of learning a wide variety of stimuli as well as responses. The training set of one of these networks induces an equal distribution of cells sensitive to the inputs. If there are more vertical lines in the set, for instance, there will be more cells tuned to vertical lines. The specificity of cells also changes with training making them more narrowly tuned. More units will be tuned to more frequent stimuli, thus allowing

the possibility of finer tuning to more frequent stimuli.

The McCollough-Malsburg Model or M^3

von der Malsburg's Model

von der Malsburg's model consisted of 19 retinal cells each connected to all 169 excitatory cortical cells. Each excitatory cortical cell connected to its 6 nearest neighbors in the hexagonal array, to the corresponding 6 cells in the 169-cell inhibitory layer (I), and to the I cell corresponding to itself. Each I cell in turn connected to a 12-cell surround in the excitatory layer (E). (See Figure 1.) Each cell computes a weighted sum of its inputs from all layers, subtracts a threshold, and

[Insert Figure 1 about here]

outputs the result if it is positive and zero otherwise. The postsynaptic potential of each cell at each step is calculated by

$$E_k = aE_k + \sum_{i=1}^{19} s_{ik}R_i^* + \sum_{i=1}^{6} C_{EE}E_i^* - \sum_{i=1}^{12} C_{IE}I_i^* \qquad (1)$$

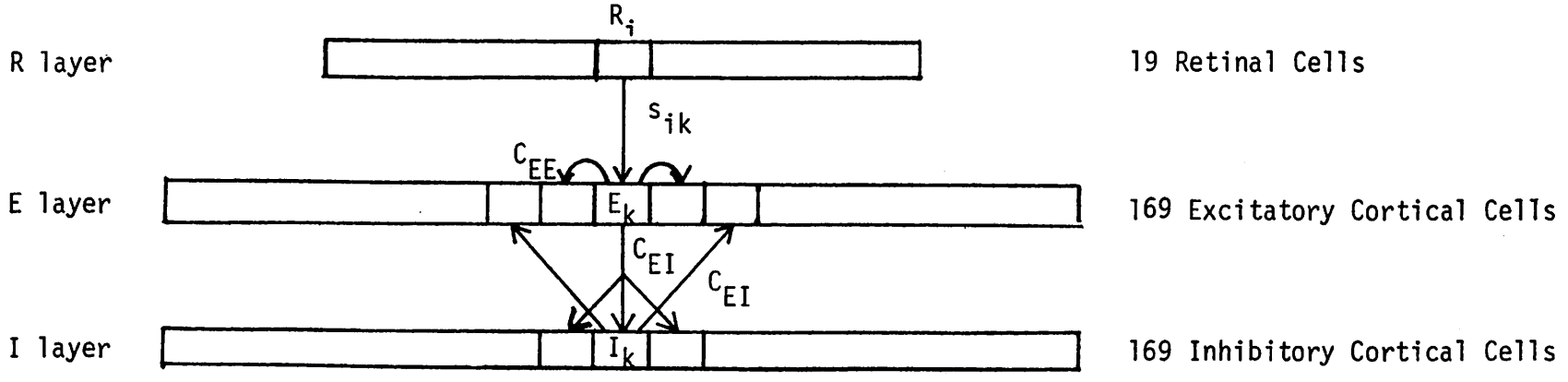
for excitors and

$$I_k = bI_k + \sum_{i=1}^{7} C_{EI}E_i^* + C_I \qquad (2)$$

for inhibitors, where a and b are decay constants for each iteration, s_{ik} is the synaptic weight from retinal cell R_i to cell E_k, C_I is a constant input to I and C_{AB} is the synaptic weight constant from a cell in layer A to one in layer B. A synapse s_{ik} is modified when R_i and E_k fire simultaneously according to the rule:

$$s'_{ik} = s_{ik} + \Delta \cdot R_i^* \cdot E_k^* \qquad (3)$$

Figure 1: Side view of hexagonal retina and cortical arrays.


R layer
R_i
19 Retinal Cells
s_{ik}
C_{EE}
E layer
E_k
169 Excitatory Cortical Cells
C_{EI}
C_{EI}
I layer
I_k
169 Inhibitory Cortical Cells

where s'_{ik} is the new value, s_{ik} is the old, Δ is the learning constant, and $*$ is the thresholding function:

$$x^* = \begin{cases} x - \theta & \text{if } x > \theta \\ 0 & \text{if } x \leq \theta \end{cases} \tag{4}$$

θ is the threshold. The sum of each cortical cell's input synapses is held constant at C_s by renormalization after the modification step:

$$s_{ik} = s'_{ik} \frac{C_s}{\sum_{i=1}^{19} s'_{ik}} . \tag{5}$$

The input set consists of 9 lines at different orientations, each line stimulating 7 retinal units. (See [48] for further details.)

M^3 Version I

M^3 is a color version of von der Malsburg's self-organizing model of cortex. The major change is that M^3's retina sees red and green in addition to black and white. Instead of having achromatic on-cells in the retina M^3 has a red-on cell and a green-on cell for every achromatic retinal cell in the von der Malsburg model. Thus, there are a total of 38 retinal cells each connected to all the 169 excitatory cortical cells. The spatial arrangement of the retina is identical to von der Malsburg's except that each retinal point contains a red and green cell in the same retinal location.[2]

The stimulus set for M^3 varies not just in orientation but in the amount of red/green saturation. The variable v represents a point on a

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[2] The word "retina" is used here merely to correspond with von der Malsburg's first layer. The red and green cells in M^3 are much more complex than red or green cone cells. They are probably more on the order of ganglion, lateral geniculate, or possibly cortical cells.

saturation continuum from pure green through white to pure red of equal luminance. Some care was necessary in choosing a function of v to represent color that would allow E cells to be tuned to a continuous range of v rather than just two values: red (v = 1) and green (v = 0). The need for this will become clear in the discussion section.

