

A Multi-Stage Color Model

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The first stage of our model has three cone types, with L:M:S cones in ratios of 10:5:1. In the second stage, retinal connectivity leads to three pairs of cone-opponent, and one pair of cone-nonopponent systems. At a third (cortical) stage of color processing, the S-opponent cells are added to or subtracted from the L- and M-opponent units to split and rotate the one effective parvo geniculate response axis into separate RG and YB color axes, and separate luminance from color. We also discuss changes with eccentricity, and connectivity based on correlated neural activity.

Color model Opponent colors Unique hues

INTRODUCTION

The first "models" of color vision, or of most other aspects of vision, were essentially simple one-stage models. There are at least three good reasons why this was so. First, there are valid scientific justifications for developing as simple theories as are possible. Secondly, the many complexities of vision with which we are now acquainted were not then well understood. Thirdly, most problems were considered individually, in isolation from other visual processes.

If almost any specific problem in vision is examined in isolation, it is often possible to find a one-stage solution to it, and this is what happened in the case of early theoretical formulations of various aspects of vision. The visual system, however, does not have the luxury of solving just one specific problem at a time. Rather, it must work towards the solution of many visual problems simultaneously, especially in the early stages of processing. At the very least, the solution of one problem must not entail steps which prevent the later solution of other problems. Furthermore, the visual system has many sharp constraints in terms of the types of algorithms that can be implemented, the distances over which interactions can take place, and so forth. Finally, there is the special constraint in the early stages of the visual system that all the information must be passed through the bottleneck of the optic nerve (De Valois & De Valois, 1988).

Both of the classic theories of color vision in the last century, those of Helmholtz (1867) and of Hering (1878), were, as first formulated, essentially one-stage models which (we can now see) accounted for only small and largely nonoverlapping fractions of the facts. That these two theories for so long coexisted in opposition to each other was primarily due to the fact that their adherents argued from different sets of findings, from color-mixing experiments on the one hand and perceptual obser-

vations on the other. Although there were several proposals of two-stage models that joined these supposedly-opposing ideas (e.g. Donders, 1881; von Kries, 1905), they were not taken very seriously, primarily because the field was long dominated by those whose primary interest lay in questions that we now know are primarily determined at the receptor level. They saw no need to consider the possibility of something as vague and seemingly unlikely as opponent interactions.

It was not until Jameson and Hurvich (1955, 1956) and Hurvich and Jameson (1955, 1956) quantitatively formulated what was in effect a two-stage model, and until physiological evidence from primates (De Valois, Smith, Kitai & Karoly, 1958; De Valois, 1965; De Valois, Abramov & Jacobs, 1966; Wiesel & Hubel, 1966) and fish (Svaetichin & MacNichol, 1958) provided firm evidence for the actual existence of spectrally-opponent cells in the visual path that the situation changed. Since that time, some version of a two-stage model encompassing three cone types combined in a later opponent organization has become the accepted dogma in color vision, the Standard Model (e.g. Guth, Donley & Marrocco, 1969; Ingling & Tsou, 1977; Boynton, 1979; Guth, Massof & Benzschawel, 1980).

Although we, like others, were most impressed with finding opponent cells, in accord with Hering's suggestions, when the Zeitgeist at the time was strongly opposed to the notion, the earliest recordings revealed a discrepancy between the Hering-Hurvich-Jameson opponent perceptual channels and the response characteristics of opponent cells in the macaque lateral geniculate nucleus (LGN). Thus we noted, "the good agreement between recording and psychophysical data breaks down at the short wavelengths" and suggested that "it may indicate that the blue system is amplified in effect at some cortical level" (De Valois *et al.*, 1966). Later investigators also found the same discrepancy, with different stimulation techniques (Derrington, Krauskopf & Lennie, 1984; Kaplan, Lee & Shapley, 1990), and made the same point. Here we suggest (albeit somewhat

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belatedly) a third stage of color processing to reconcile this discrepancy. After formulating our model, we were struck by how similar certain aspects were to suggestions put forth much earlier by Müller (1930) and quantitatively formulated by Judd (1949). That Müller's and Judd's ideas had so little apparent impact can no doubt be attributed to the lack of perceived necessity at the time for such a complicated theory. We believe that at present, however, there is sufficient physiological, psychophysical and perceptual evidence to show that simpler models are not in accord with all the facts, and something more extensive is required.

There are other factors besides the discrepancy between the characteristics of monkey LGN opponent cells and perceptual color space that led us to consider a multi-stage model. One is that, because of the spatial arrangement of their inputs from different cone types, most LGN cells respond to both color and luminance variations, and confound them. As shown by Wiesel and Hubel (1966), most opponent cells in the parvocellular LGN layers appear to have input in the RF center from one cone type, and in the surround from a different cone type. Such cells have sometimes been incorrectly characterized (e.g. Lennie & D'Zmura, 1988) as having a receptive field (RF) that is *both* spatially and chromatically opponent. But we have pointed out (De Valois & De Valois, 1975) that such a cell should be considered to have not one, but two *different* RF organizations, one for luminance and a quite different one for color. A cell with an excitatory L-cone center and an inhibitory M-cone surround, for instance, will have a center-surround *antagonism* for luminance variations, since intensity changes will drive both L and M cones in the same directions and they feed into the LGN cell in opposite directions. An equiluminant color change, however, will drive the L and M cones in *opposite* directions and thus produce a center-surround *synergy*. One would thus predict not only that such an LGN cell would fire to both luminance and color changes (thus confounding these perceptually very different variables) but that it would have different spatial and temporal tuning characteristics for the two. These predictions have been verified (De Valois, Snodderly, Yund & Hepler, 1977). Thus, a second goal for our color model is to provide, at a third processing stage, for the unconfounding of color and luminance.

A third goal for our color model is to incorporate recent information on retinal anatomy and to explore the extent to which essentially random connectivity (as suggested by the anatomy) might result in a sensible color organization. Indeed, we arrived at this model from the bottom up, rather than top down. That is, we assumed certain anatomically-suggested connections and calculated what those would produce at subsequent levels.

Finally, much recent anatomical evidence confirms the extreme paucity of S cones. The Standard Model has one color system (the RG system) based on the outputs of the L and M cones, some 90–95% of the cone population, whereas the whole YB system is centered on just the remaining 5–10% of the cones, the S cones. Such an

imbalance seems inherently implausible, and one of the considerations that led us to our current model was that of attempting to arrive at a more balanced arrangement between the inputs to the red–green and the yellow–blue color systems. One can reasonably argue that the preponderance of L and M cones reflects the fact that these cone types alone are used for luminance detection. However, with current color models, this still leaves one with either an imbalance between the two chromatic systems or the equally distasteful suggestion that only a fraction of the spectrally-opponent information from L and M cones contributes to color vision.

THE MODEL

First stage: three cone types containing three to five cone pigments

We now have very good evidence that the first stage in the processing of visual information is the absorption of light by cones containing different photopigments, and we know quite precisely the spectral absorption functions of the photopigments involved. The most important residual uncertainty is with respect to whether there are in fact two "normal" L-cone pigments, these being separated in peak sensitivity by a few nanometers from each other, and perhaps also two "normal" M-cone pigments (Neitz, Neitz & Jacobs, 1991). If so, there would be some individual differences among "normal" observers, and also differences between males and females. (By "normal" we mean nothing more than that such a system is possessed by the large majority of individuals.)

