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Color Vision, Neural Basis of

Intermediate article

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CONTENTS

The brain determines color from different wavelengths. Specialized cells in the retina, thalamus, primary visual cortex and higher brain areas achieve color perceptions by taking into account chromatic context, which explains various color illusions and color constancy.

INTRODUCTION

A world without color is bleak. Despite this, the benefits of color vision are difficult to quantify. Picasso said, 'When I run out of blue I use red', by which he meant that it is the brightness of a pigment and not its color that describes the three-dimensional shape of objects. Matisse demonstrated this point beautifully in his painting *Femme au Chapeau* (Figure 1). A gray-scale reproduction shows that the values of the pigments do not interfere with an accurate representation of the play of light across his subject's face. That the painting reads well as a face despite the radical color transitions shows that color is not an important cue to shape. In fact, object shapes are easily recognizable even in dim light when color vision is

(a) (b)

Figure 1. [*Figure is also reproduced in color section.*] (a) Henri Matisse, *Femme au Chapeau* (Paris, autumn 1905). Oil on canvas, 80.5 cm × 60 cm; San Francisco Museum of Modern Art (bequest of Elise S. Haas). The gray-scale reproduction (b) shows that the surprising color transitions do not interfere with an accurate representation of the woman's face. Yet the color reproduction is obviously more appealing – why? The dissociation of color and form, clear in this picture, shows that color is processed by the visual system separately from other stimulus attributes, like form.

absent. Moreover, many people function perfectly well with impaired color perception: about 1 in 12 men are red–green color-blind and many of them are unaware of it. However, color cues are useful. In monkeys, they assist the discrimination of ripe fruit and of suitable procreative partners, and in humans, color is more than a cue for discriminating objects, for unlike shape and texture, color has emotional significance. One is 'green' with envy, 'red' with anger, 'blue' with sadness. Indeed, Matisse used this to push his portrait past mere representation. Moreover, his picture is much more appealing in color than in black and white. It is probably the emotional quality of color that has fueled color vision research, and it also helps explain the passionate controversies that fill this field's history.

COLOR VISION: WHAT IS IT?

Color vision is the ability to discriminate surfaces based on the spectral content of the light reflected from them, taking into account the light reflected from surrounding objects. Color vision, which has evolved in many animal groups such as insects and birds, is crude in most mammals except some primates, such as humans. Research has focused on color vision in Old World monkeys because their color vision is virtually identical to that in humans (De Valois *et al*., 1974).

THE ROLE OF CONES

In 1802 the English physician Thomas Young proposed that color was subserved by three classes of sensors, each maximally sensitive to a different part of the visible spectrum (Figure 2a). This trichromatic theory, extended by Helmholz in 1866, resolved a profound problem. Though we can see millions of different colors, our retinas simply do not have enough space to accommodate a separate detector for every color at every retinal location, as proposed by Newton. Given that almost all hues can be matched by the combination of three primary colors, and that the number of receptors for color at every retinal location must be small, Young's proposal of three sensors was reasonable. Moreover, it changed the way we think about color: the bottleneck imposed by the small number of sensors implied that color was a neural construction reflecting both physical properties of light and biological properties of photopigments, neurons and networks of neurons. Color is a perception, and not a property of the world.

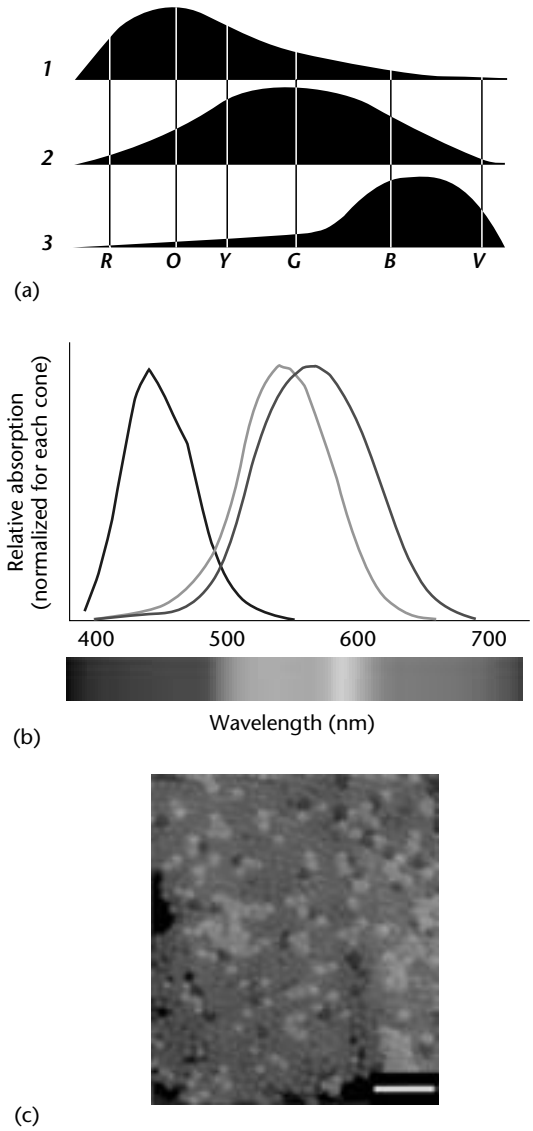


Figure 2. [*Figure is also reproduced in color section.*] Color perception begins in the retina of the eye with three classes of photoreceptors called cones. (a) The absorption spectra of the three detectors proposed by Helmholtz in 1866: shorter wavelengths (V, or violet) were represented on the right. (b) The actual cone absorption spectra of the three cone classes, L, M and S, based on the cone fundamentals of Smith and Pokorny (1972). Convention today puts shorter wavelengths on the left. Below the plot is the visible spectrum. (c) The cone mosaic of a patch of living human retina made visible with adaptive optics, from Roorda and Williams (1999). The S cones are represented by blue, M by green and L by red. Scale bar, 5 arc minutes of visual angle.