Let w_1 and w_2 be the synaptic weights associated with the green and red retinal cells, respectively. The first function tried is the most obvious:

$$f_0(v) = w_1(1 - v) + w_2(v). \tag{6}$$

Since f_0 is a linear function of v there can be only two possible maxima over $v \in [0,1]$ for fixed synaptic pairs (w_1,w_2). These maxima are at $v = 0$ or at $v = 1$. Using f_0 as input an E cell can be tuned only to pure red or pure green.

A second function tried,

$$f_1(v) = w_1[1 - kv^2]^* + w_2[1 - k(v - 1)^2]^* \tag{7}$$

with $\theta = 0$, is based on the general shape of red and green opponent-color cells in LGN over wavelength, disregarding the inhibitory component [9]. The trouble with this formulation of the color input is that it does not allow tuning to a specific combination of red and green that reflects v. To illustrate this point let's suppose that E cells have only two inputs with weights w_1 and w_2. Also suppose that $w_1 + w_2 = C$ for all cells and that v is fixed at some $v_0 \in (0,.5)$. We wish to train the best responders to v_0 such that they fire maximally to v_0. The best responders to v_0 are those with $w_1 = C$ and $w_2 = 0$. But these cells fire even more strongly to $v = 0$. So differential training to v_0 cannot occur, only to $v = 0$ or $v = 1$.

The third function tried,

$$f_2(v) = (w_1 + w_2)[1 - k(v - \frac{w_2}{w_1+w_2})^2]^* \qquad v \in [0,1] \qquad (8)$$

with $\theta = 0$, has the same general shape as opponent-color cells' response in LGN but also has the additional property that for a given v_0 the maximum responders at a constant $w_1 + w_2$ are those with $w_2/(w_1 + w_2) = v_0$, a specific synaptic pair ratio reflecting v_0. This is the necessary property for specific tuning to v_0. k is a tuning-width parameter. (See Figure 2).

A second feature of this network that was different from von der Malsburg's is an increase in the amount of inhibition by 5 times so that fewer cells would go on for each stimulus. This was necessary so that cortical cells could differentiate more stimuli. In addition, the excitatory and inhibitory thresholds in this model vary according to the average retinal input to E cells. (See Table 1).

[Insert Table 1]

In the original model the average input to cortical cells was approximately constant over all stimuli, with only slight variations due to randomly assigned weights. In M^3 synaptic weights are assigned from a uniform distribtuion over the interval [0,.1]. Thus, synaptic pair maximums, $w_2/(w_1 + w_2)$, over v are derived from a uniform distribution over [0,1]. However, the maximum over v of a cortical cell summing over all its synaptic pairs is derived from a distribtuion over [0,1] that approaches a Gaussian with mean at $v = .5$. Therefore, the level of firing of the naive network is greatest at $v = .5$ and very low at $v = 0$ or 1.

Figure 2: Red/green synaptic pair response to saturation, v.