Another source of individual differences is in the amount of preretinal absorption, primarily by the lens and by the macular pigment. This not only varies from one individual to another, but, in the case of the macular pigment, varies across the retina within an individual eye. Both of these have major effects on the effective pigment curves at short wavelengths, including the short-wavelength portions of the L and M pigments and the sensitivity peak of the S pigment. We have here assumed the values for the lens and macular pigment filters that were assumed by Smith and Pokorny (1975).

Figure 1 shows the spectral sensitivities of the pigments assumed for the first stage. The estimates we have

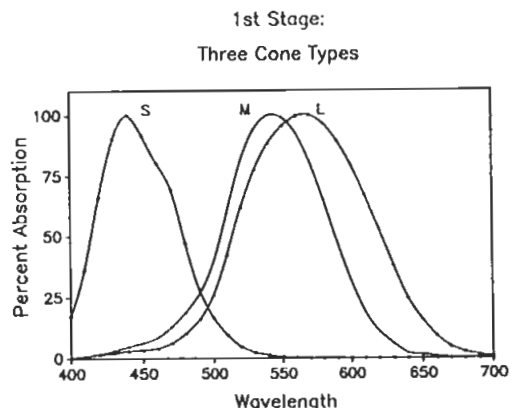


FIGURE 1. Absorption curves for the three pigments assumed for the first stage. We assume the Smith–Pokorny (1975) functions.

used in the graphs shown here are the Smith and Pokorny (1975) functions, which have been shown to agree well with the suction-electrode measures of Schnapf *et al.* (1987), adjusted for preretinal absorption. If other L-pigment curves, shifted by a few nanometers, had been used instead, the curves shown for the second and third stages would be slightly different but not significantly changed.

We assume that, regardless of the number of cone pigments in an eye (whether three, or four, or five), they are contained in only three types of cones, the L, M and S cones. The S cones can now be distinguished anatomically, and constitute only some 3–10% of the cone population, depending on eccentricity, in both macaque (de Monasterio, McCrane, Newlander & Schein, 1985) and human (Ahnelt, Kolb & Pflug, 1987; Curcio, Allen, Sloan, Lerea, Hurley, Klock & Milam, 1991) retinas. Although L and M cones cannot as yet be differentiated anatomically, considerable psychophysical evidence (e.g. Cicerone & Nerger, 1989) suggests that the L cones are on average about twice as numerous as M cones (in the human, but perhaps not in the macaque retina). We therefore assume that the proportions of L:M:S cones are 10:5:1.

A third assumption at this stage is that of how the receptors are arranged spatially. We assume that the S cones are rather regularly arranged, but that the L and M cones are randomly distributed. As a result, there will often be clumps of a number of L cones together (and, less frequently, clumps of M cones as well).

Second stage: cone opponency

The second stage of color processing we postulate takes place in the retina and leads to the development of spectrally opponent and nonopponent cells. There are of course two synaptic levels in the retina, the receptor-horizontal cell–bipolar synapse, and the bipolar–amacrine cell–ganglion cell synapse. We do not know enough in the primate about bipolar cell responses, or about what transformation takes place at the next level to be able to specify at which level (or whether at both) the postulated interactions take place. Thus while we write about the anatomical connections at the receptor–bipolar level, some or all of the interactions specified may instead be at the bipolar–ganglion cell level.

There is also an apparent discrepancy between anatomical studies, which produce a picture of largely random connectivity, and physiological studies, which suggest specific inputs from different cone types to the surrounds of retinal cells. The distinctive feature of our model is in the postulated third stage processing. We have found that it makes little difference to the third stage output of our model whether second stage surrounds are random or cone-specific. We therefore offer two different versions of this second stage.

Most color models have taken as their starting point certain psychophysical data, and modeled what receptor and neural combinations and interconnections are required to account for the psychophysical data. They thus postulate certain specific types of interconnections. We

have reversed this process, by assuming essentially random neural connectivity and then seeing whether the results correspond to psychophysical data. Given the problems of wiring up the brain with its billions of cells from extremely limited genetic instructions, there can be at most a few very general wiring specifications. Random connectivity, which clearly is the simplest to implement, is likely often to be the rule. Furthermore, the current picture of retinal anatomy suggests a largely random connectivity. We proceed on these grounds in developing the random-connectivity version of the second stage. Others have also suggested retinal processing models based on random connectivity (e.g. Paulus & Kröger-Paulus, 1983; Young & Marrocco, 1989; Lennie, Haake & Williams, 1991), but these are somewhat different from ours, e.g. assuming different proportions of different cone types, or random connections in both center and surround.

At the second stage in our model, we assume (in accordance with considerable anatomical evidence) the presence of two different types of bipolar cells picking up from the cones: midget bipolars (and ultimately midget ganglion cells), which [in the central 5–10° of the retina (Wässle, Grünert, Röhrenbeck & Boycott, 1989; Curcio & Allen, 1990)] contact just a single cone, and diffuse bipolars, which contact a group of neighboring L and M cones (Boycott & Dowling, 1969). Note that we are considering the S-cone bipolar described by Mariani (1984a) to be a variety of midget bipolar, since it contacts only a single cone type. In the periphery, the anatomical evidence indicates that the L and M midget bipolars pick up from more than one cone, and they do not appear to distinguish M from L cones (Mariani, 1984b). We consider the resulting complications in the Discussion (*vide infra*). However, we assume, as indicated by Mariani (1984a), that even in the periphery where the S-cone bipolar branches to connect to several cones, it contacts only S cones, avoiding the intervening L and M cones.

In each case, there are two of each type of bipolar contacting a given receptor: one invaginating and one flat midget bipolar, and correspondingly so for the diffuse bipolars. These respond in opposite directions to light changes, one depolarizing to increments and the other to decrements. Thus we assume that each cone is contacted by four bipolars. The midget bipolars contact midget ganglion cells which feed in to the parvocellular geniculate layers, and the diffuse bipolars contact parasol ganglion cells which feed in to the magnocellular LGN layers. We thus postulate that these two paths, with four pairs of cell types, remain separate up to the cortex.

The two alternative versions of the second stage of our model differ with respect to the nature of the inputs to the surrounds of the midget bipolar and ganglion cells. In one version, we postulate an indiscriminate surround, by way of the horizontal cells; in the other version, we postulate cone-type-specific surrounds, produced perhaps at the bipolar–amacrine–ganglion cell synaptic level.

