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The first systematic studies of the visual cortex of mammals with well-developed color vision presented an intriguing, and as yet unsolved puzzle. Most retinal ganglion cells and most cells in the lateral geniculate body are color coded; most cells in the primary visual cortex (V1) are not (Wiesel and Hubel 1966; Hubel and Wiesel 1968). In the lateral geniculate body, most cells are overtly color opponent. They are activated by light over one range of wavelengths, suppressed by other wavelengths, and less responsive to white. Presumably, all or very nearly all the signals coming from the lateral geniculate to our occipital lobes must pass through the primary visual cortex. Why, then, are color-coded cells so scarce there?

For physiologists interested in color, the lateral geniculate body had already presented two major related puzzles. The commonest cell type in the parvocellular layers of the geniculate is known as type 1. Its receptive field has a small center, excitatory (on) or inhibitory (off), and an antagonistic (off or on) surround. The center and surround are color opponent, and this color opponency is of two subtypes, depending on the cones that supply the center and surround. The red (long wavelength) cones are in opposition to the green (middle wavelength) cones, or the blue (short wavelength) cones are opposed to the red and green cones combined. (For cells in which blue is opposed to red + green, i.e., yellow, the center almost always is fed by blue cones and is almost always "on.") Because these cells show both spatial and chromatic opponency, they can obviously carry both luminance and wavelength information (Fig. 1). It is surprising, in view of the somewhat subsidiary role of color in primate vision, that 80% of cortical inputs should be color coded.

The second geniculate puzzle concerned the relationship of the organization of type 1 receptive fields to what was known about the psychophysics of color perception. The opponency of red versus green and blue versus yellow corresponds well with Hering's psychophysics (1874), but the receptive-field organization seems exactly wrong for mediating color contrast and color constancy—the fact that colors of objects, in contradistinction to the wavelength composition of the light they reflect, can be largely independent of the wavelength composition of the incident light. To mediate red-green spatial effects, we would expect a cell with a red excitatory center to have a green *excitatory* surround, not a green *inhibitory* surround.

It is far easier to imagine a role in color perception for the less common geniculate type 2 cell, in which the opponent cone inputs have the same spatial distributions. They, too, come in red versus green and yellow versus blue subtypes, and in any given region of the visual field, the combined activity of these two types of color opponency determines unambiguously a point in a chromaticity plane. Since these cells lack center-surround opponency, they cannot carry the lateral interactions needed for color constancy. The receptive fields of type 2 cells are larger than the field centers of type 1 cells.

A third, also less common, type of parvocellular cell, the type 3, lacks color coding altogether. Like type 1 cells, these have center-surround fields, but the red and green cone inputs (perhaps also blue) to the receptive-field center are not opponent but of the same sign, either both excitatory or both inhibitory. Moreover, the spatially antagonistic surround also receives input from both red and green cones. These cells are often termed "broadband."

Cells in the magnocellular layers share some properties with parvocellular cells, but in many respects, they are quite different. They are similar in that their receptive fields are radially symmetric and have a center-surround organization. They differ in four major ways: in their temporal characteristics, color selectivity, contrast sensitivity, and spatial resolution.

1. Magno cells respond faster and more transiently than parvo cells (Wiesel and Hubel 1966; Gouras 1968, 1969; deMonasterio and Gouras 1975; Dreher et al. 1976; Schiller and Malpeli 1978; Hicks et al. 1983). Responses of most magno cells to a long-duration stimulus are brief. A minority show a sustained response component, but even these give a brisk burst of impulses at the onset of the stimulus. This sensitivity to the temporal aspects of a visual stimulus suggests that the magno system may play a special role in detecting movement. Many cells at higher levels in this pathway are indeed selective for direction of movement (Dubner and Zeki 1971; Maunsell and Van Essen 1983).
2. Most parvocellular cells are color opponent, whereas magnocellular cells are not, at least for the more conspicuous, transient part of the response; they have broadband receptive-field centers, like the parvocellular type 3 cells (DeValois et al. 1966; Wiesel and Hubel 1966; Gouras 1968, 1969; deMonasterio and Gouras 1975; DeValois et al. 1977; Schiller and Malpeli 1978; Derrington et al. 1984). In the macaque monkey, many magnocellular cells, both