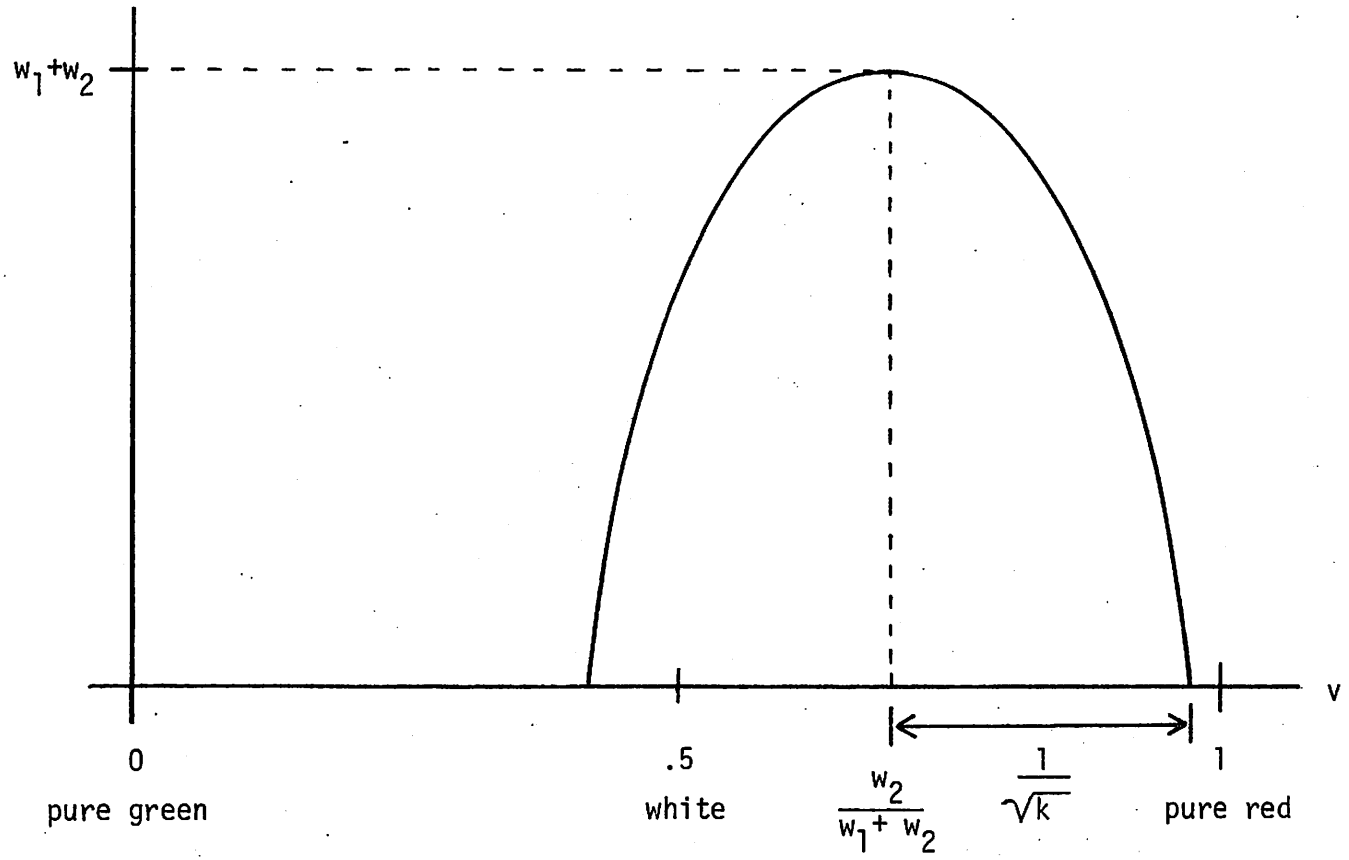

w_1+w_2
v
0
pure green
.5
white
$\frac{w_2}{w_1+w_2}$
$\frac{1}{\sqrt{k}}$
1
pure red

Table 1

Parameters for M^3 versions I and II.

	VERSION I	VERSION II	
a	.6	.6	E-cell decay constant
b	.3	.3	I-cell decay constant
C_{EE}	.16	.16	E to E-cell weight
C_{EI}	1.0	1.0	E to I-cell weight
C_{IE}	.6	.6	I to E-cell weight
C_I	$\overline{E}$	.4	input to all I-cells
C_s	$38\overline{s}$	$38\overline{s}$	sum of synaptic weights
$\overline{s}$	.05	.05	average synaptic weight
Δ	.05	.05	synaptic increment constant
θ_E	$\overline{E}/.4 + .25$	1.125	E threshold
θ_I	$\overline{E}/.7 + .01$	.58	I threshold
k	4.	7.1	color curve width constant
$\overline{E}$	$\frac{1}{169}\sum_{k=1}^{169}\sum_{i=1}^{38} S_{ik}R_i$	$\frac{1}{169}\sum_{k=1}^{169}\sum_{i=1}^{19} S_{ik}R_i$	average input to E-cell for a given stimulus

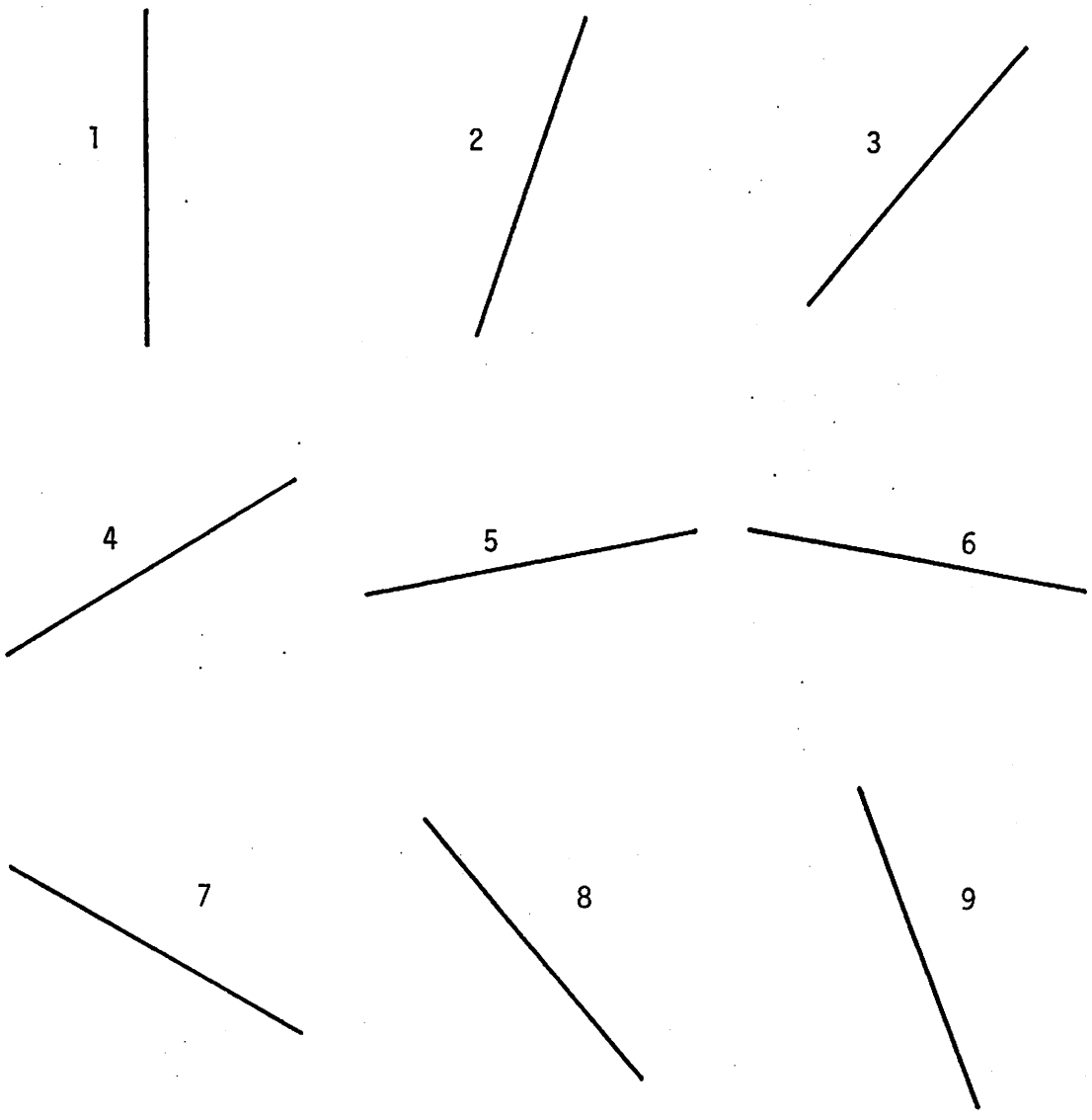
But in order to insure uniform training to all stimuli the total firing of the network must be about constant over stimuli, otherwise the resulting distribution of units after training will not equal the input distribution. So in order to keep the level of firing and the number of cells on approximately constant, a variable threshold was used for E and I layers in addition to a variable input to I. The variable threshold approximates a normalized input. Stanley [42] has shown by computer simulation that a constant output in a network can be achieved also by recurrent inhibition, but I used a variable threshold to achieve the same effect because recurrent inhibition increased the relaxation time of the network so much that computation time became prohibitive.