Each cone is also contacted by about four horizontal cells, each of which picks up from a number of surrounding cones (Boycott, Hopkins & Sperling, 1987). In the indiscriminate-surround version of the model, we assume that the bipolar then differences the direct input from the receptor and the indirect input coming from neighboring receptors by way of the horizontal cells. The anatomical evidence indicates that the horizontal cells pick up from all the cones in the region, not showing the selectivity which might suggest that they were contacting just some cone types and skipping others (Wässle, Boycott & Röhrenbeck, 1989; Dacheux & Raviola, 1990). Assuming that to be so, the horizontal cells would be summing over all the cones in a region and subtracting this $L + M + S$ signal (which we will abbreviate as LMS , remembering that these cone types are assumed to be in the proportion of 10:5:1) from the output of each cone type. The signals at the midget bipolars which are depolarized (or excited) by the direct receptor input at this second stage, then, are $L - (LMS)$, $M - (LMS)$, and $S - (LMS)$. There are, in addition, midget bipolars which are hyperpolarized (or inhibited) by the direct input (and excited by the indirect input). These units we will term $-L + (LMS)$, $-M + (LMS)$ and $-S + (LMS)$, there being some question as to the existence or at least the prevalence of the last type. For simplicity, we can also designate these various second stage units L_o , M_o , S_o , $-L_o$, $-M_o$, and $-S_o$, respectively (the subscript symbolizing "opponent").

The physiological evidence from geniculate cell recording (especially Reid & Shapley, 1992) argues for a more discrete surround for parvo cells, specifically, that the cells having an L-cone center input receive an antagonistic input just from M cones and vice versa [as in fact we initially modeled them (De Valois, 1965)]. Thus in this version we model L_o cells as $L - M$; $-L_o$ as $-L + M$; M_o as $M - L$; and $-M_o$ as $-M + L$.

The diffuse bipolars appear to receive a direct input from a small, indiscriminate number of both L and M cones (Mariani, 1981). In the central retina, this is in the order of a direct input from about six cones. Given our assumption of a 2:1 L:M ratio, then, the direct input would on average be four L cones and two M cones. The

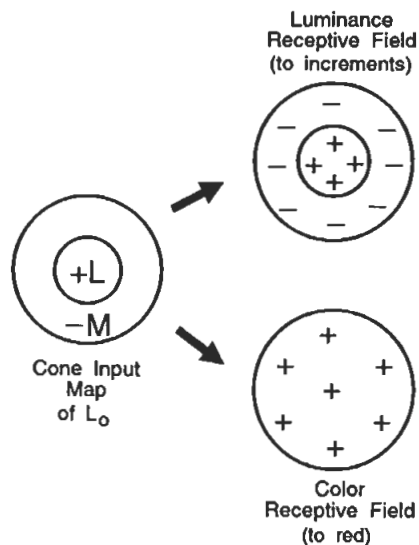


FIGURE 3. Distinguishing cone input maps from receptive fields (after De Valois & De Valois, 1975). An LGN opponent cell with one cone type feeding into the center and another into the surround has not one, but two different RFs. An L_o cell has an excitatory center and inhibitory surround (top) for luminance increments, but a uniform excitatory RF for a color shift towards long wavelengths.

diffuse bipolars also receive an antagonistic horizontal cell input, apparently from all the neighboring cones, so they can be designated as $L + M - (LMS)$ and $-L - M + (LMS)$. These are assumed to form the input to the magno pathway, from diffuse bipolars to parasol ganglion cells to magno LGN cells. These cells would be spectrally nonopponent, with a spectral sensitivity on average corresponding to the photopic luminosity function, V_{λ} . Note that the S cones, by virtue of our assumption of random connectivity of horizontal cells, are postulated to have a small *negative* input to this luminance system (this would be in accord with the psychophysical evidence of Stockman, MacLeod & DePriest, 1991). We emphasize again that the postulated surround inputs may involve amacrine cells at the next level in addition to, or instead of, that of the horizontal cells at the earlier level, as suggested by Kolb (1991).

In general, the receptive field centers of cells early in the visual path are similar in strength to their surrounds. We have thus in the indiscriminate version given a weighting of 16 for the center signal, and a weighting of 16 also for the total surround, assuming that the horizontal cells feeding into a given cone pick up from an average of 16 surrounding cones so that the surround signal is $(10L + 5M + S)$. For diffuse illumination of the RF, input from the same cone type in center and surround would to some extent cancel. Thus L_o is modeled as $16L - (10L + 5M + S) = 6L - 5M - S$. Such a cell would be very similar to the conventional "RG" cell modeled as $L - M$. In the discrete surround version, then, we model these cells as $6L - 5M$, and end up with essentially the same total output whether the surround is assumed to be indiscriminate or discrete. This is even more the case since the S-cone input to the indiscriminate surround of L_o and M_o cells would largely cancel out at the postulated third stage, where we sum

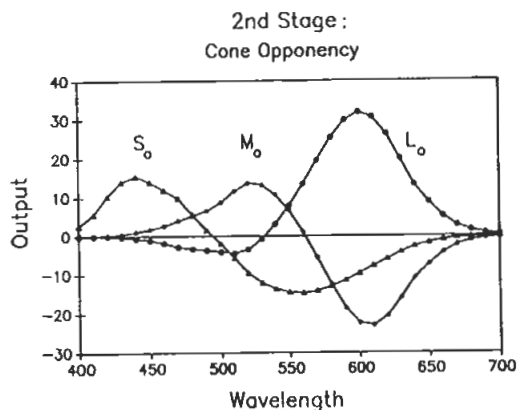


FIGURE 2. Response functions of the three proposed cone-opponent types: L_o [$L - (LMS)$], M_o [$M - (LMS)$], and S_o [$S - (LMS)$]. Each would also have a mirror-image mate, $-L_o$, $-M_o$ and $-S_o$.

M_o and $-L_o$, and L_o and $-M_o$ cell types. So also M_o would be $16M - (10L + 5M + S) = 11M - 10L - S$, or essentially $M - L$. Correspondingly, S_o would end up $15S - 10L - 5M$, or as the conventional "YB" cell modeled as $S - LM$. Note that this organization is arrived at in the indiscriminate version with purely random neural connections, given midget bipolars that receive their direct input from only one cone. In Fig. 2 are shown the calculated responses of the opponent cells we have termed L_o , M_o , and S_o , at this second stage, to increments of different wavelengths of light.

This second stage is based to a considerable extent on anatomical evidence, but there are some other less firmly supported assumptions in addition. In particular, we postulate that L and M cones are in 2:1 proportions and randomly arrayed, and that the surround input is subtractive rather than divisive.

Third stage: perceptual opponency

We assume that the magno system, from the sum of L and M cones to diffuse bipolars to parasol ganglion cells to magno LGN cells, continues at the third stage as a separate system and is responsible for the photopic luminosity function V_l , which is well modeled as the sum of L and M cone pigments in a 2:1 ratio (Vos & Walraven, 1971; Smith & Pokorny, 1975). This is one of the main reasons we assume a 2:1 L:M cone ratio. The

transformations we postulate at the third stage involve not the cells in the magno path, but the cells in the parvo pathway.

We discussed above the presumed six parvocellular LGN types at the second stage. We suggest that the outputs of these various cell types are summed together in various combinations by units at the third stage. The ways in which this summation leads to a separation of color and luminance and to a rotation of the color axes will be discussed in turn. We will then present the complete model of the third stage.