We now know that color perception begins in the retina with photoreceptors called cones (Figure 2b, c). Cones are more densely packed in the portion of the retina corresponding to the center of gaze (the fovea), and become less dense in the periphery. A second type of photoreceptor, rods, are absent from the fovea. Rods function best in dim light, when the cones do not function well. Because we only have one class of rods, and a comparison between at least two classes of photoreceptor is required for color vision, rods for the most part are not involved in color perception, and we do not see color in very dim light.

Cones are divided into three classes according to their peak absorptions: the S cones absorb shorter wavelengths optimally (peak 440 nm); the M cones absorb middle wavelengths (peak 535 nm) and the L cones absorb long wavelengths (peak 565 nm). All cone classes are somewhat sensitive to wavelengths throughout most of the spectrum (Figure 2b). Thus a single class of cones is color-blind because it cannot distinguish between a dim light of optimal wavelength and an intense light of less optimal wavelength. Moreover, at any given point in the retina there is only one cone, so the retina is color-blind on a spatial scale of single cones. Despite these facts, the cones are often loosely called 'blue', 'green' and 'red', because these names are somehow more intuitive, but we must be cautious because these are not even the color names that we assign to the region of the spectrum to which each class is maximally sensitive. That single cone classes do not code the perception of single colors is proof that the simple trichromatic theory cannot fully explain color perception.

It was once assumed that cones in the primate retina would be regularly distributed (to facilitate uniform sampling of wavelength), as is the case in the goldfish. Primate S cones are distributed fairly regularly (Curcio *et al*., 1991), but L and M cones are surprisingly patchy (Roorda and Williams, 1999) (Figure 2c). The resulting clumpiness may help us detect fine-grained luminance variations, but only at the cost of color resolution. Indeed, variations in color are harder to resolve than variations in luminance (for a review see Livingstone and Hubel, 1987). Two objects that differ only in color are described as equiluminant, and you can find examples of them in the Matisse painting (equiluminant colors will come out roughly the same gray in a gray-scale copy).

The very center of the fovea (0.1° of visual angle) is devoid of S cones. Our eyes are focused for about 550 nm light, where the L and M cones have their peak sensitivities. Consequently shorter-wavelength light will be blurred. Evolution may have selected against having S cones in the center of the fovea where high spatial acuity is the goal because the short-wavelength light to which the

S cones are most responsive will be an unreliable source of spatial information. The absence of S cones in the center of the fovea has surprisingly little impact on color vision, probably because the spatial extent of the S cone hole is finer than the coarse resolution of color vision.

The wavelength sensitivity of a given cone cell is attributed to the specific photopigment protein that it expresses (Nathans, 1999). The M and L photopigment genes, which are encoded on the X chromosome, are fairly similar in sequence, suggesting that the M and L photopigments arose from a common ancestral gene that duplicated not so long ago – around 30–40 million years ago, shortly after the continents of Africa and South America separated. The similarity of the M and L gene sequences predisposes them to recombination during meiosis. This has led to a polymorphism of the L and M photopigments, which is more commonly manifest in males because they only have one X chromosome on which to rely for their M and L photopigments. The polymorphism can be a complete loss of L cones (protanopia), loss of M cones (deuteranopia) or, more frequently, the expression of a mutant M/L hybrid. It is these polymorphisms that underlie the range of so-called red–green color blindness, the most famous case of which is that of Sir John Dalton (a deuteranope), who in 1794 was the first to describe the condition. The deletion of the S cone gene, on the seventh chromosome, is possible (tritanopia), but rare.

COLOR IS AN OPPONENT PROCESS

In 1880 the German psychologist Ewald Hering proposed that color was mediated not by trichromacy but by opponency. Hering observed that we cannot perceive a continuous mixture of colors as predicted by the trichromatic theory – we cannot perceive (or even conceive of) reddish-greens or bluish-yellows. Some colors are mutually exclusive of others. So, Hering argued, color must be determined by the activity of opponent mechanisms, and he proposed three: a red–green mechanism, a blue–yellow mechanism and a black–white mechanism. Today we can appreciate another reason why color must be an opponent process: the L and M cone fundamentals are so similar that to perceive long-wavelength light as distinct from middle-wavelength light (i.e. red and not yellow) the responses of the M cone must be subtracted from those of the L cone.

Each opponent mechanism can be thought of as one axis in a three-dimensional space that encompasses all colors. So any given color could be uniquely defined by three variables: the activity along the red–green axis, the activity along the blue–yellow axis and the activity along the black–white axis. The scientific dispute between Hering and Helmholtz and their supporters is the source of much animosity in the field of color, even today, although studies have now reconciled them.

The retinal ganglion cells, which receive input from the cones, project to neurons in the lateral geniculate nucleus (LGN). These in turn project to neurons in the primary visual cortex (Figure 3). The most common retinal ganglion cells are the midget cells and the parasol cells. Midget cells project to type I cells in the four parvocellular layers of the LGN. Parasol cells project to broadband cells in the two magnocellular layers. Most type I cells receive antagonistic inputs from M and L cones and therefore respond in opposite ways to different colors (DeValois *et al*., 1958; Wiesel and Hubel, 1966; Reid and Shapley, 1992). A type I cell may be excited by long-wavelength light and suppressed by middle-wavelength light. These cells represent the sort of building block for Hering's red–green opponent process; the three cone types represent Young and Helmholtz's trichromacy.