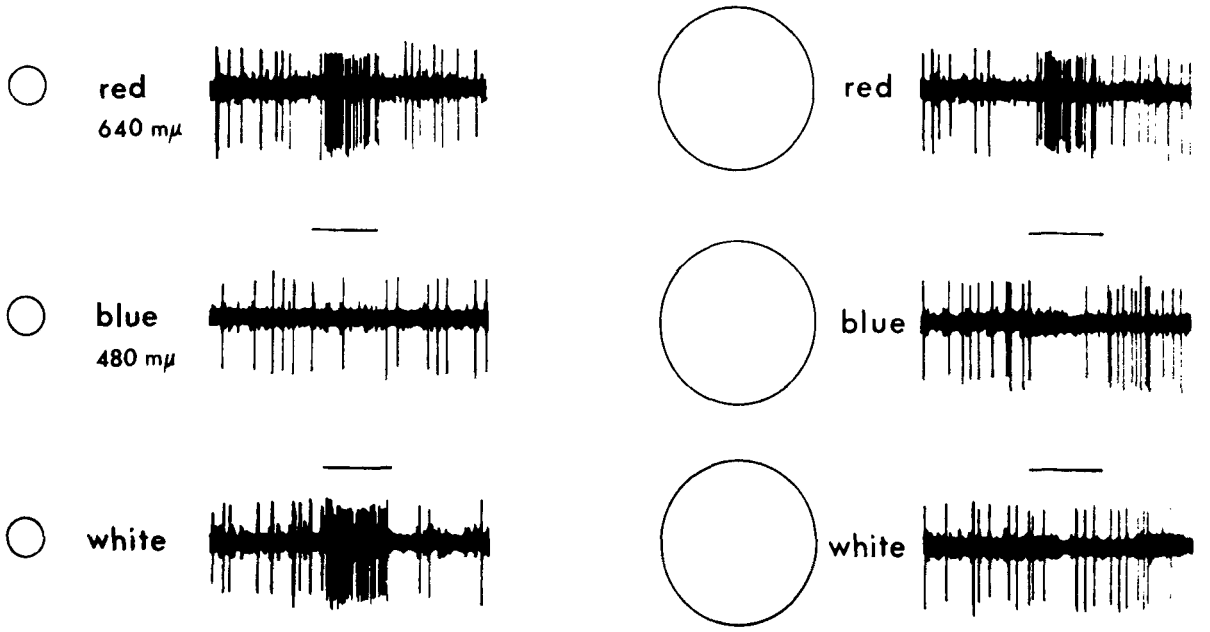

Figure 1. Response properties of a typical parvocellular type 1 cell, demonstrating both its color opponency and spatial opponency. This cell receives excitatory input from red cones over a small central region and inhibitory input from green cones over a larger surrounding area. It shows color opponency in that it is excited by red spots (either small or large) and inhibited by large blue or green spots; it shows spatial opponency in that it is excited by small white spots and not by large ones. (Reprinted, with permission, from Wiesel and Hubel 1966.)

on-center and off-center, show a characteristic sustained suppression of their resting activity in response to a diffuse red light, but not to diffuse white light. To this extent, many magno cells do show color opponency, but what the consequences are in perceptual terms we have no idea.

3. For magno cells, the response as a function of contrast rises much more steeply than for parvo cells, and it saturates at lower contrasts (Shapley et al. 1981; Kaplan and Shapley 1982, 1986; Derrington and Lennie 1984).
4. The final difference between magno and parvo cells is their field-center sizes. At any given eccentricity, magnocellular centers are about twice as large as the centers of the parvo type 1 or type 3 cells (Derrington and Lennie 1984).