In Figs 3 and 4 we illustrate how the model yields a separation of color and luminance. As discussed in the Introduction, LGN opponent cells respond to both color and luminance variations, but with *different RFs* in the two cases (De Valois & De Valois, 1975). Thus, as shown in Fig. 3, a $+L - M$ cell (L_o) has an excitatory center and inhibitory surround RF in response to a luminance increment, but it has a uniformly excitatory RF for an equiluminant color change towards long wavelengths. On the other hand, a $-M + L$ cell ($-M_o$) has an inhibitory center and excitatory surround RF for a luminance increment but, like the L_o cell, it has a uniform excitatory RF for a color change towards long wavelengths.

Figure 4 illustrates what happens when the outputs of various cell types are combined in different ways. In Fig.

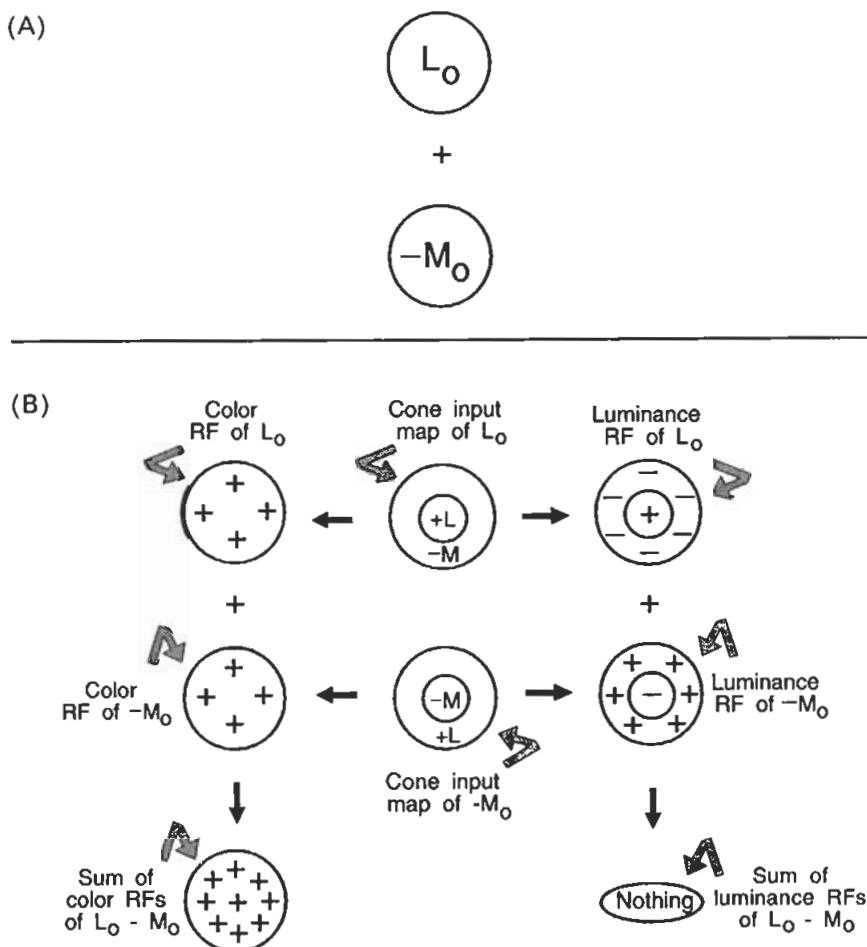


FIGURE 4 (A, B). *Caption overleaf.*

(C)



(D)

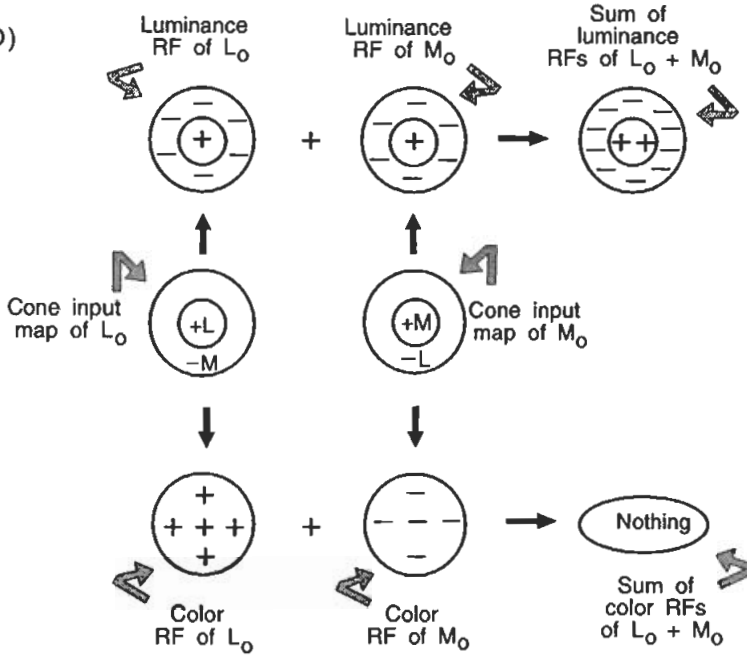


FIGURE 4. Combining stage 2 units in different ways at stage 3 would separate color and intensity information. In (A) and (B) are shown the summation of L_0 and $-M_0$ to extract color; in (C) and (D), L_0 and M_0 are summed to extract intensity. In (B) the L_0 and $-M_0$ cells shown in (A) are shown along with their respective luminance and color RFs. The L_0 cell has $+L$ input to its center and $-M$ to the surround. This gives it a uniformly-excitatory RF to a color shift to long wavelengths (left) but a spatially-antagonistic RF to intensity variations (right), with excitatory center and inhibitory surround to an increment. The $-M_0$ cell, below, also has an excitatory RF to color (left), but an inhibitory center and excitatory surround to intensity (right). Adding the outputs of these two cells would thus sum the color responses and cancel their intensity responses. In (C) and (D) we show how adding this same L_0 cell to a M_0 cell sums the intensity and cancels the color RFs. The cells' intensity and color RFs are now shown above and below their cone input maps. The two cells have the same intensity RFs, an excitatory center and inhibitory surround to an increment, and thus adding their outputs would sum the intensity responses. However, their color RFs are opposite, and would cancel.

4(A, B) we combine a L_0 cell and a $-M_0$ cell to extract color and cancel luminance; in Fig. 4(C, D) we illustrate how adding the same L_0 cell to a M_0 cell extracts luminance and cancels color. The cells being combined are arranged in a column in Fig. 4(A, B), and in a row in Fig. 4(C, D) to correspond to the arrangement of these cells in the complete model shown in Fig. 6.

Figure 4(B) shows the effect of adding together the outputs of the L_0 and $-M_0$ cells shown in Fig. 4(A). It can be seen that since their intensity RFs are opposite, their responses to intensity changes *cancel*. Their responses to color changes, however, *sum* since their color RFs are the same. The resulting third stage cell would thus show a purely chromatic sensitivity. It would respond to color changes but not to luminance changes. Another way to state this is that the two cells in Fig. 4(A,

B) are both essentially differencing $L - M$; they are both what we earlier would have termed $+R - G$ cells. But they respond to a luminance change in opposite directions since their RF centers and surrounds are opposite to each other. Summing them would thus cancel the luminance components of their signals.