A third type of retinal ganglion cell is the bistratified cell (Dacey and Lee, 1994). Bistratified cells are excited by S cones and suppressed by a mixture of L and M cones, making them likely candidates for Hering's blue–yellow mechanism. Bistratified cells project to the (S versus M + L) type II cells in the koniocellular layers of the LGN (Figure 3).

Black–white cells must contribute to our perception of color. For example, the addition of black changes the color of orange to brown. Many parvocellular cells do not show cone opponency, and could therefore represent black–white, but it is unclear whether these or the broadband cells of the magnocellular layers (or an unidentified class of cells) underlie the black–white color axis (Wiesel and Hubel, 1966).

COLOR CONSTANCY

Perhaps the greatest misperception about color is that it is equated to wavelength. This misperception is cultivated early in our education when we are taught (incorrectly!) that long-wavelength light is 'red' and short-wavelength light is 'blue'. Though wavelength is the critical determinant of color, it is not the only determinant. For example, our perception of white depends on responses from all three cone classes, which is normally achieved when we see broadband light. However, after viewing a colored surface, one class of cones

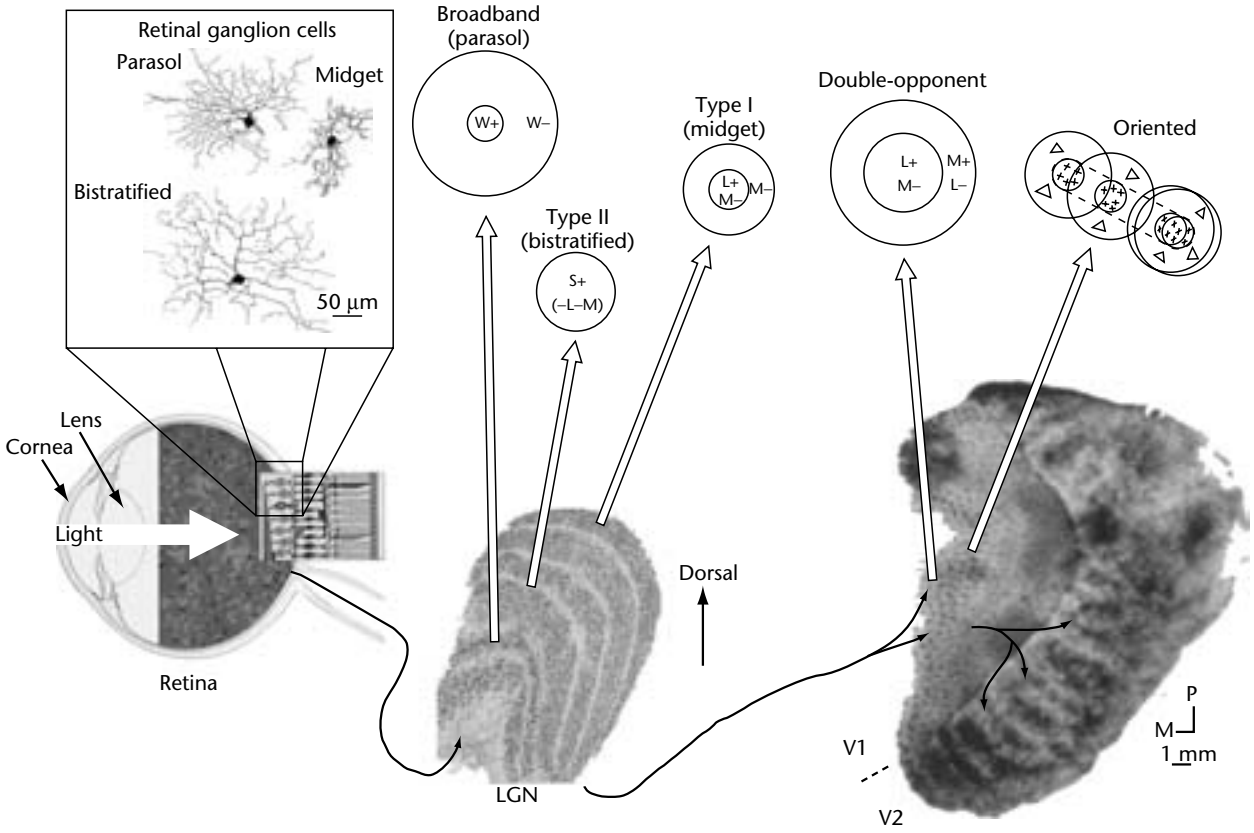
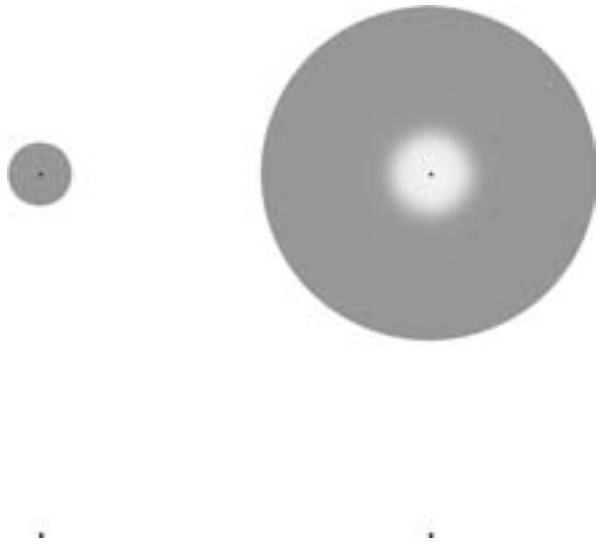