Since most parvocellular cells are color opponent and most magno cells are not (except in the sense mentioned above), cells in these two subdivisions respond quite differently to changes in the wavelength of a spot, or to moving color-contrast borders. A type 1 cell will respond when a spot covering the field center is replaced by a spot of a different wavelength, unless the two colors have equal effects on the single type of cone that feeds into the center. The relative intensities at which the effects are equal are, of course, directly predicable from the spectral sensitivity of the center cone type and will be very different for the three types of cones (Fig. 2) (Hubel and Livingstone 1990). Mag-

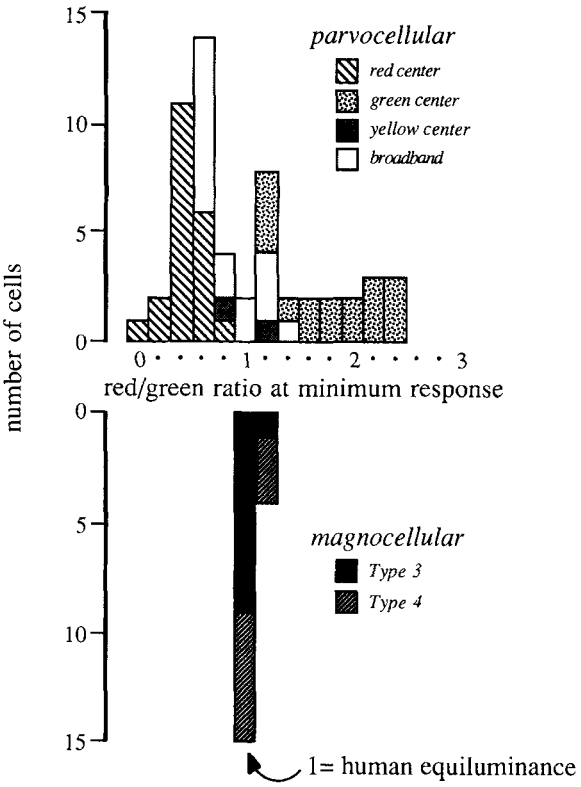

Figure 2. Distribution of parvocellular and magnocellular neurons according to the red/green brightness ratio giving a minimum response. (Reprinted, with permission, from Hubel and Livingstone 1990.)

nocellular receptive-field centers, on the other hand, receive an input of the same sign, either excitatory or inhibitory, from both red and green cones; one would therefore expect these cells to be insensitive to the alternation of two colors whose relative brightnesses are such that the sum of the effects on the two cones is the same. If, from cell to cell, the relative influences of the red and green cones feeding into the center are close to the same, then the positions of the minima that occur as relative brightnesses are varied should all be similar. This is indeed the case, as shown in Figure 2. Krüger (1979) and later the two of us (Hubel and Livingstone 1990) found that magnocellular geniculate cells show a response minimum to a moving color-contrast border at a particular relative brightness—a brightness ratio that is close to a human observer's equiluminant point. (Logothetis et al. [1990] have shown, and we have confirmed, that many magno cells, although showing a minimum at some setting of relative intensities, do not null completely at equiluminance, but respond briefly and weakly at both the red-to-green and the green-to-red transition. Evidently, the effects of removing one color are not precisely matched by introducing the other. Thus, some color sensitivity does persist, although the cells are insensitive to sign of the color contrast.) The fact that magnocellular cells respond so poorly at equiluminance was one of several lines of reasoning that prompted our hypothesis that the aspects of visual perception that deteriorate at equiluminance do so because they are carried predominantly by the magno system (Livingstone and Hubel 1987).

In the primary visual cortex, the pathways stemming from the two major geniculate subdivisions, magnocellular and parvocellular, seem to keep their separate identities. Several anatomical possibilities for cross talk do exist (Lund and Boothe 1975), and indeed it would be hard to imagine a complete independence, given the fact that at least two columnar systems, for ocular dominance and orientation, extend through the entire cortical thickness. So far, however, we have no other convincing physiological indications for extensive interactions.

In the cortex, cells in layers derived from the magnocellular system again show a marked decline or loss of responsiveness at the red-green intensity ratio that corresponds to human equiluminance. In the parvocellular system, things are more complicated, as one might expect from the wide variety of parvo geniculate cells. Here, we confine our discussion to layers 2 and 3. These represent the part of the (mainly) parvocellular path in V1 that projects to other cortical areas and are thus presumably involved more directly in perception than the subcortically projecting infragranular layers (5 and 6).