Consider now the consequence of summing in the other direction in Fig. 4. In Fig. 4(D) we show the result of adding together the output of the same L_0 cell with a M_0 cell. Each of these cells has an excitatory RF center and an inhibitory surround in response to luminance variations. Adding them together would produce a cell with a strong response to luminance variations. Their color responses, however, are opposites. One cell fires to a shift towards red and inhibits to a shift towards green; the other fires to green and inhibits to red, so the chromatic

components of the cells' responses cancel. Thus summation in this direction results in a cell which responds to a luminance change but not to a color change.

In short, $L_o - M_o$ sums color and cancels luminance; $L_o + M_o$ sums luminance and cancels color. The same would obviously hold for $M_o - L_o$ and $M_o + L_o$. This proposal is similar to the suggestion made by Lennie and D'Zmura (1988) of how one could at some central site separate color and luminance information from LGN parvo cell responses.

Now consider the problem of the chromatic axis rotation. A simple diagram of our proposal for the color axis rotation at the third stage is diagrammed in Fig. 5. We postulate in effect that the outputs of the S_o opponent cells, doubled in weight, are added to (or subtracted from) those of the $M_o - L_o$ and $L_o - M_o$ cells, producing the four perceptual color systems, to give the red-green and yellow-blue axes of perceptual color space. Thus S_o is added (solid line) to $M_o - L_o$ to give blue, and subtracted from it (dashed line) to give green. So also, S_o is added to $L_o - M_o$ to give red and subtracted from it to give yellow. Note that we are assuming proportions of $10(L_o)$ to $5(M_o)$ to $2(S_o)$ at this level.

In essence, we propose that there is effectively just one color axis in the parvo retino-geniculate path: that formed by the 90-95% of the cells with RF center inputs from L and M cones. One can think of this as being not along a unique hue axis, but is instead nearer to an orange-cyan chromatic axis. The role of the S-opponent system, we postulate, is to break this one predominant color axis at some cortical locus into separate RG and YB axes by modulating the LM opponent cells in two different ways. It can thus be seen that in our proposal the M_o and L_o cells are the main contributors not just to the red-green system (as in the Standard Model), but to both the RG and the YB systems. So ultimately, for instance, the M, not the S, cones would provide the primary contribution to the blue signal.

The simple diagram in Fig. 5 does not show the different receptive field types of second stage cells. A more elaborate representation of our model, incorporating that as well as the color-luminance separation, is shown in Fig. 6. In this more detailed diagram, one can see that by summing the second stage units in various ways, color and intensity information can be separated (as illustrated in Fig. 4), while the third stage units outlined in Fig. 5 are being formed by modulation from

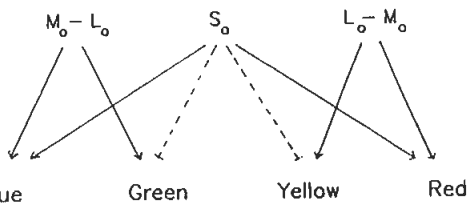


FIGURE 5. the S-opponent units are added to or subtracted from the L_o and M_o difference signals to produce the proposed perceptual color systems at stage 3. The S_o cells are thus seen not as constituting the yellow-blue color system, as in the Standard Model, but as serving a modulatory role on the L_o and M_o systems to produce both the RG and YB color systems.

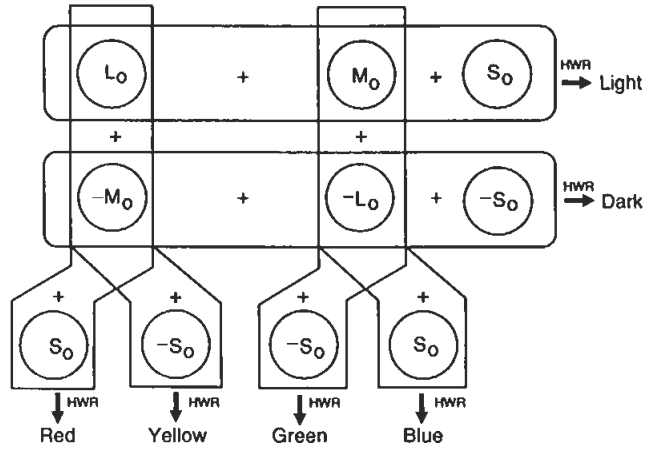


FIGURE 6. A complete diagram of the proposed stage 3, combining the features outlined in Figs 4 and 5, plus the half-wave rectification we propose at a fourth stage. In the horizontal rows, cone-opponent units with the same center sign are added together ($L_o + M_o + S_o$) and ($-L_o - M_o - S_o$) to give achromatic units, since the luminance RFs of the cells add, but their color RFs cancel [as shown earlier in Fig. 4(D)]. In vertical columns, we add cone-opponent units of different center sign. Hence luminance signals cancel and color signals add [as illustrated earlier in Fig. 4(B)]. Note that S-opponent units serve a modulatory role, being added to or subtracted from each combination of L_o and M_o units.

the S-opponent system. This diagram also incorporates the half-wave rectification that we propose for the fourth stage, see below.

In Fig. 6 we show the S-opponent channel as being added to or subtracted from each of the other cell combinations. Along the horizontal rows we add together cells with the same luminance RFs but different color RFs, and thus sum luminance and cancel color to give two opposite achromatic systems. Along vertical columns we sum cells with different luminance RFs and thus cancel luminance while building up different color responsivities.

In Fig. 7 are shown the responses of units at the hypothesized third stage, before half-wave rectification. (Each of these curves would also have its mirror-image mate.) There is a reasonable agreement between these response curves and the colors seen by normal human observers in different spectral regions. Consider the

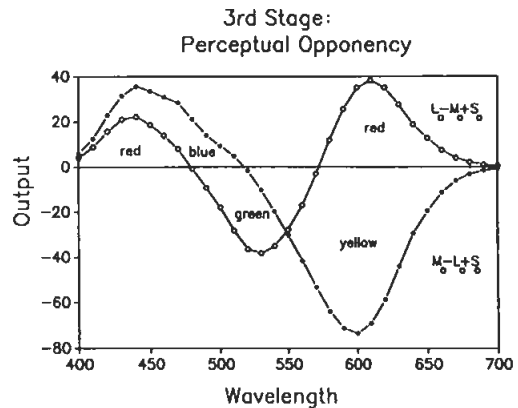


FIGURE 7. The response functions of the third stage units. It can be seen that there is good agreement between these proposed third stage responses and perceptual color space.

unique hues. Unique blue and yellow occur at the cross-points of the red-green system, and unique green at the cross-point of the yellow-blue system. The average unique hues set by some 19 observers in experiments by Purdy (1931), Dimmick and Hubbard (1939), Boynton, Schafer and Neun (1964), Boynton and Gordon (1965), and Werner and Wooten (1979) are as follows: unique yellow, 578 nm; unique green, 514 nm; unique blue, 473 nm; with a range of 10–20 nm around each value. The corresponding cross-points in the postulated third stage functions (see Fig. 6) are 573, 517, and 479 nm, respectively. These are all close to the points set by most observers. The fourth unique hue, red, is extraspectral, at a combination of short and long wavelengths which would just cancel the yellow-blue function.