Figure 3. [*Figure is also reproduced in color section.*] A summary of color processing in the visual system. Light enters the eye and is focused on the retina by the cornea and lens. The three classes of cones respond to the light. Different retinal ganglion cells (inset; adapted from Dacey and Lee, 1994) sample the cone mosaic and provide input to the lateral geniculate nucleus (LGN). The retinal ganglion cell names 'midget' and 'parasol' reflect the relative sizes of their dendritic fields, which in turn reflect the relative sizes of their receptive fields. The cells of the LGN, here stained with Nissl substance, comprise six well-defined layers: four dorsal (or parvocellular) layers and two more darkly staining ventral (or magnocellular) layers. Each purple dot is a single cell, about 10 µm in diameter. The parvocellular layers contain type I cells; the receptive field of an L-ON center/M-OFF surround type I cell is given. The magnocellular layers contain the broadband cells. Between the darkly staining parvocellular and magnocellular layers are the koniocellular layers. Type II cells reside in these layers. Broadband cells, type II cells and type I cells are the LGN targets of parasol, bistratified and midget ganglion cells, respectively.

Neurons in the LGN send their axons to the primary visual cortex (V1). In this figure, V1 is represented by a tangential section of one hemisphere of unfolded and flattened squirrel monkey cortex that has been stained with the metabolic enzyme cytochrome oxidase (M, midline; P, posterior). Cytochrome oxidase (CO) staining clearly demarcates the border between V1 and the second visual area, V2, and reveals CO blobs in V1 and the CO stripes in V2. Color information is processed by the double-opponent cells, which reside in the V1 blobs and send their axons to the thin CO stripes of V2 (arrows). Between the blobs are cells that are sensitive to the orientation of a visual stimulus.

may become fatigued, perhaps by bleaching of the photopigment, and a previously 'white' surface will appear as a colored afterimage (Figure 4a).

Chromatic context also affects our perception of color. This is well known by artists, who place red against a green background to make it redder. Another example where wavelengths do not correlate directly with color is the phenomenon of induced colors: a gray spot can be made to appear colored if it is surrounded by a large colored annulus (the larger the annulus and the less sharp the boundaries, the stronger the effect). In fact spatial context even colors afterimages (Figure 4b). The discrepancies between physical cues and our perceptions can leave us confused. However, as Edwin Land pointed out, one should avoid asking the question, 'What color is it really?' as if to imply that our visual systems are deceiving us. Color is a product of our physiology and its interaction with the physical world; visual illusions simply point out what



(a) (b)

Figure 4. [*Figure is also reproduced in color section.*] There is more to color than meets the eye! Stare at the fixation dot in the middle of the green disk (a), being careful to hold your gaze steady. After 20 s or so, transfer your gaze to the fixation dot below; you should see a reddish afterimage. Now consider the small, fuzzy gray disk in the center of the colored annulus (b). After prolonged viewing the gray seems to adopt a weak reddish tinge. Such induced colors are much more striking when the colored annulus occupies the entire visual field surrounding the central gray spot. Try generating an afterimage to the gray spot. The afterimage to the gray spot is surprisingly green! This shows that the spatial configuration of a scene affects both the color of perceived images and the color of afterimages.

our visual systems are constantly (and usually effectively) doing.

An object will appear colored if it selectively absorbs some wavelengths and reflects others. The spectral distribution of reflected light is a product of the absorptive properties of the object's surface and the spectral properties of the light source (the illuminant). So if the illuminant changes, the reflected light will change too. Illuminants are constantly changing. A bright sunny day, under a blue sky, will contain a large proportion of shorter wavelengths, while a tungsten light bulb will produce longer wavelengths. The paradox of color vision is that despite these different illumination conditions, and the resulting difference in reflected light, the color of objects is fairly constant. A red apple is red, for example. It is not that a red apple is red only when viewed under a certain illumination condition such as a blue sky. This color constancy is mostly a property of our visual systems and not a function of memory.

It is easy to see why our visual systems have evolved in this way. If we were to assign a color to an object based solely on the light reflected from it then we would assign different colors to the same object depending on the conditions under which the object was viewed. Color constancy means that colors are properties of objects (which are constant) and not viewing conditions (which are continually changing).

Edwin Land, the inventor of instant photography, went to great lengths to reiterate that the color we assign an object is largely independent of the spectral content of the illuminant but is correlated with the absorption properties of the object or surface (Land, 1977). He was prompted by the familiar problem faced by color photographers: a scene photographed under tungsten light comes out with a reddish cast, and one photographed outside on a sunny day with a bluish cast. This is in contrast to our perceptions of the scene under the two illumination conditions – we see neither a reddish cast nor a bluish cast. Land concluded that our visual systems do not simply equate color and reflected wavelengths. In his experiments, Land used different light sources to illuminate different patches of a colored 'Mondrian' display, and varied the spectral content of his illuminants. He was able to show that two differently pigmented patches could be made to reflect the same spectral distribution and yet, remarkably, the patches still appeared as different colors. He also showed that an identical surface could be made to reflect a different spectral distribution and yet appear the same color.