Leaving aside for the moment the blob system in layers 2 and 3, there are still some exceptions to our opening statement that cells in V1 lack color coding. A few upper-layer cells, although showing about as much orientation selectivity as their neighbors, give far stronger responses to colored bars than to white bars. Such cells make up about 10% of the upper-layer population and may be located at the edges of blobs (Livingstone and Hubel 1984; Hubel and Livingstone 1990). Curiously, of the cells that prefer colored bars, those that prefer red bars are by far the commonest. Over many years, we have seen one or two that preferred blue, and none that preferred green. Many cells respond well to a black bar on a light background, and hardly at all to a light bar on a dark background. The great majority, however, show no color preference and respond about as well to light bars as to dark.

In 1979, Gouras and Krüger made the remarkable observation that many cortical cells that show no overt color preference do carry color information of a more subtle kind. When they used a red bar on a green background to test cells that responded well to both dark and light bars, about half of the cells responded well at all red-to-green intensity ratios. This meant that the color information coming into the cortex from the geniculate had not been entirely lost. We have recently replicated their result (Fig. 3) not only with bars, but also with red-green sine-wave gratings at low spatial frequencies, at which chromatic-aberration effects are negligible. It therefore seems clear that interblob cells in layers 2 and 3 can detect borders on the basis of either luminance or color. Although subtle, this is probably the most prevalent form of color coding in the striate cortex.

In the early 1980s, we discovered a completely distinct color system in the upper layers of V1 (Livingstone and Hubel 1984). About half the cells in the cytochrome-oxidase blobs were overtly color coded, and all the blob cells showed poor orientation selectivity or none. Some of the color-coded cells were similar to type 2 geniculate cells, with color opponency but no receptive-field surround. Most, however, had more complex receptive fields, with a receptive-field center like that of a type 2 cell, but also a surround that suppressed the response at all wavelengths (Fig. 4). A typical cell of this type might give on responses to a small red spot, suppression of firing and off responses to a small green spot, and little or no response to a white spot of any size, or to a large spot at any wavelength. Such cells were reminiscent of the "double opponent" cells that had been seen by Nigel Daw (1968) in goldfish retina.

Several questions remain unresolved concerning the physiology of these blob cells:

1. The use of the term "double opponent" has been criticized (T'so and Gilbert 1988). One objection to its use has been that annular stimuli, confined to the field surround, often produce no response. Yet the absence or weakness of an explicit surround response is a common feature in all types of center-surround cells in both cat and monkey, and also in simple cortical cells. It was first emphasized by Barlow (1953), who pointed out that even in cells that showed no response to annuli, the surround re-