We have assumed simple linear summation at each level, and the only weighting factor we have introduced (except for assuming a 10:5:1 ratio of L:M:S cones) is that of doubling the contribution of the S_0 units at the third stage. Thus the final curves can be specified in terms of the contributions of the various cone types to the final result. In the indiscriminate version, these are in the proportion of $90L - 115M + 25S$ for the RF system, and $-130L + 95M + 35S$ for the YB system; in the discrete version, these are $90L - 115M + 30S$ and $-130L + 95M + 30S$, respectively. The computations are given in Table 1.

Fourth stage: color-selective complex cells

The final stage of our model is the production of what we might consider to be color-selective complex cells. It is well known (see De Valois & De Valois, 1988 for discussion) that because of their lack of a maintained discharge, most simple cells in effect give a half-wave rectified output. Complex cells can be modeled as summing together half-wave rectified simple cell responses. This, then, is what we propose for this last stage.

One reason for proposing the fourth stage is the evidence that most cells past V1 appear to be complex cells. In fact, one of the things which has led to vastly different estimates by different investigators of the number of "color cells" in later regions, such as V4, is the assumption that "color cells" are all opponent cells like those in the LGN, firing to some wavelengths and inhibiting to others. We suggest that such overt opponency is usually lost at the simple-cell level in V1, and that later color cells are not overtly spectrally-opponent

in their responses. Rather, they simply fire to selective spectral regions and give no response to others. This assumption might appear heretical, quite contrary to the arguments of Hering and of Hurvich and Jameson, which we strongly endorsed in arguing for the opponent organization of color space, but this is not the case. Although we propose that separate cells at this fourth stage fire to red and to green, for instance, these cells will always be in an opponent relation to each other, never both firing to the same stimulus. On the other hand, the cells that fire to, say, red and those that fire to yellow can both be activated by the same stimulus. The chromatic opponency at this stage is between, not within, individual cells.

To understand why the red and green cells in our fourth stage model will always be in opponent relation despite being separate cells (as is true also for blue and yellow cells), consider the following with respect to Fig. 6. At top left we diagram four LM cells, $+L - M$, $+M - L$, $-L + M$ and $-M + L$ which are summed in various ways. The crucial point is that the center mechanisms of these four cells do not pick up from four different receptors, but only two: one L cone and one M cone. The $+L - M$ and $-L + M$ cells are getting their center inputs ultimately from the two midjet bipolars receiving from the *same* L cone, but they respond in opposite ways to the cone and horizontal cell transmitters. These two cells, then, will always be in opponent relation to each other, since they are being driven by the same cones (in the surround as well as in the RF center). So also the $+M - L$ and $-M + L$ cells, which pick up in their RF centers from just a single M cone, will be in opponent relation, as will the $+S - LM$ and $-S + LM$ cells. Thus the summation in the channels that lead to the "red" and "green" fourth stage cells will just be in different (opposite) directions from the same receptors, and the two resulting fourth stage cells will always be in opponent relation to each other in their firing to various stimuli. Of course, most cortical cells have large receptive fields, encompassing not just two receptors as discussed above, but large numbers of L and M cones. The same principle will nonetheless hold, that the $L_0 - M_0$ and $M_0 - L_0$ inputs will not be from the same *groups* of L and M cones and will be in opponent relation to each other.

Exactly the same argument as above holds for the yellow and blue cells. The red and the yellow fourth stage cells, however, will both fire to many of the same stimuli, as will the yellow and green cells, the blue and green, and the blue and red ones.

TABLE 1

Second stage	
$L_0 = 16L - (10L + 5M + S) = 6L - 5M - S$	
$M_0 = 16M - (10L + 5M + S) = -10L + 11M - S$	
$S_0 = 16S - (10L + 5M + S) = -10L - 5M + 15S$	
Third stage	
RG	YB
$10L_0 = 60L - 50M - 10S$	$5M_0 = -50L + 55M - 5S$
$-5M_0 = 50L - 55M + 5S$	$-10L_0 = -60L + 50M + 10S$
$+2S_0 = -20L - 10M + 30S$	$+2S_0 = -20L - 10M + 30S$
Total: $90L - 115M + 25S$	Total: $-130L + 95M + 35S$

The achromatic system(s)

We suggest that there are two non-color-selective systems: the magno pathway, and the achromatic component of the information in the parvo pathway. As indicated in the discussion of the second stage of our model, we assume that the magno path has its origin in the diffuse bipolar cells (feeding into the parasol ganglion cells), which receive their direct input from a small number of both L and M cones (the number

varying with retinal eccentricity, but being about six in the central retina). With an average RF center of 4L and 2M, these cells would have the spectral sensitivity, as well as the characteristic spatiotemporal tuning, of V_L , as indeed magno cells have been shown to have (Lee, Martin & Valberg, 1988).

Although the magno pathway may be exclusively responsible for the classic "luminance" function, there are ample theoretical (De Valois & De Valois, 1975) and experimental (De Valois *et al.*, 1977) grounds for considering that much if not most of the achromatic information involved in vision comes up the much larger parvo pathway, multiplexed with the chromatic information. In fact, one of the main motivating factors in the development of this model was that of suggesting a mechanism for separating the parvo chromatic and achromatic information.

For the (tiny proportion of) cortical units tuned to the highest spatial frequencies the detection of which demands resolution down to the level of the individual cones, it could well be that the L_o and M_o cells are used separately (and indiscriminately) for detecting and characterizing achromatic patterns. Their respective spectral sensitivities for high spatial frequency patterns would approximate those of the L and M cones, which are highly similar to each other. We assume that units tuned to lower spatial frequencies would indiscriminately sum the outputs of groups of L_o and M_o , and perhaps S_o units, as shown in Fig. 6. The half-wave rectified output of such a system would be a three-humped function reflecting the three second stage units feeding into it, not the single smooth V_L function. If the output of this parvo achromatic system were added to the presumed V_L -like output of the magno cells a curve very similar to the perceptual brightness function would result. We thus assume that the relative brightness of various spectral regions corresponds to the sum of the outputs of these two non-color-selective systems.

DISCUSSION

Several factors have led us to this proposal. One is the paucity of S cones in the retina. They constitute at best 10% of the cone population, with the M and L cones comprising the remaining 90%. Yet conventional color models base the whole yellow-blue system on differencing this tiny fraction of the receptors, the S cones, from the rest. The red-green system, on the other hand, is conventionally modeled as being based on differencing the much more populous M and L cones. The Standard Model thus suggests an enormous imbalance between the YB and the RG systems, for which there is scant perceptual evidence. In our model, the M and L cones are the main players in both the RG and the YB color systems, with the outputs of the few S cones operating in effect to modulate the outputs of the others, and thus to shift the color axes to different directions.