Procedure. Initially all synaptic weights from layers R to E -- 38×169 of them--were randomly assigned from a uniform distribtuion over [0,.1]. Synaptic modification occurred after each presentation of a randomized set of 9 line orientations by 11 color saturation values at equal intervals between [0,1]. All 99 stimuli were presented twice. After each presentation the network was allowed to run to approximate equalibrium, 20 iterations, and the synapses of each E cell that was on at this point was modified by applying (3) and (5).

After training to a uniform distribution over orientation and saturation the McCollough stimulus pair was presented alternately: line #1 with $v = 0$ followed by line #5 with $v = 1$. (See Figure 3).

[Insert Figure 3]

Figure 3: Line orientations presented to the network.


1
2
3
4
5
6
7
8
9

The synapses were modified in the same fashion after each stimulus presentation. The pair of stimuli were presented alternately 20 times.

The same McCollough experiment was tried on the uniformly trained network with each orthogonal line pair possible: (1,5), (2,6), (3,7), (4,8), (5,9) paired with saturation combinations (0,1) and (1,0): ten experiments in all.

The response of the network was calculated as follows. For every cell on after 20 iterations its maximum orientation-saturation stimulus was calculated. Then the average orientation and saturation was calculated over all responding cells weighted by their degree of firing. The response was always close to but not exactly equal to the stimulus value.

Results. Table 2 shows the results of ten McCollough experiments. The response of the network to $v = .5$, a white line of orientation indicated, is shown before and after McCollough presentations. The difference column shows the start minus the final response. A negative increment indicates a change toward red while a positive increment indicates a change toward green. The results show that the response to a white line of a given orientation that was paired with green during adaptation changes toward the red end of the continuum, while the response to an orthogonal white line paired with red changes toward the green end. The means of the differences, $\bar{d}_0$ and $\bar{d}_1$, the standard deviations of the differences, δ_0 and δ_1, the standard error of the mean, s_Δ, degrees of freedom, df, t value, and level of significance are also shown. The change in response for the two groups of cells differentially adapted is significantly different ($t = 9.909$, $p < .0005$).

[Insert Table 2]

Table 2

The response of M^3 version I to white test stimuli (v = .5) before and after alternate hue-orientation presentations. Each row represents one McCollough experiment.

ADAPTATION	v = 0				v = 1		
TEST	v = .5				v = .5		
LINE	START	FINAL	d_0	LINE	START	FINAL	d_1
1	.485	.635	-.150	5	.535	.498	.037
2	.457	.582	-.125	6	.526	.430	.096
3	.569	.608	-.039	7	.445	.395	.050
4	.505	.540	-.035	8	.467	.334	.133
5	.535	.626	-.091	9	.552	.291	.261
[5	.535	.626	-.091]*	1	.485	.405	.080
6	.526	.694	-.168	2	.457	.418	.039
7	.445	.623	-.178	3	.569	.413	.156
8	.467	.603	-.136	4	.505	.472	.033
9	.552	.662	-.110	[5	.535	.498	.037]*

$\bar{d}_0 = -.115$ $\bar{d}_1 = .098$

$\delta_0 = .052$ $\delta_1 .075$

$S_\Delta = .0125$ $t = 9.909$ $df = 18$ $p \ll .0005$

* duplicate results are left out of the computation.