The puzzle remained. How could the visual system achieve different color judgments for two areas if the light from the two areas were the same? Land devised the retinex algorithm, which is capable of determining illuminant-independent colors (Land, 1977). According to this algorithm, the critical determinant of the color of a surface is the chromatic context in which the surface appears. This might sound like a reiteration of what artists already knew empirically, but it went further. It provided a testable hypothesis about the visual system. It claimed that color is determined by abrupt changes in the relative cone activities across a scene. For example, retinex would identify a region as 'red' only when the long wavelength light reflected from it is surrounded by regions reflecting shorter wavelengths. Thus we would not expect to 'see' the reddish cast of a tungsten light because the cast is diffuse.

DOUBLE-OPPONENT COLOR CELLS

The cone-opponent retinal ganglion and LGN cells could subserve wavelength discrimination. However, color is not simply wavelength discrimination (see above). Rather, color is achieved through a spatial comparison of wavelengths across an image. A cell having a receptive field fed by a single cone class in a spatially opponent fashion might be the building block for such a comparison: for example, a cell excited by L cones in one part of visual space but suppressed by L cones in an adjacent part of visual space (an L-ON center/L-OFF surround cell); but despite intensive searches, no retinal ganglion cells or LGN cells like this have been found. The cone-opponent type I cells (see Figure 3) have spatially opponent receptive fields, but the centers and surrounds are fed by different cone classes, and the opponency is in the wrong direction to subserve color constancy. So where in the primate visual system is such a comparison made?

In 1968, Nigel Daw showed that some cells in goldfish retina have receptive fields that are both chromatically and spatially opponent, and therefore capable of computing simultaneous color contrast (Daw, 1968). Computational studies have shown that such 'double-opponent' cells could subserve color constancy in primates: a single double-opponent cell exceeds the requirement of a spatial comparison for one cone class: it is a spatial comparison for two cone classes. A common type of double-opponent cell, for example, is L-ON center/L-OFF surround and M-OFF center/M-ON surround.

The existence of double-opponent cells in the primary visual cortex (V1) of primates has been controversial, but there is now a consensus that they do exist (Conway, 2001; Johnson *et al*., 2001) (Figure 5). Cortical cells that show simple chromatic opponency (such as LGN type II cells) also exist, although they probably do not represent a distinct cell class but rather the end of a continuum of cone-opponent cells that show very weak surrounds.

In addition to mediating spatial color contrast, double-opponent cells may also play an important part in temporal color contrast (a red spot is redder if preceded by a green spot) because they respond to both the onset and cessation of a stimulus (Cottaris and DeValois, 1998) and show stronger responses to sequences of oppositely colored stimuli, e.g. green and then red (Conway *et al*., 2002).

Cortical cone-opponent cells represent about 10% of the total population of cells in V1. Both red–green double-opponent cells – i.e. L versus M – and blue–yellow double-opponent cells – i.e. S versus (L + M) – are found, and these, with a class of opponent achromatic cells, could be the sole basis for color perception despite their relative scarcity, because color perception is coarse and would require many fewer cells than (say) form perception. Perhaps not by coincidence, color in color televisions requires only about 10% of the bandwidth.

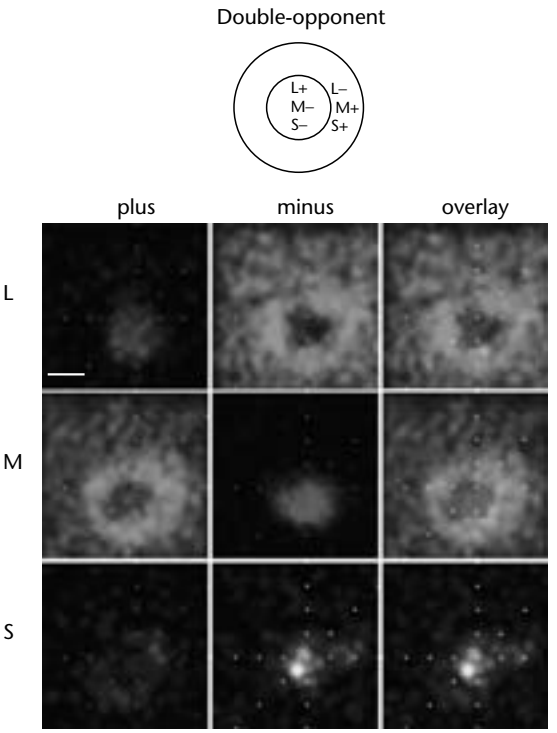


Figure 5. [*Figure is also reproduced in color section.*] The receptive field of a double-opponent cell in monkey V1. The left-hand column shows the spatial extent of the cell's response to increasing activity of the three cone classes (L, top; M, middle; S, bottom); the middle column shows the same cell's response to decreasing the activity of the three cone classes. Comparing the maps (right-hand column) shows that this cell's receptive field is both spatially and chromatically opponent. This double-opponent structure is critical to color constancy – our ability to determine an object's color despite changing illumination conditions. From Conway (2001).

Curiously, some red–green cells appear to receive S cone input. This needs to be studied further. It also remains to be shown how double-opponent cells are constructed from the LGN inputs and why there are more red–green double-opponent cells than blue–yellow ones.

