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Physiology of Color Vision in Primates

Robert Shapley

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Summary and Keywords

Color perception in macaque monkeys and humans depends on the visually evoked activity in three cone photoreceptors and on neuronal post-processing of cone signals. Neuronal post-processing of cone signals occurs in two stages in the pathway from retina to the primary visual cortex. The first stage, in in P (midget) ganglion cells in the retina, is a single-opponent subtractive comparison of the cone signals. The single-opponent computation is then sent to neurons in the Parvocellular layers of the Lateral Geniculate Nucleus (LGN), the main visual nucleus of the thalamus. The second stage of processing of color-related signals is in the primary visual cortex, V1, where multiple comparisons of the single-opponent signals are made. The diversity of neuronal interactions in V1cortex causes the cortical color cells to be subdivided into classes of single-opponent cells and double-opponent cells. Double-opponent cells have visual properties that can be used to explain most of the phenomenology of color perception of surface colors; they respond best to color edges and spatial patterns of color. Single opponent cells, in retina, LGN, and V1, respond to color modulation over their receptive fields and respond best to color modulation over a large area in the visual field.

Keywords: color, cone photoreceptors, retina, visual cortex, single-opponent cells, double-opponent cells

This article is about the neuronal mechanisms for color perception. It focuses on the retino-cortical pathway in primates and more specifically Old World primates such as macaque monkeys and humans. It is even more focused on how signals from different cone photoreceptors are combined by post-receptoral neurons to produce responses related to color. Before considering neuronal mechanisms, we must review color phenomenology—that is, the experience of seeing color—in order to understand what the neuronal mechanisms have to explain.

Color Phenomenology

Color is a vivid part of our visual experiences, from blushing skin to green valleys. Philosophers believe that color is an objective material property (Hyman, 2006) not a subjective experience. However, the philosophers' conclusion is arguable. Those who study visual perception know that surrounding colors have a great influence on the perception of the color of a target region (Katz, 1935; Brainard, 2004), a fact that implies that color is not simply a property of objects but also of the brain's attempt at computing the surface properties of visual objects.

It is generally believed that when the brain does its computations that lead to color perception, it is most influenced by large regions of color or by color patterns of low spatial frequency. In other words, color perception is supposed to be a spatial integrator. The reason for this belief is the powerful influence of the results in the classic paper Mullen (1985). Mullen measured sensitivity (1/detection threshold) for sinusoidal, colored grating patterns that were equal in luminance (equiluminant) with the background. The gratings in Mullen's study were defined only by color contrast. Best detection was at the lowest spatial frequency for both red-green and blue-yellow grating patterns. That is, color detection was low pass. There are many psychophysical studies that support the concept of low-pass color perception. For example, Poirson and Wandell (1993, 1996) fit large datasets they collected on color appearance and color detection with two low-pass color mechanisms, one for red-green and one for blue-yellow, and one band-pass luminance mechanism. Another example from another highly respected group of color psychophysicists: Gowdy et al. (1999) reported "two sides of the chromatic split field are detected essentially independently by red or green 'blob' detectors." In a commentary to a book chapter, the distinguished vision scientist Hans Irtel wrote, "It is well-known that there is a fundamental difference between the achromatic and chromatic system in the spatial domain: the chromatic system behaves like a spatial low-pass filter while the achromatic system behaves like a spatial band pass" (Irtel, 2003).

However, several psychophysical studies about spatial context's effect on color perception (Katz, 1935; Brainard, 2004) implied that supra-threshold color perception was not a spatial integrator. Rather, color perception depended on the edges of a colored target region. Initially evidence came from studies of the influence of form on color perception. One example is filling-in of color in stabilized images across long distances from an unstabilized boundary (Yarbus, 1967). Color filling-in in the periphery of the visual field can be seen without image stabilization, with voluntary fixation (Krauskopf, 1963). These perceptual results suggest that the color appearance of a region may be more dependent on color contrast at the boundary of the region than it is on the spatially integrated spectral reflectance of the region's interior.

Color perception's dependence on edge color contrast is further suggested by the Chevreul illusion in color, studied by Daw (1964). The figure in the left-hand panel of Figure 1 is an example of a color-Chevreul illusion where the concentric, uniformly colored regions (in which the colors run from green to orange-red) appear to be shaded in color. The color of each circular ribbon (or annulus) appears redder near the outer boundary and greener near the inner boundary. On the right, the color edges between regions have been outlined with thin black circular lines, and the Chevreul illusion, the color shading and the heightened color saturation, disappears. Such phenomena indicate the importance of color edges to color perception. The color-Chevreul illusion result is just one of many examples that suggest that ordinary color perception (i.e., supra-threshold color appearance) is governed by neural mechanisms in the cerebral cortex that are color-sensitive and edge-sensitive (Friedman, Zhou, & von der Heydt, 2003).

