Color vision: **Putting it together**

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Color vision depends on the visual system comparing signals that originate in different classes of cone photoreceptors. New work shows that the different classes of cones are not only distributed irregularly, but in different individuals they are present in very variable proportions. Surprisingly, this does not affect color vision.

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The fundamentals of color-vision have been well-understood for a long time. By the early 19th century, Young had deduced that the normal human eye contained three sensing mechanisms selectively tuned to different but overlapping regions of the visible spectrum. Each of these mechanisms — now known to be distinct classes of cone photoreceptors — signals the number of photons it absorbs, but nothing about the spectral composition of the light. Changing the spectral composition of the light alters only the probability that photons will be absorbed by a cone. The color signature of any point in a scene is therefore represented in the nervous system by just three numbers that specify the photon catches in each of the three classes of cones. This is known as trichromacy. Many physically different spectral distributions of light can give rise to the same three numbers, and the normal visual system will see these as identical. This fact is the foundation of color rendering systems that create a wide range of colors by mixing a small number of primary sources in appropriate quantities.

The principle of trichromacy tells us which different spectral mixtures will look identical, but it does not tell us why these mixtures look the way they do. Interesting observations about color appearance that are beyond the reach of the principle of trichromacy led Hering to suggest in the mid 19th century that the mechanisms of color vision are organized as three pairs of polar opposites: light–dark, red–green and yellow–blue. The principle of opponent organization explained an array of facts about color appearance — including why red, green, yellow and blue have special stature as anchoring colors; why one does not experience reddish–green hues; and why particular mixtures appear white. But the three mechanisms on which color opponency was suggested to depend are not like those postulated to account for trichromacy.

Trichromacy and color-opponency are reconciled by recognizing that they characterize different stages in the analysis of color: the three classes of cones constitute the first stage, and their signals are later combined at a second stage that gives rise to opponent mechanisms. These principles, which have been established through studies of human perception, provide sharp guidance to biologists studying the underlying machinery, and have made physiological studies much more incisive than they could have been otherwise. The big ideas have been reassuringly confirmed over the past thirty years [1,2], but recent studies show unsuspected and intriguing complexities in the organization of chromatic mechanisms in the retina and at later stages of the visual pathway.

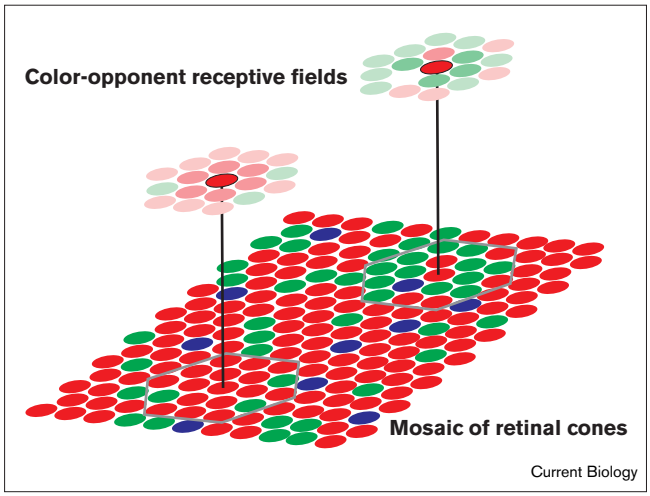
One puzzle is highlighted in a collection of papers published recently in the *Journal of the Optical Society of America* on the "chromatic topography of the retina" [3]. To understand how the chromatic machinery is put together, we need to know the proportions and distributions of the long-wavelength sensitive (L), middle-wavelength sensitive (M) and short-wavelength sensitive (S) cones. The S cones are morphologically and histochemically distinctive; they constitute about 8% of cones and are arranged in a quasi-crystalline mosaic [4]. Until very recently, the L and M cones were enigmatic. Individual cones could be identified as L or M by measuring their spectral characteristics, but we had no secure means of assessing their relative numbers or distributions on the retina. The difficulty probably stems from the fact that distinct L and M cones — reliably present only in old-world primates — are a recent evolutionary development from a single ancestral cone type [5], and their genes and gene products are very much alike.

Recent work has substantially cracked this problem. The new papers assemble converging evidence — from perceptual experiments, analysis of the amounts of L and M cone mRNAs in the retina, very high-resolution imaging of the photoreceptor mosaic in the living eye, and recording of the electroretinogram — that L cones outnumber M cones by about 1.6:1 in the average eye. Much more intriguing is the finding that this ratio varies by at least a factor of four among individuals, yet this seems to have negligible effect on their color vision [6]. We would not expect variations in cone ratios to affect the underpinnings of trichromacy, because physically different mixtures of lights that cannot be distinguished give rise to identical signals in every cone, but we might be very surprised that different ratios of cones have so little effect on the opponent mechanisms that gather and compare signals from different classes of cones, and do influence appearance.