A second fact which led us to consider this model, one important aspect of which is the suggestion that M, not S, cones are the primary input to the blue system, is that

under some circumstances the whole spectrum up to 550 nm or more is seen as blue. S-cone sensitivity above about 520 nm becomes vanishingly small, and the positive part of the S - LM difference signal would cut off at even shorter wavelengths. It is difficult to see how even 500 nm in normal foveal vision, to say nothing of 550 nm under other circumstances, could be seen as at least partially blue if the sensation of blue depended on the output of just the S cones. One circumstance under which the short wavelength half of the spectrum looks blue is in the case of tritanopia, in which the S cones are presumed to be absent (or at least totally nonfunctional). Both Ohba and Tanino (1976) and Alpern, Kitahara and Krantz (1983) report that a unilateral tritanope, with only L and M cones functional in the affected eye, says that all wavelengths below the neutral point of about 570 nm in that eye look mainly or entirely blue. The same occurs in the normal observer under conditions which decrease S-cone contributions (Drum, 1989). This led Drum to make a similar suggestion, that M cones provide a major input to blue. To explain these findings within a conventional model, Mollon (1982) proposed a rather complicated "cognitive" explanation.

A third observation that suggested this model for the third stage was that a M-cone input to the blue side of the yellow-blue system would produce the desired rotation of the supposed "YB" LGN system from the tritan to the yellow-blue perceptual axis. There are those who would argue that there is no need for an axis rotation to the perceptual RG and YB axes because psychophysical experiments such as those of Krauskopf, Williams and Heeley (1982) have shown that the cardinal axes are the tritan and constant-S-cone axes, not the perceptual ones. We could not disagree more. There is indeed a stage (ganglion and LGN cells) at which color information is carried by cone-difference signals, and this stage appears to be reflected in the experiments of Krauskopf *et al.* (1982). However, that in no way precludes the existence of a later stage at which the axes correspond to perceptual red-green and yellow-blue, any more than the existence of an LGN opponent-cell stage is precluded by the existence of an earlier three-independent-cone stage. Certain psychophysical procedures allow one to tap in to one neural processing stage; other procedures tap a different neural level.

The identification of the perceptual color axes of red-green and yellow-blue are based on both experimental studies [e.g. hue-cancellation and color naming (e.g. Hurvich & Jameson, 1956; Boynton *et al.*, 1964)] and cross-cultural linguistic evidence (e.g. Berlin & Kay, 1969; Heider & Olivier, 1972) that are every bit as compelling and as valid as the adaptation experiments of Krauskopf *et al.* (1982). There is no reason to assume that either invalidates the other. We argue that they just reflect different processing stages.

Correspondence of postulated third stage with cortical physiology

The core of our model is the proposal of a third, cortical, processing stage at which color and luminance

are separated and the color axes rotated. One might reasonably ask whether this proposal agrees at all with studies of the color characteristics of striate cortex cells. Our first response is that given the wildly varying reports from different cortical studies, it is hard to conceive of any theory about the chromatic response properties of striate cells that would not be in agreement with some and in disagreement with other reports. Consider, for instance, reports on the number of spectral types. Some (Michael, 1978) have reported only red-green cells in cortex; others (Vautin & Dow, 1985; T'so & Gilbert, 1988) report just (or primarily) two types: red-green and yellow-blue (though it is not clear whether these are like LGN cells, or whether they have color axes rotated to true perceptual red green and yellow-blue); still others (Thorell, De Valois & Albrecht, 1984; Lennie, Krauskopf & Sclar, 1990) have reported essentially no grouping of spectral peaks, with cells tuned to each of many different color directions. Reports on the proportions of color-responsive striate cells have also varied from about 10% (Hubel & Wiesel, 1968) to 80% (Thorell *et al.*, 1984). One can thus cite some cortical study in support of (or against) almost any suggestion about cortical color processing.

We suggest that this confusion occurs because (a) the vast majority of striate cells have some chromatic input (that can be seen if one looks carefully for it) reflecting the fact that some 80% of the striate input is from color-opponent parvo LGN cells, and (b) the vast majority of these cells are probably *not*, however, involved in the specification of color, but are merely using color information (along with luminance information) to specify the spatial (or other) characteristics of the stimuli. There are some hundreds of times as many striate cells as LGN cells, and surely only a tiny proportion of these cortical cells is required to specify the color of a region. The problem is that there is no clear agreement on which cells might be relevant and which not.

It might be instructive to consider another stimulus dimension, motion. Virtually every striate cortex cell responds to moving stimuli better than to static patterns, and might thus be characterized as a motion-sensitive cell. However, theorists who have developed motion models agree that only a small subset of these—the directionally-selective cells—are involved with motion discrimination. Motion models are designed to account for the characteristics of just this subset and are not

distressed by the presence of many other motion-sensitive cells with other characteristics. This selection of cells is also aided by the fact that a later visual area, MT, receives its input just from that subset.

In the case of color, there is no agreement on which subset of striate cells should alone be considered relevant to color processing *per se*. Furthermore, the evidence is decidedly mixed as to whether there is a later critical grouping of color cells comparable to MT and motion, although V4 or some subregion thereof is certainly still a possibility.

Variation with eccentricity

The model as discussed above considers only the central region of the retina in which each midget bipolar picks up from just a single cone. Spectral opponency can thereby be produced without having to postulate specific connectivity, without, for instance, having to assume L-cone bipolars which make scattered connections just to L cones, bypassing the intervening M cones. However, one has color vision not only within the central 5–10° in which midget bipolars connect to just a single cone, but over essentially the whole retina. It is also the case, however, that there are losses in color vision with eccentricity, e.g. monochromatic lights become less saturated with eccentricity (Abramov, Gordon & Chan, 1991). It is thus of interest to examine how a model based on random connectivity fares in the peripheral retina where midget bipolars no longer pick up from just a single cone.

Let us examine the consequences of extending our random model to those retinal regions in which the midget bipolars contact more than one cone. The anatomical evidence indicates that the S-cone bipolar maintains a specificity of connectivity even out in the periphery, where it splits its dendrites to make contact with multiple widely-separated S cones, ignoring the intervening M and L cones here as in the fovea. However, we assume that when the M and L cone bipolars pick up from more than one cone, they contact M and L cones indiscriminately. Paulus and Kröger-Paulus (1983) and Young and Marrocco (1989) have made somewhat different but related analyses.

One obvious consequence of the midget bipolar's contacting more than one cone is that there will be occasions in which it will make direct contact with two different receptor types. When each bipolar contacts just one cone, 63% will have L-cone centers and 31% M-cone centers, and 6% S-cone centers, assuming as we do a 10:5:1 L:M:S ratio. All of these will of course be "pure" centers, with just one cone type in the center. When each bipolar receives direct input from two cones, there are three possibilities for the RF centers related to the L and M cones: LL, LM (and the equivalent ML), and MM. These will occur in the proportions of 45% (0.67×0.67), 44% ($2 \times 0.67 \times 0.33$) and 11% (0.33×0.33) of this population, respectively. Assuming that S cones are a fixed 6% of the population (and that they always have "pure" cone centers), the overall percentages of "pure" cone centers with eccentricity will be as shown in Table 2(A). As can be seen, the

TABLE 2. Variations with retinal eccentricity

(A) "Pure" cone centers				
Number of cones in RF center	1	2	3	4
Percent only L cone	63	42	28	19
Percent only M cone	31	10	4	1
Percent only S cone	6	6	6	6
Total percent "pure"	100	58	38	26
(B) Cone dominance				
Number of cones in RF center	1	2-4	5-7	
Percent L dominant	63	42	35	
Percent M dominant	31	38	21	
Percent S dominant	6	6	6	
Percent nonopponent	0	14	38	

percentage of "pure" cone centers drops from 100 to 26% as the average of cones directly contacted by each bipolar increases from one to four.