Discussion. How does a positive modification in response to a stimulus lead to negative aftereffects? If you start out with unit responses evenly distributed over orientation and saturation (see Figure 4) training to a red-vertical (R-V) stimulus causes a shift in the R-V population response toward R-V which depletes the slightly red-sensitive cells from the black-and-white vertical (W-V) population response. (See Figure 5). Likewise a shift in the response of green-horizontal (G-H) units toward G-H depletes

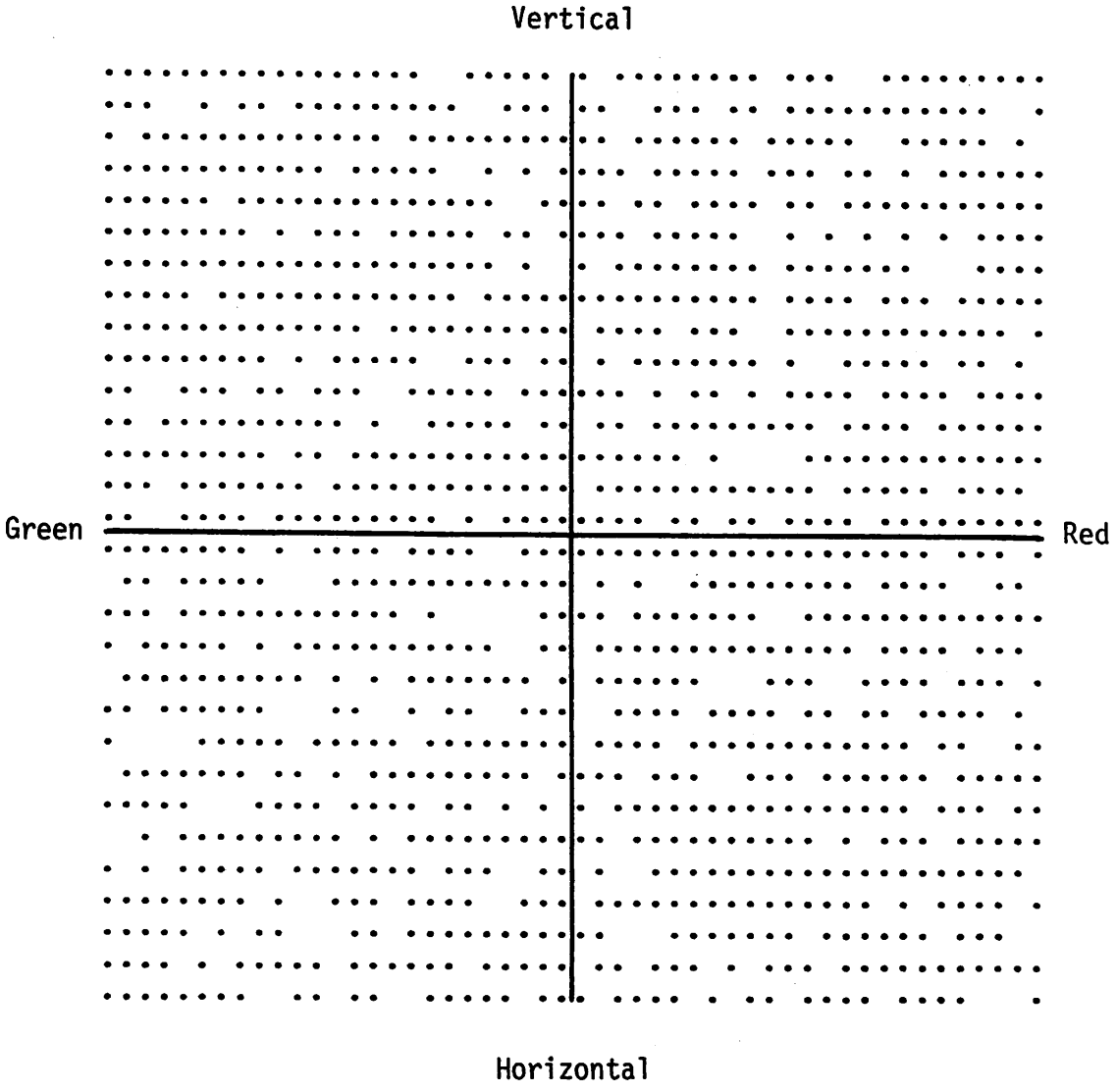
[Insert Figures 4 and 5 here]