There is an additional puzzle: using an adaptive optical system tuned to compensate for local aberrations in the optics of an individual's eye, Roorda and Williams [7] have obtained astonishingly high-resolution images of the mosaics of L, M and S cones in the normal living eye, and have shown not only that there are large inter-individual variations in the L:M cone ratio, but that neither mosaic is at all regular, instead being clumpy, possibly random. All this ought to frustrate the assembly of the red–green opponent mechanisms that compare L and M cone signals locally in the image, and variable color perception should result.

We know that opponent mechanisms arise in the retina: midget ganglion cells, whose axons convey visual information from the retina to the brain, are color-opponent, being excited by light in one wavelength band and inhibited by light in another. Each cell picks up signals from cones in a small region of retina — its 'receptive field', organized in concentric 'center' and 'surround' regions that antagonize each other, one being excited by illumination, the other inhibited (Figure 1). We know that, in most receptive fields, center and surround have different spectral sensitivities [8], and it has been generally presumed that center and surround are each fed signals from a single class of cone. But given wide variation across individuals in the relative proportions of the L and M cones, and irregular arrangements of them, how can one assemble a small receptive field that segregates different classes of cones in center and surround?

Figure 1



How red–green color-opponent mechanisms might be formed in the retina. The different classes of cone photoreceptors are distributed irregularly in the retinal mosaic, illustrated at the bottom of the diagram. The short-wavelength sensitive (S) cones, colored blue, constitute less than 10% of the total, and are not part of the red–green mechanism. The middle-wavelength sensitive (M) cones, colored green, and the long-wavelength sensitive (L) ones, colored red, are present in proportions that vary widely among individuals, with the L cones outnumbering the M cones, on average, by perhaps 1.6:1. The red–green opponent mechanism, two examples of which are depicted by the smaller circular arrays at the top of the diagram, probably has its origin in the midget bipolar cell, which is connected directly to a single cone that forms the center of its receptive field, and indirectly, via horizontal cells, to a pool of neighboring cones that form the antagonistic surround. The small circles in the circular arrays each represent a cone in the receptive field, with color representing the weight of the signal (hence the central cone is in a saturated color, and those in the surround are paler, representing their lower weight). The strength of color-opponency depends on the particular pool of signals that make up the surround. In the two examples shown here, the single L cone in the center, identified by the line connecting it to the mosaic, is opposed in one case by a surround that contains few M cones (leading to weak color-opponency), and in the other case by a surround that contains predominantly M cones (leading to strong opponency).

An intriguing answer to this question, for which evidence is accumulating, is that the visual system does not bother to, or cannot, do this in any principled way. Instead it relies on a simple expedient: each receptive field forms its center from one or a few juxtaposed L and M cones, and forms its surround by drawing indiscriminately on all L and M cones in a larger surrounding area. If the center draws input entirely or mostly from a single cone — very likely to happen in the fovea and nearby retinal regions — and not all cones in the surround are of the same class, then the neuron must be color-opponent. When receptive field centers become larger, as they do with increasing distance from the fovea, the center will draw on several cones, which might not all be of the same class. Color-opponency will therefore be weakened, leading to a corresponding perceptual decline, but even in the retinal periphery the clumping of cones will ensure that some centers gather signals from cones of a single class.

Until now there has been no direct evidence for this idea, but it draws support from a series of important findings by Dacey and colleagues [9–11], who have recorded signals from generally inaccessible neurons in the primate retina. L and M cones provide signals to both centers and surrounds of midget ganglion cells in peripheral retina [9]. Bipolar cells, the intermediaries between cones and ganglion cells, have center–surround receptive fields like those of ganglion cells, and probably account for the ganglion cells' receptive fields [10]. The bipolar cell's surround seems to arise in the H1 class of horizontal cell, which collects signals from only L and M cones in proportions that vary greatly from cell-to-cell and probably reflect the numbers of L and M cones available locally in the retina [11].

Retinal circuitry thus seems to be haphazard in its dealings with L and M cones, yet its indiscriminate handling of their signals finds no expression in our color vision. How does the visual system mask the huge variation, within and between individuals, in the signals L and M cones provide to chromatic mechanisms? Part of the answer probably lies in the way that ganglion cells, through bipolar cells, weigh their connections with cones in the receptive field's center

and surround. We know that, in general, center and surround are equal in their aggregate sensitivity — that is, when one illuminates the receptive field uniformly, the cell does not respond. If this normalization is an organizational imperative, then variations in the proportions of L and M cones in the retina will be substantially discounted automatically [12]. More stability in color perception is contributed by the way in which later stages of the visual pathway aggregate the signals from color-opponent cells in the retina. Lots of perceptual evidence points to the fact that we sense color variations in images on a relatively coarse spatial scale — coarser than the sizes of retinal receptive fields. Mechanisms in the cortex must therefore collect signals from many retinal inputs, thereby smoothing-out the local variations among them.

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