One can consider the consequences of this computation from either side. It is clear that a strong opponent response would be maintained with random connectivity, even out to considerably eccentric regions. On the other hand, it is also apparent that there would be increasing numbers of units containing mixed cone centers (and therefore presumably less chromatic information) with increasing eccentricity. Both of these conclusions are in qualitative agreement with the perceptual evidence. Color vision is present in the periphery, but it becomes diluted, desaturated, with increasing eccentricity.

A closer examination of the situation reveals it to be somewhat more complex. The proportion of "pure" L-cone to "pure" M-cone centers increases drastically with increasing numbers of cones feeding into the RF centers, assuming a 2:1 L:M cone ratio, see Table 2(A). The ratio of pure L to pure M would increase from 2/1 at the one-cone stage, to 4/1 at the two-cone stage, then to 8/1 and then 16/1, etc. However, this would be more than compensated for by the fact that the majority of units with mixed-cone centers would in fact be biased toward M_o rather than L_o . This can be seen, for instance, in considering the various combinations of 3 (M and L) cone centers: LLL, LLM, LMM, and MMM, which would occur 30, 44, 22, and 4% of the time, respectively. Of the two mixed-cone combinations, only LLM would be nonopponent, having the same 2:1 L:M cone ratio in both center and surround. The LMM combinations would have M cones dominant in the RF center and L cones dominant in the surround. Thus, at this three-cone-center eccentricity, 44% of the (ML) cells would be nonopponent, 30% L-cone centers and 26% all or largely M-cone centers, a higher proportion of M-cone centers than in the fovea. If one sums all the cell possibilities at different eccentricities, see Table 2(B), it can be seen that the percentage of non-opponent units increases from 0 to 38% over the one-cone to the 5-7 cone RF center range, and that the ratio of L to M cone dominance decreases.

The anatomical evidence does not clearly indicate exactly what retinal eccentricities are involved here. The one-cone-per-midget-bipolar (and per midget ganglion cell) regime appears to extend out to about 5° from the fovea, that is, over the central 10° or so of the visual field (Wässle *et al.* 1989). From the ganglion cell and cone counts of Wässle *et al.*, (1989), we can conclude that the 2-4 cone-per-midget-ganglion-cell regime extends from there to perhaps 15° eccentricity, and the 5-7 cone region still further eccentric.

In short, the simple probability considerations we have been examining indicate that random connections of bipolar cells to cones would maintain a considerable degree of cone opponency even out to quite far retinal eccentricities, to where each midget bipolar was contacting a number of neighboring cones. However, some of the resulting cells would be nonopponent, the proportion

of nonopponent (parvo) units increasing with increasing eccentricity.

It is of interest to compare qualitatively these simple computations from our model not only to the psychophysical data, but to single-cell recording evidence as well. A number of investigators have reported the presence of spectrally nonopponent units in the parvo (as well as those in the magno) LGN layers of macaque monkey (De Valois, 1965; De Valois *et al.*, 1966; Wiesel & Hubel, 1966; de Monasterio & Gouras, 1975), although others have argued that these may not be totally nonopponent (Padmos & van Norren, 1975). It has also been reported (de Monasterio & Gouras, 1975) that the percentage of color-opponent units decreases and the percentage of nonopponent parvo units increases with increasing eccentricity. One would predict, from our random cone distribution and connectivity model, that there would be a certain very small number of parvo nonopponent cells in the fovea, corresponding to units that have a center from, say, an L cone and a surround also by chance made up entirely of L cones. With increasing retinal eccentricity, however, the number of nonopponent parvo units should greatly increase, due to the presence of both L and M cones in the RF centers of the same cells, as we have been discussing above. The physiological evidence both for the presence of parvo nonopponent cells and for their increase with retinal eccentricity, then, is consistent with the first two stages of our color model.

Wiring up the third stage

One of the major problems faced by the nervous system is that of developing precise processing circuits from very limited genetic instructions. We have already considered this issue in one version of the second stage of our model, in which we postulate random retinal connectivity. Here we raise the same issue with respect to the central connections postulated for the third stage. The task here is to develop central cells which differentiate between intensity and wavelength variations in the stimulus. We suggest that there may not be a need to develop special central mechanisms to construct circuits to sum the various LGN cell types in these two different ways. The appropriate connections could possibly be made developmentally on the basis of correlations between firing patterns of nearby cells.

The degree of correlation between the stimulus-driven activity of adjacent cells of various second stage (LGN) types should be in the following order.

(1) The highest positive correlation would be between cells of the *same* polarity and *same* cell type (e.g. L_o and L_o cells) in neighboring locations. Such cells will always show a positive (but not perfect) correlation except when a very sharp border, perfectly focused, falls half way between their RF center locations.

(2) The next highest positive correlation should be between cells with the *same* center polarity but *different* ones of the two longer-wavelength-cone-type inputs to their RF centers, e.g. L_o and M_o cells. Given the great similarity between the spectral sensitivities of the L and

M cone pigments, intensity variations in a scene would affect these cells very nearly the same. Red-green color variations would affect them in opposite ways. Intensity variations are probably both more common and larger than wavelength variations in visual scenes, so two adjacent cells of these different cell types should show moderately correlated firing patterns.

(3) The firing patterns of adjacent cells of *different* RF center polarity and *different* cone-type inputs, e.g. L_o and $-M_o$ cells, should be slightly negatively correlated. These cells would respond in synchrony to wavelength variations, but in opposite directions to intensity variations. Thus some of the time they will fire in near synchrony but at other times be out of phase. Again, if intensity variations dominate, these cells should be slightly negatively correlated.

(4) Finally, the firing patterns of neighboring cells with *different* RF center polarity but the *same* cone-type inputs, e.g. L_o and $-L_o$ cells, would be highly negatively correlated.

From this analysis, it can be seen that wiring together in a single cortical RF neighboring cells whose firing patterns are positively correlated (Nos 1 and 2 above) would give a system responsive to intensity variations. Wiring together neighboring cells whose responses are largely uncorrelated or slightly negatively correlated (No. 3 above) would give a system sensitive to wavelength variations in the stimulus. Finally, cells whose responses are strongly negatively correlated (No. 4 above) should never be summed together in a single RF subdivision (of course, inputs from these two types of cells in different RF subdivisions, e.g. center vs surround, or in adjacent bands, would be useful in developing spatial frequency selectivity). Since it is well-established that the correlation of firing patterns between neighboring cells is used in neural development (e.g. Stryker & Harris, 1986), it is plausible that such a mechanism might be used to wire up a system such as we have proposed with minimal instructions.

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