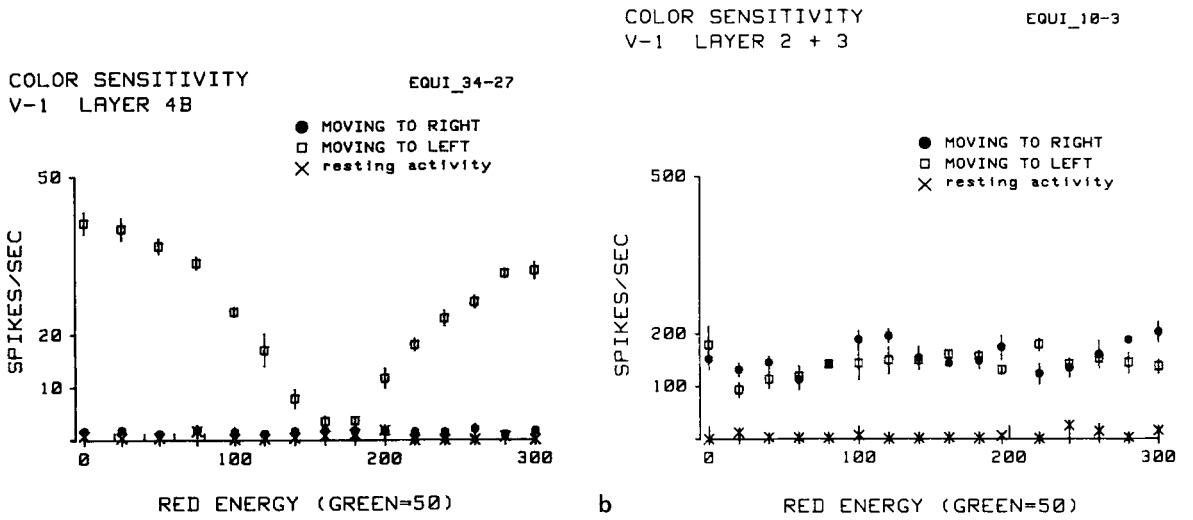

Figure 3. Responses of cortical cells to a red bar moving across a green background as the red brightness is varied. Red = 150 corresponds to human equiluminance as determined by flicker photometry. (*a*) Typical layer 4B cell. Cells in layer 4B, which receive predominantly magnocellular input, show deep minima at equiluminance. (*b*) Response of a typical layer 2/3 cell to the same stimulus. This cell does not show a response decrement at any particular red/green ratio. About half the cells in layers 2 and 3 respond well at all red/green ratios. (Reprinted, with permission, from Hubel and Livingstone 1990.)

sponses could be brought out by tonic illumination of the field center. Explicit responses from the field surround seem to be easier to elicit in the goldfish, but even there they may be weak (N.W. Daw, pers. comm.).

2. The surrounds of these receptive fields present a more perplexing difficulty, noted by T'so and Gilbert (1988). The simple description of the cell's behavior just given—color opponency plus failure to respond to large spots—led us to think of the receptive field of, say, an R^+ G^- center cell as having an R^- G^+ surround. If such a summary diagram is taken literally, then we would expect the cell to give an especially powerful response to a red center-size spot surrounded by green. In most cells, however, the response to a center-size red spot is strongly suppressed by a green annulus. Clearly, no simple summation of responses can adequately characterize such a cell. T'so and Gilbert (1988) call these cells "modified type 2" and describe them as having a broadband inhibitory surround. This seems to us inappropriate, since "inhibitory" does not adequately describe a surround that suppresses both on and off responses. Moreover, we found that in some cells, a red surround is more powerful than a green in inhibiting the response to red stimuli in the receptive-field center, and a green surround is more powerful than a red in inhibiting the green center response. Such cells may therefore be suited to building up the kinds of responses needed for the perception of color contrast and color constancy.
3. About half the cells in the blobs lack any obvious color coding; qualitatively, they are similar to the broadband type 3 cells in the geniculate, except that they tend to have larger field centers. It is not clear whether these cells get their input from broadband geniculate cells and, if so, whether magnocellular or parvocellular.
4. A full understanding of the blob cells and their relation to color vision depends on knowing their inputs. In the squirrel monkey, Fitzpatrick et al. (1983) have provided evidence that the interlaminar plexuses of the geniculate project selectively to the blobs. For the macaque monkey, we have hints of the same thing. The type 2 cells we have recorded from have tended to be near the interlaminar plexuses (Livingstone and Hubel 1982, 1984, and unpubl.), and these cells are the most logical candidate for building up double-opponent cells. This system may have its origin in the small-cell population of retinal ganglion cells (W-cells) (Casagrande et al. 1990).

Although our present knowledge of the anatomy and physiology of the pathway subserving color is too fragmentary to justify any firm pronouncements, it may be useful to propose a hypothesis that has the merit of being testable. If it evokes as lively a response as our recent ideas on the role of the magnocellular system in movement and depth perception, we will feel fully vindicated!

Our notion is that color vision, in the sense of recognizing and distinguishing colors, as opposed to recognizing shapes or borders through the use of wavelength differences, may depend on the relatively rare type 2 cell (center only, with color opponency) and not on the far commoner type 1 cells (center-surround, with color opponency). We propose that inputs from type 1 cells are pooled in the interblob regions of the upper layers of V1; this pooling of color-opponent inputs results in the loss of sensitivity to the sign of color contrast while retaining responsiveness to color borders at all relative