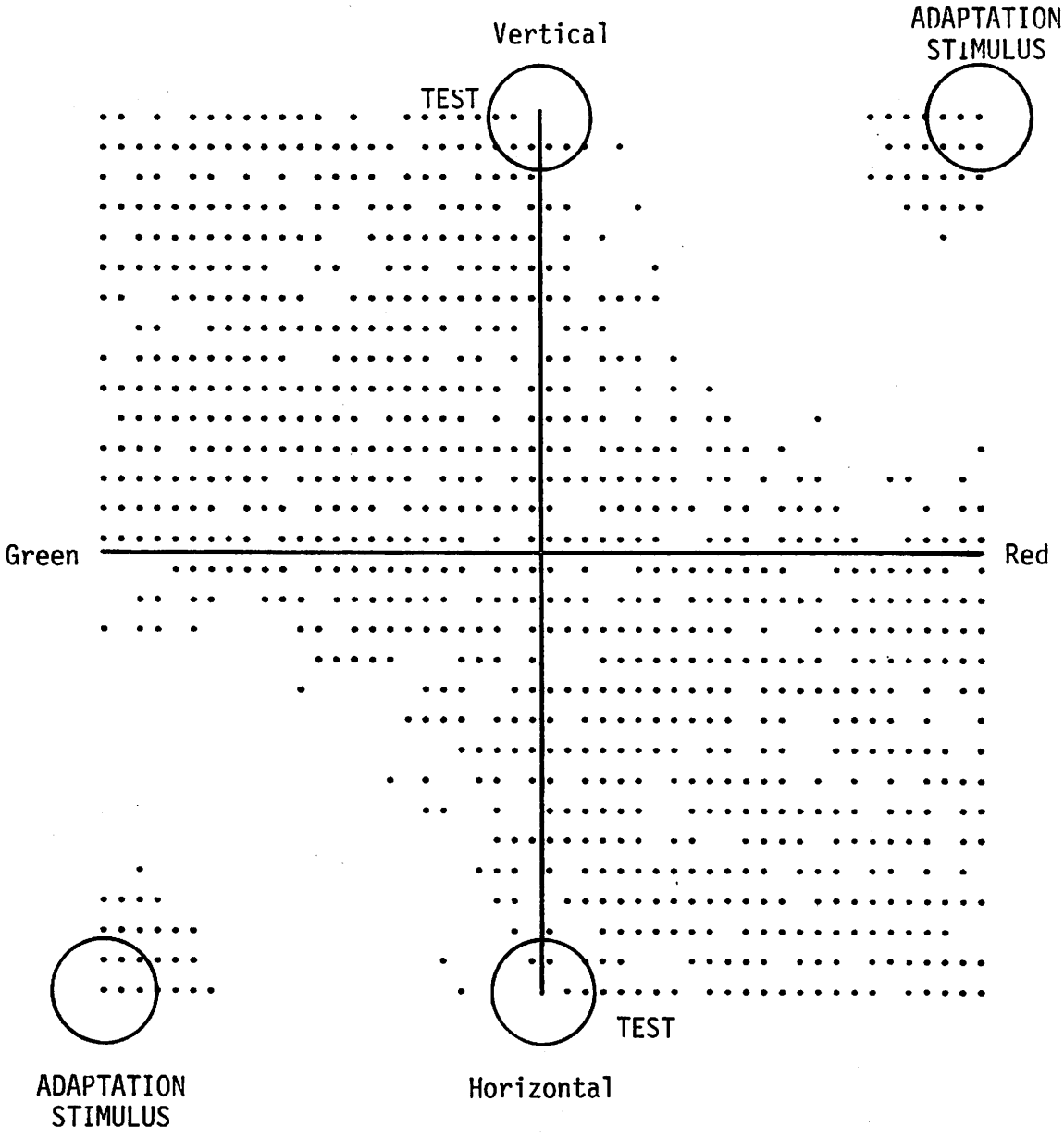
the slightly green-sensitive cells from the black-and-white-horizontal (W-H) population response. The result is a slightly green biased response to W-V and a slightly red biased response to W-H. This is a possible explanation of why the McCollough effect is always so desaturated. The tuning width of retinal cells to color is crucial to the direction of the effect, i.e., whether adaptation will result in a positive or negative aftereffect at the neutral point. If the tuning width is too broad, $k = 1$, the aftereffect at $v = .5$ will be positive. If the width is too narrow, $k = 16$, there will be no change at $v = .5$.

One problem with version I is that there is no physiological basis for a synaptic red/green pair that behaves as in (6), and no way of arranging the circuit so that synapses will be modified according to (3). The synaptic connection from R to E is strictly a mathematically convenient one. A more plausible assumption is that there exists somewhere in the retina, LGN, or visual cortex units maximally sensitive to specific values of saturation of red or green. These elements could be broad-band cells with variations in the amounts of red or green bias, or they could be

Figure 4: Conceptual illustration of a uniform distrubution of unit responses over orientation and color.

Figure 5: Conceptual illustration of the distribution of unit responses after presentation of complementary orientation-hue stimuli.


Vertical
Green
Red
Horizontal


Vertical
TEST
ADAPTATION
STIMULUS
Green
Red
TEST
ADAPTATION
STIMULUS
Horizontal

opponent-color cells with maximum sensitivity anywhere on the color spectrum [9, 16, 17, 18]. If we choose these to be opponent-color cells, the white component of the saturation sensitivity spectrum must be made up of equal proportions of blue and yellow sensitive cells. White light input is correspondingly made up of equal components of blue and yellow.

M^3 Version II

In this version there are 19 retinal positions each making 169 connections to the E layer -- 169×19 in all. Each connection is randomly assigned a peak saturation-sensitivity, $m_{ik} \in [0,1]$, and weight, $s_{ik} \in [0,.1]$. m_{ik} is never modified as in version I. It is assumed that interneurons between R_i and E_k determine m_{ik} for each connection in the following fashion. v is the red saturation response and $1 - v$ is the green saturation response of units at R_i in the retina. These are converted to sigmoidal [50] responses dependent on m_{ik} and k by interneurons (Figures 6a and 6b). The outputs of this pair is multiplied before reaching E_k (Figure 6c). A quadratic function is used to simulate the multiplied pair of sigmoids:

$$f_3(v) = s_{ik}[1 - k(v - m_{ik})^2]^*. \qquad (9)$$

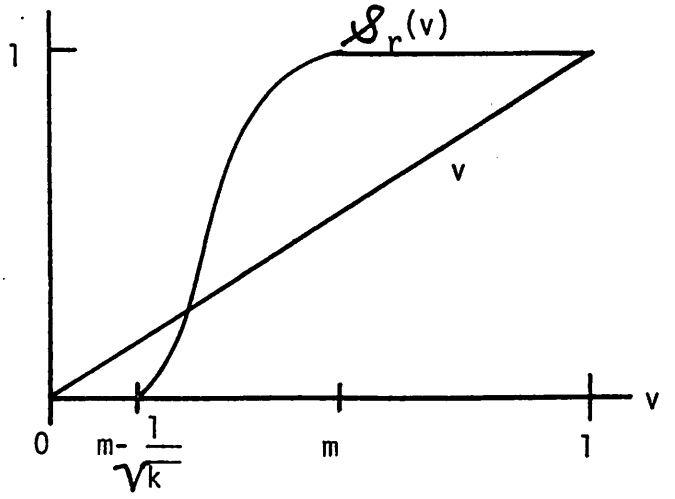
The thresholds in the E and I layers and the input to I is held constant. (See Table 1). The tuning width of (9) is narrower, $k = 7.1$.