Double-opponent cells reside in the cortex in clusters; moreover, these clusters are coarsely localized in metabolically distinct regions of cortex

called 'blobs' (Figure 3, bottom right) (Livingstone and Hubel, 1984). Blobs are easily identified in primate visual cortex by staining with the metabolic enzyme cytochrome oxidase. Why double-opponent cells are localized to the cytochrome oxidase blobs remains a mystery – perhaps these cells require more energy and therefore express higher levels of this metabolic enzyme. Surprisingly, other animals with poor color vision have cytochrome oxidase blobs (although the blobs in some of these mammals, such as cats, are not as prominent). Thus, it may be that the blobs represent regions of cortex dedicated to a more generic function, like parsing surfaces, of which color is only one component.

Nevertheless, the segregation of color cells in the LGN (in the parvocellular and koniocellular layers) and in the primary visual cortex (in the cytochrome oxidase blobs) shows that color is largely processed separately from other visual attributes. This segregation of color processing is evident perceptually (see below) and was used to advantage by Matisse (see Figure 1).

COLOR IS PROCESSED SEPARATELY FROM FORM AND MOTION

Color signals carried by the blobs of V1 are relayed to V2 and then to higher visual areas. Like V1, V2 displays an interesting pattern of staining for the enzyme cytochrome oxidase; unlike V1, the staining consists of alternating thick and thin stripes separated by interstripes (see Figure 3). Cells residing in the blobs of V1 send their axons to the thin stripes of V2 (Livingstone and Hubel, 1984). Not surprisingly, cells in the thin stripes are more likely to be color selective than cells in the thick stripes. The color cells in the V2 thin stripes respond best to colored spots, but they do so over a larger area of visual space. They are not responsive to a large field of color that encompasses the entire region over which small spots are effective. Such 'complex' color cells may be useful in identifying color boundaries present anywhere within a large area.

Color signals carried by cells in V1 and V2 are relayed to subsequent areas where, presumably, color percepts are elaborated. The V2 thin stripes project to V4; the V2 thick stripes, on the other hand, project to the middle temporal area, an area specialized for analyzing motion. Many V4 neurons respond better to some wavelengths than to others (Zeki, 1983), suggesting they are involved in color vision. Their receptive fields are much larger than the receptive fields of V1 cells, suggesting they are involved in elaborating color constancy.

Extrastriate visual areas are better described in the macaque monkey than in the human, but it becomes difficult to compare the areas of humans and monkeys the further the areas are from V1. In humans, for example, an area (V8) situated in the inferotemporal cortex is specialized for computing color (Hadjikhani *et al.*, 1998); it is debated if monkeys have a homolog of this area. Perhaps V4 is the monkey equivalent of human V8. Neurons in V4 are color biased (Zeki, 1980) but they are also selective for other attributes of a stimulus, such as the stimulus orientation (Schein and Desimone, 1990), suggesting that V4 is not simply a color area. To complicate the matter further, unlike lesions of V8 in humans, partial lesions of V4 in monkeys have little effect on tasks requiring color vision. Perhaps V4 is involved in piecing together form and color information; conversely, V4 may actually be a complex of areas, one devoted to form processing and another to color processing. The increasing resolution of functional magnetic resolution imaging may soon make it possible to address these issues.

Certain people who have damage to V8 following a stroke show a profound loss of color perception. Remarkably, this acquired achromatopsia does not interfere with their perception of form and motion – further suggesting that color is processed separately from other visual attributes. Oliver Sacks described one such stroke patient who was an artist (Sacks, 1995). After the patient had lost his color vision, he made peculiar color choices in his paintings; but he was still able to represent luminance and shape, because the areas of his brain dedicated to processing those aspects of the visual world were unaffected by the stroke. Moreover, he had no loss of motion perception.

CONCLUSION

An observer would say that the color and the shape of an object are inextricably linked. If color and form are processed separately by the cortex, how do they then become bound? How would this binding be manifest in the brain? The binding might simply be found in the correlated activity of the two pathways: an orange ball would produce separate sensations of 'round' in the form pathway and 'orange' in the color pathway, but the sensations would be elicited simultaneously. The reliability of the simultaneous activation of the two pathways, possibly reinforced by neural connections joining separate areas, could be enough to bind the 'round' ball with its 'orange' color. But

how to test this hypothesis, or any hypothesis for binding for that matter, is challenging.

Parallel processing is an efficient means of computing information because it is fast: multiple aspects of a scene can be processed simultaneously. Although many lines of evidence support a parallel processing model for visual perception (Livingstone and Hubel, 1988), some anatomical and physiological studies show that the visual system does not operate according to such a simple model. There are cells in the superficial layers of V1 (not confined to blobs) that are both selective for stimulus orientation and also more responsive to some wavelengths than others. What contribution do such 'oriented color' cells make to color perception? What are the LGN inputs to red–green double-opponent cells? Do the same LGN cells provide input to sharply orientation-tuned cells? How is the activity of the three chromatic axes in V1 integrated to bring about the perception of specific hues, and how is hue then bound with form? Perhaps most compelling, how do colors bring about emotional responses? These and many more questions will keep the field of color vision research alive for many years to come.