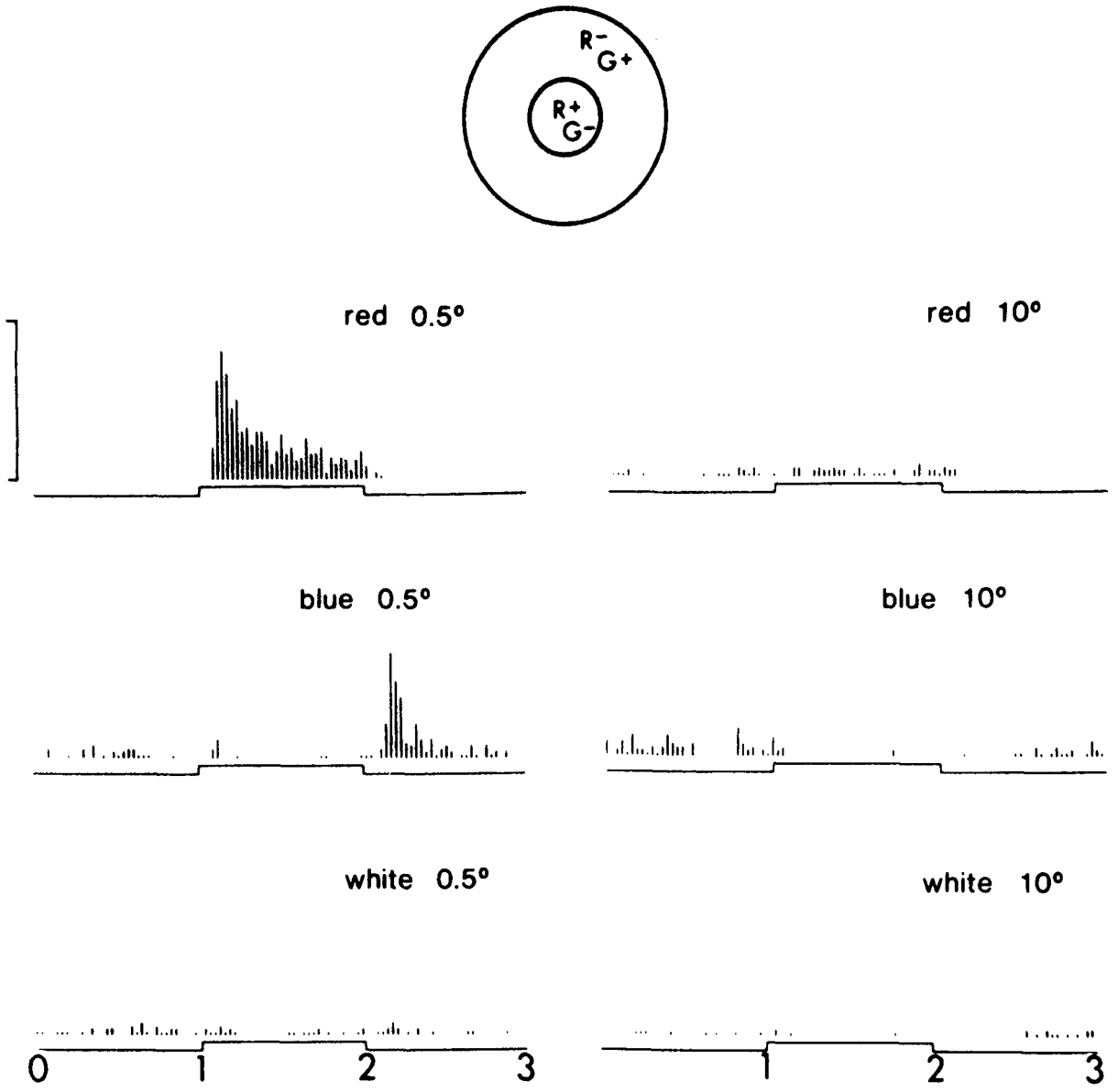

Figure 4. Responses of a double-opponent cell in a cytochrome-oxidase blob of V1. Although annuli alone give no responses, the surround inhibition is apparent as a reduction in the response to larger spots for both red and blue light. Since the surround antagonizes both the on-center and the off-center responses, it cannot be simply a broadband inhibition. (Reprinted, with permission, from Livingstone and Hubel 1984.)

intensities. Upper-layer interblob cells can thus use color contrast as well as luminance contrast for perception. To camouflage itself, a plant or animal must blend in with its background, and this is obviously harder to do for both luminance and wavelength than for luminance alone. We suggest that it is the type 2 cells and the double-opponent cells in the blobs that are responsible for the perception of color as such.