Initially the network was trained to a uniform distribution over orientation and saturation by presenting eight random sequences of the same 99 input stimuli used in version I. All other procedures and parameters were the same as in version I.

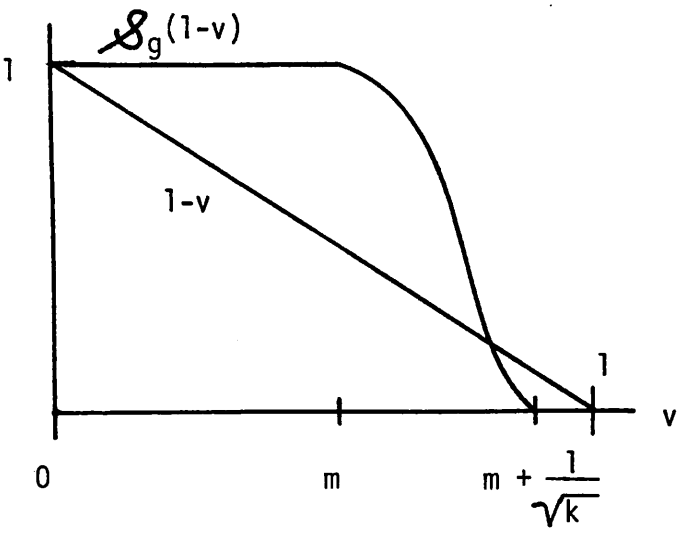
Results. The results of ten McCollough experiments on the uniformly trained

Figure 6: A possible organization of red/green interaction producing maximum firing to a specific combination of red and green. a: sigmoidal response of red interneuron; b: sigmoidal response of green interneuron; c: circuit transforming v into $s(1 - k(v - m)^2)$.

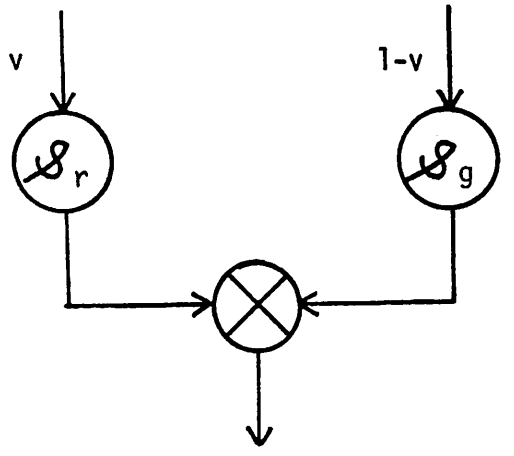
a.


1
$\mathcal{S}_r(v)$
v
v
0
$m-\frac{1}{\sqrt{k}}$
m
1

b.


$\mathcal{S}_g(1-v)$
1
1-v
1
v
0
m
$m+\frac{1}{\sqrt{k}}$

c.


v
1-v
$\mathcal{S}_r$
$\mathcal{S}_g$

network, M^3 version II, are summarized in Table 3. Again the line orientations paired with red adaptation stimuli exhibit a green bias to white test stimuli--a positive increment--while orientations paired with green exhibit a red bias to white. The difference between the two groups is significant at the .005 level.

[Insert Table 3]

Discussion. The second version of M^3 reproduced the McCollough effect without having to assume that synaptic inputs shift their spectral sensitivity with training. Of course, cortical cells still shift their sensitivity by selective training of synapses from cells with different spectral sensitivities. However, more training is required, 8 presentation sequences, and a narrower spectral specificity of retinal cells, $k = 7.1$.

Issues

Specificity

The only assumption made about the specificy of cells in this model is that R cells are location specific and saturation or spectrally specific. The model does not assume the existence of hue-orientation specific units. These fall out of the training procedure, not as static single-unit detectors but as flexible, changeable population responses. There is, however, some evidence of line orientation and color specific cells in cortex [11, 18], but color and form specificy were found to vary inversely. If a cell was narrowly tuned to color, it was very broadly tuned to form, and vice versa. The same is true of the present model. By varying the parameter, k,

Table 3

The response of M^3 version II to white test stimuli (v = .5) before and after alternate hue-orientation presentations.