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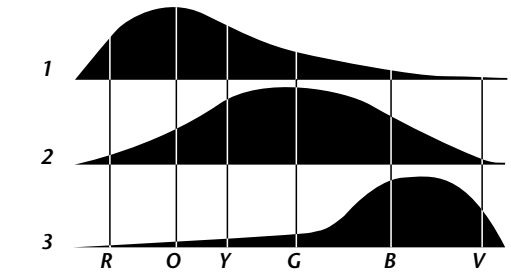
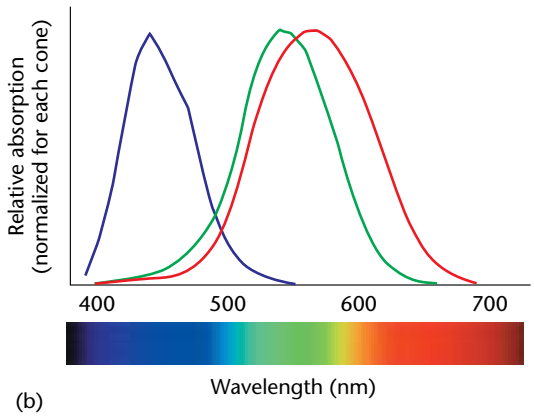
(a)

(b)

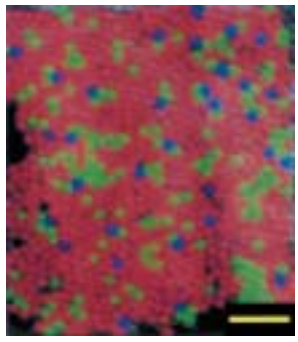
Plate 12 [Color Vision, Neural Basis of] (a) Henri Matisse, *Femme au Chapeau* (Paris, autumn 1905). Oil on canvas, 80.5 cm × 60 cm; San Francisco Museum of Modern Art (bequest of Elise S. Haas). The gray-scale reproduction (b) shows that the surprising color transitions do not interfere with an accurate representation of the woman's face. Yet the color reproduction is obviously more appealing – why? The dissociation of color and form, clear in this picture, shows that color is processed by the visual system separately from other stimulus attributes, like form.



(a)



(b)



(c)

Plate 13 [Color Vision, Neural Basis of] Color perception begins in the retina of the eye with three classes of photoreceptors called cones. (a) The absorption spectra of the three detectors proposed by Helmholtz in 1866: shorter wavelengths (V, or violet) were represented on the right. (b) The actual cone absorption spectra of the three cone classes, L, M and S, based on the cone fundamentals of Smith and Pokorny (1972). Convention today puts shorter wavelengths on the left. Below the plot is the visible spectrum. (c) The cone mosaic of a patch of living human retina made visible with adaptive optics, from Roorda and Williams (1999). The S cones are represented by blue, M by green and L by red. Scale bar, 5 arc minutes of visual angle.

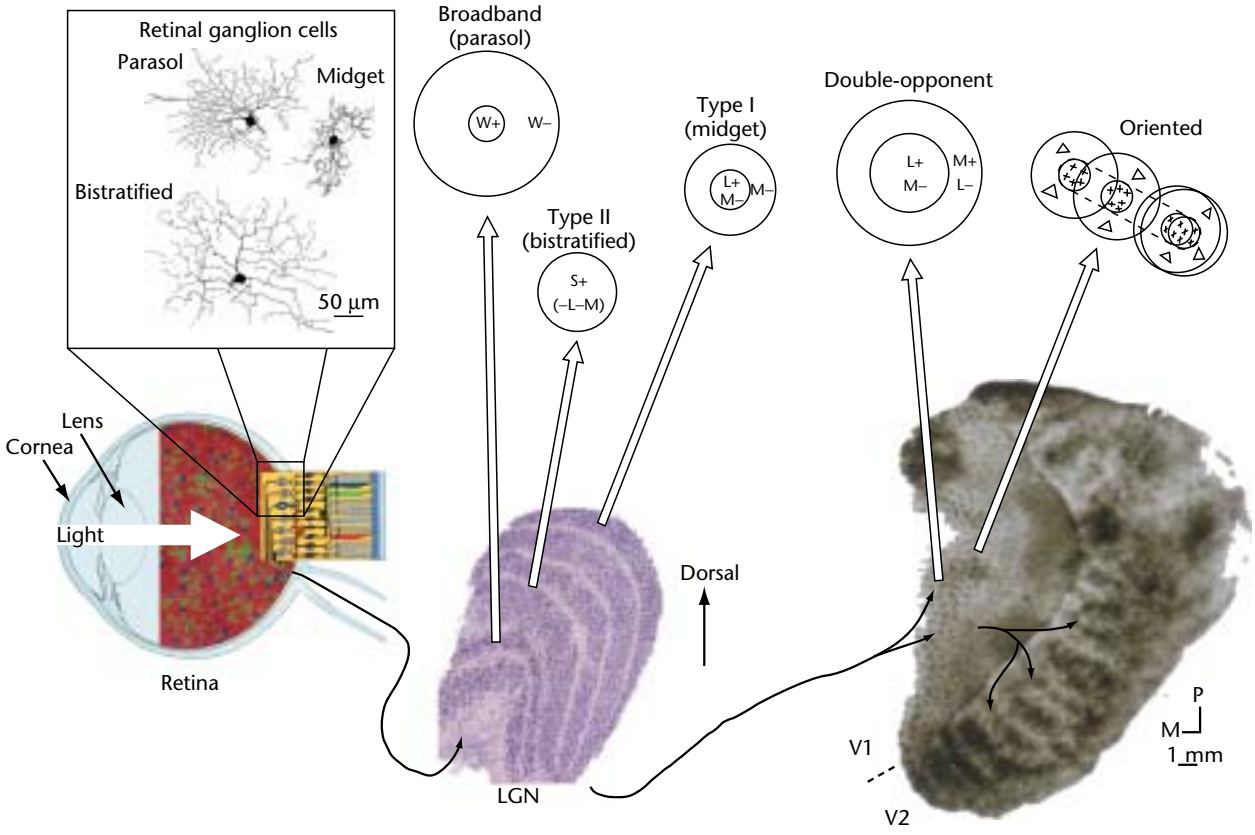
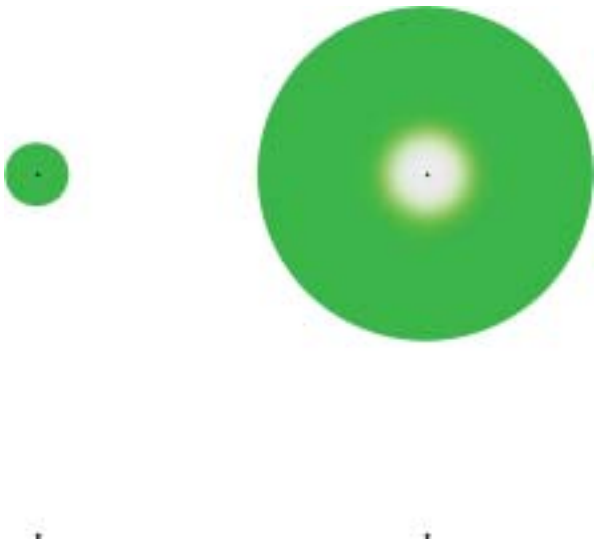