The following observations would seem to support this idea:

1. The low resolution of our color vision, as psychophysically determined, matches the large field size of type 2 cells. The much tinier centers of type 1 cells are a better match for our spatial resolution.
2. It may seem perverse to imagine that the obvious color coding of the type 1 geniculate cells is thrown away, or at least not used for our color sense as usually construed, but rather for the seemingly more humble task of defeating camouflage in the high-spatial-resolution form system. The color opponency of type 1 cells may, however, simply be a by-product of their high spatial discrimination. In the fovea, at least, it seems clear on anatomical grounds that each receptive field center receives input from only a single cone (Polyak 1941; Boycott and Dowling 1969). That single cone must be of one kind or other—specifically either a red cone or a green cone, given the absence of blue cones in the fovea (König 1894; Wald 1967). This cone purity of the

field center is by itself enough to make the cell a carrier of color information. It is less clear why the receptive field surround should be selective for the opponent color rather than broadband. Yet the evidence that type 1 cells have color-opponent surrounds seems clear (Fig. 1). With long-wavelength stimuli, a red on center cell gives on responses of equal magnitude to small and large spots, whereas with short wavelengths it gives a much weaker response to a large spot than to a small. Moreover, spectral sensitivity curves of the surround (Wiesel and Hubel 1966) seem to indicate that they are not broadband. This wavelength selectivity of the surround poses another puzzle. Horizontal cells in the monkey are thought on anatomical and physiological grounds to receive inputs of the same sign from all cone types (Boycott et al. 1987; Dacheux and Raviola 1990); they should therefore be broadband. If both the anatomy and physiology are correct, we may be forced to ask whether the field surrounds of type 1 cells in primates are dependent on amacrine cells rather than on horizontal cells.

3. Using a technique developed by R. Savoy and M. Burns (pers. comm.), we can change the three phosphors of a color monitor in such a way that when a red spot replaces a gray spot, the activity in only the red cones is changed; similarly, a spot can be made to turn from gray to green and change activity only in the green cones, or gray to violet and affect only the blue cones. A protanope is quite blind to a large spot as red as a tomato, and similarly for a deuteranope and an unripe tomato. If we attempt to drive our cortical cells with such stimuli, we find that whereas type 2 cells typically respond vigorously, type 1 cells respond grudgingly or not at all. The contrasts that can be obtained with such stimuli are limited by the maximum brightnesses obtainable with the TV phosphors, and by the fact that the lowest one can go in intensity is to turn the phosphor off. Type 1 cells, whose contrast sensitivities are low, are apparently too insensitive to respond. The fact that a stimulus we have no trouble seeing as colored fails to drive type 1 cells seems to be further evidence that we can see colors without them.

On anatomical and physiological grounds, the blobs project to the thin dark stripes of visual area 2, which in turn project to area V4 (Livingstone and Hubel 1984; DeYoe and Van Essen 1985; Shipp and Zeki 1985). Area V4 may be homologous to the region in the human inferior occipital lobe whose destruction leads to achromatopsia (Pearlman et al. 1979; Damasio et al. 1980). In this rather rare condition, color perception, in the sense of recognition or discrimination of colors, may be completely destroyed with little or no impairment of form vision. Such patients should still be able to use their type-1-to-interblob color information to detect color-contrast borders at all relative brightness, while unable to tell the colors that make up the border. We hope to test this prediction.

We do not know the mechanisms responsible for color contrast or color constancy, or even whether the necessary circuits are present in V1. Evidence from Zeki's work indicates that V4 is involved (Zeki 1980, 1983). Wherever the circuits are, they are presumably fed by cells in the striate cortex, and one should at least expect those cells to have properties not incompatible with color constancy. As already mentioned, type 2 cells are compatible.

We have tried to describe how, despite the enormous amount of information we have about the organization, function, and development of the retina, the geniculate, and the visual cortical areas, color perception still is largely a puzzle. It is hardly any consolation that form perception is even more of a mystery.

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