ADAPTATION	v = 0				v = 1		
TEST	v = .5				v = .5		
LINE	START	FINAL	d_0	LINE	START	FINAL	d_1
1	.507	.507	0	5	.435	.436	-.001
2	.474	.474	0	6	.443	.427	.016
3	.476	.484	-.008	7	.490	.487	.003
4	.443	.444	-.001	8	.460	.450	.010
5	.435	.442	-.007	9	.520	.466	.054
[5	.435	.442	-.007]*	1	.507	.472	.035
6	.443	.443	0	2	.474	.473	.001
7	.490	.522	-.032	3	.476	.469	.007
8	.460	.455	.005	4	.443	.448	-.008
9	.520	.520	0	[5	.435	.436	-.001]*

$\bar{d}_0 = -.005$ $\bar{d}_1 = .013$

$\delta_0 = .011$ $\delta_1 = .019$

$S_\Delta = .00517$ $t = 3.463$ $df = 16$ $p < .005$

* duplicate results are left out of the computation.

related to the spectral tuning width of R cells, I could get units narrowly tuned to color but very broadly tuned to lines, or units narrowly tuned to lines but broadly tuned to color. As Harris [24] suggests, the amount of specificity a single cell, or a small group of cells, for that matter, can handle is probably limited. As form becomes more and more abstract, curved lines or complex figures, the spectral specificity widens. The present model produced positive aftereffects with the McCollough paradigm if too broad a spectral tuning curve was used. A psychophysical study done by Viola [47] produced a positive McCollough effect in some subjects with curved lines. It is more likely, however, that units sensitive to complex figures have no spectral specificity whatsoever [11, 18], and cannot duplicate the McCollough effect.

Positive vs. Negative Aftereffects

Most adaptation studies report negative aftereffects. M^3 as it stands predicts a positive aftereffect in the neighborhood of the adaptation stimulus. There are several possible natural extensions of the model that could produce negative aftereffects. Note that a negative aftereffect in the region of the adaptation stimulus would not change the prediction of a negative aftereffect at the neutral test point.

One possible extension is adding recurrent inhibition of a form that will not disrupt the relaxation time of the network. Direct inhibition from an E cell's corresponding I cell could drive a vigorously stimulated cell to lower levels than a weakly stimulated cell. In addition, depending on the spatial properties of the inhibition more strongly tuned cells, with higher peaks but narrower bandwidths, would be less likely to fire to

extraneous stimuli and thus reduce the average firing of the network. So even though the connection strengths of particular populations of cells increased, the overall output of the network may decrease, depending on the characteristics of the inhibition. The payoff in training would be in the increased decisionary power and precision of the modified network. See Montalvo for a discussion on decisionary networks [40].

Another way of driving up the inhibition is to strengthen connections from inhibitory to excitatory cells with correlated firing as Wilson [49] did in his study of spatial frequency adaptation.

These are all questions for further study.

Spatial Frequency vs. Line Detectors

M^3 and von der Malsburg's model, from which M^3 was derived, are designed to simulate a small patch of cortex. They make no assumptions about how bar detectors interact to produce populations of cells sensitive to spatial frequency in wider areas of retina and corresponding cortex. Since the McCollough effect is specifically dependent on spatial frequency I should mention how M^3 would fit into a larger theory embodying sensitivity to spatial frequency.

We could suppose simply that a larger network can be trained to periodic stimuli. Lenherr [33] did just that. He trained a similar network to three-bar stimuli of different orientations, not just single bars. But the trouble with specific training to periodic stimuli is that 3-bar receptive fields are found only in the rarest of circumstances [30, 41]: through visual deprivation.

A second option is to suppose that populations of various size bar and edge detectors with on and off-cell receptive fields are organized in

subsequent layers maximally sensitive to spatial frequency rather than bar width [23]. Such units could be texture elements used for segmentation rather than anything as specialized as spatial frequency detectors. There is some evidence that texture-orientation features of some form play an important role in segmentation of regions [21, 37, 51]. In this case M^3 is merely modelling a small area of the first layer of processing, that which identifies orientation and color.

Line Detectors vs. Dipoles

M^3 makes no assumptions about the degree of specificity of line detectors. They may be as amorphous as receptive fields in the untrained network having randomly assigned weights or they may be as exact as bar templates. Both the untrained and the over-trained network exhibit the McCollough effect after alternate complementary form-color presentations. All that is really necessary is units more responsive to one orientation than to others. This is the case in the untrained network with random input connections otherwise units could not be trained initially to a preferred orientation. Whether we call these units line detectors or dipoles [49] is irrelevant. These effects can occur over a wide range of orientation specificity.

Conclusion

An associative learning model for the McCollough effect in the form of synaptic modification between two layers of neurons was tested by computer simulation. The results are consistent with the experimental evidence.

Positive synaptic modification associated with the adaptation stimulus draws units away from the black and white population response to the same line orientation, such that the net effect to an achromatic line is bias toward the complementary color. No assumptions about fatigue, inhibitory rebound, or a neutralizing response [31, 32] were necessary.

Although the model as it stands cannot explain experimental reults that reduce the sensitivity of units to the adaptation stimulus or that cause neighboring stimuli to appear less like the adaptation stimulus [5, 6] it is believed that an extension of the model that includes modification of inhibitory synapses, recurrent inhibition, or stronger nonlinear inhibition can go a long way in explaining these results. The proper balance of positive and negative effects must be struck to encompass both the tuning of feature detectors to familiar stimuli and a reduction of interference from competing units.

In the meantime the model provides a good example of an adaptive metric along two perceptual dimensions: color and orientation. This choice of dimensions is incidental to a more general way of looking at "feature detectors." The type of feature detectors modelled here are not rigid templates. They are not represented by stationary single points along a perceptual continuum. They are not single cells, but rather clumps of cells that are responsive to a range of inputs. They can change both the length and position of that range along a perceptual dimension depending on the current set of inputs. Their degree of specificity can be adjusted. They don't divide the input into separate channels but signal their responses to different inputs together as groups in different patterns of firing. They give us a better handle on visual feature space.

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