Plate 14 [Color Vision, Neural Basis of] A summary of color processing in the visual system. Light enters the eye and is focused on the retina by the cornea and lens. The three classes of cones respond to the light. Different retinal ganglion cells (inset; adapted from Dacey and Lee, 1994) sample the cone mosaic and provide input to the lateral geniculate nucleus (LGN). The retinal ganglion cell names 'midget' and 'parasol' reflect the relative sizes of their dendritic fields, which in turn reflect the relative sizes of their receptive fields. The cells of the LGN, here stained with Nissl substance, comprise six well-defined layers: four dorsal (or parvocellular) layers and two more darkly staining ventral (or magnocellular) layers. Each purple dot is a single cell, about 10 μm in diameter. The parvocellular layers contain type I cells; the receptive field of an L-ON center/M-OFF surround type I cell is given. The magnocellular layers contain the broadband cells. Between the darkly staining parvocellular and magnocellular layers are the koniocellular layers. Type II cells reside in these layers. Broadband cells, type II cells and type I cells are the LGN targets of parasol, bistratified and midget ganglion cells, respectively.

Neurons in the LGN send their axons to the primary visual cortex (V1). In this figure, V1 is represented by a tangential section of one hemisphere of unfolded and flattened squirrel monkey cortex that has been stained with the metabolic enzyme cytochrome oxidase (M, midline; P, posterior). Cytochrome oxidase (CO) staining clearly demarcates the border between V1 and the second visual area, V2, and reveals CO blobs in V1 and the CO stripes in V2. Color information is processed by the double-opponent cells, which reside in the V1 blobs and send their axons to the thin CO stripes of V2 (arrows). Between the blobs are cells that are sensitive to the orientation of a visual stimulus.



(a) (b)

Plate 15 [Color Vision, Neural Basis of] There is more to color than meets the eye! Stare at the fixation dot in the middle of the green disk (a), being careful to hold your gaze steady. After 20 s or so, transfer your gaze to the fixation dot below; you should see a reddish afterimage. Now consider the small, fuzzy gray disk in the center of the colored annulus (b). After prolonged viewing the gray seems to adopt a weak reddish tinge. Such induced colors are much more striking when the colored annulus occupies the entire visual field surrounding the central gray spot. Try generating an afterimage to the gray spot. The afterimage to the gray spot is surprisingly green! This shows that the spatial configuration of a scene affects both the color of perceived images and the color of afterimages.

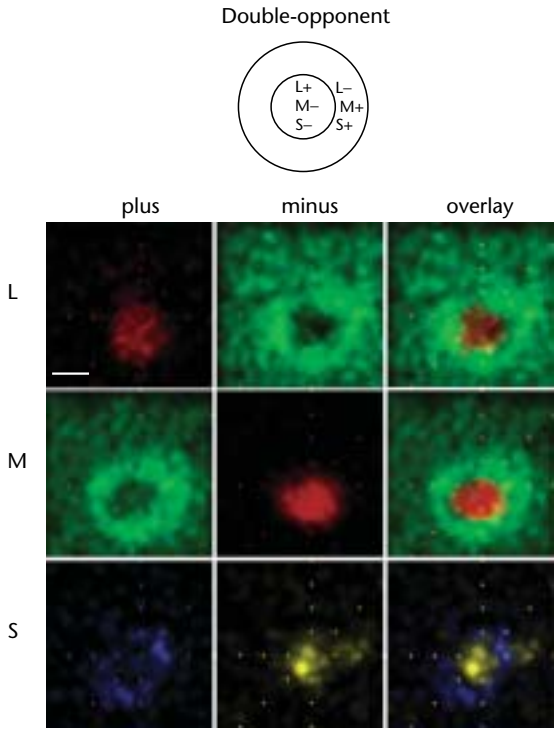


Plate 16 [Color Vision, Neural Basis of] The receptive field of a double-opponent cell in monkey V1. The left-hand column shows the spatial extent of the cell's response to increasing activity of the three cone classes (L, top; M, middle; S, bottom); the middle column shows the same cell's response to decreasing the activity of the three cone classes. Comparing the maps (right-hand column) shows that this cell's receptive field is both spatially and chromatically opponent. This double-opponent structure is critical to color constancy – our ability to determine an object's color despite changing illumination conditions. From Conway (2001).