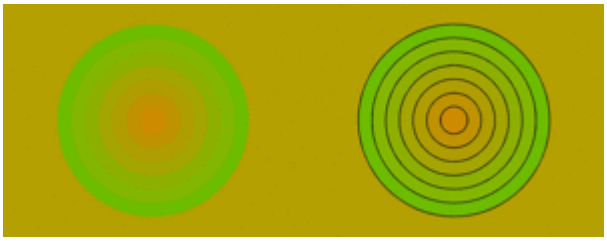

Figure 1. Color Chevreul illusion. Each colored region in the figure is uniform in its wavelength spectrum across its entire extent, but different signs of color contrast at inner and outer boundaries cause the appearance of color shading. The physical situation is revealed on the right where black outlines at the edges between the colored regions destroy the color-edge effects.

The strong influence of surrounding regions on the color of a target region has been known for a long time (Mollon, 2006). Color and form interact through the contrast that surrounding forms exert on the target colored region. Color contrast effects are assumed to be involved in the experience of color constancy (Brainard, 2004). It has been suggested that the neural substrate for color constancy is the population of orientation-selective double-opponent neurons in primary visual cortex, V1 (Gegenfurtner, 2003). Previous research suggests that V1 plays an important role in color induction caused by color contrast. For instance, the color tuning curve of single neurons in macaque monkey V1 was changed on different color backgrounds (Wachtler, Sejnowski, & Albright, 2003). The tuning curve shifted away from the background color. Wachtler and colleagues compared their electrophysiological measurements with human psychophysics and showed that the shift in the neuronal tuning curves of V1 neurons was correlated with the perceptual shift caused by chromatic induction reported by human subjects.

One might ask, what is the reason that color appearance is so dependent on the colors of the near surroundings? A functional justification is that the brain constructs a color signal to try to recover the reflective properties of surfaces. To accomplish this task, the neural mechanisms of color perception need to make comparative computations—local subtractions across object boundaries—that take into account the spatial layout of the scene as well as the spectral reflectances of target surfaces (Brainard, 2004; Shevell &

Kingdom, 2008). There may also be a separate neural mechanism to estimate illumination as well as surface color. Many investigators believe that V1 cortex plays an important role in perceiving surface color and illumination (Johnson, Hawken, & Shapley, 2001, 2008; Friedman et al., 2003; Wachtler et al., 2003, among others).

Another line of work implied that neural mechanisms that respond to color are not simply spatial integrators of visual signals: research on the influence of color on perception of spatial form. Research on this topic produced evidence for orientation-sensitive, spatial-frequency-selective color-responsive cells. For instance, orientation-dependent masking and spatial-frequency-dependent masking of equiluminant red-green grating patterns, and also cross-masking between equiluminant red-green grating patterns and black-white luminance gratings (Switkes, Bradley, & De Valois, 1988; Losada & Mullen, 1994) all occur. The masking results suggest that there are neural mechanisms of color detection that are selective for spatial frequency and orientation, while cross-masking suggests that luminance signals and color signals interact perceptually. Moreover, orientation discrimination was almost as precise with a red-green equiluminant pattern as with a luminance pattern (Webster, De Valois, & Switkes, 1990; Beaudot & Mullen, 2005). Furthermore, the tilt illusion was strong for equiluminant red-green as well as luminance patterns (Clifford, Spehar, Solomon, & Zaidi, 2003). Recent human fMRI results on V1 cortex have indicated the existence of a large population of orientation-tuned, color-responsive neurons in V1 (Engel, 2005; Sumner, Anderson, Sylvester, & Haynes, 2008).

Physiology of Color in the Retino-Thalamic-Cortical Pathway of Monkeys and Humans

The visual system of the macaque monkey resembles the human system in structure and function from the retina through to V1. This article, like most other studies of color neurophysiology, uses macaque neurophysiological data to understand human perceptual phenomena. Such an approach depends on the strong assumption that the visual pathways in humans and monkeys function in a similar way. The work of Russell De Valois and his colleagues (De Valois et al., 1974A, 1974B) supports the strong assumption (supported also by the more recent work of Hass & Horwitz, 2013). De Valois and colleagues showed that detailed behavioral measurements of spectral sensitivity, wavelength discrimination, and contrast sensitivity in Old World monkeys (e.g., rhesus or cynomolgus monkeys) resemble those in humans. Also, the neuroanatomy of the human retinocortical pathway is similar to that of Old World monkeys. Most data reviewed here were obtained in experiments on macaque monkeys, but human data will be used also when available. The scope of this article encompasses cones to V1 cells, as well as the relation of neural responses in the retino-cortical pathway to color perception. There are many interesting studies of color responses in extra-striate cortex reviewed elsewhere

(Shapley & Hawken, 2011; Kiper & Gegenfurtner, 2014). They are beyond the scope of this article.

Cone Photoreceptors

Spectral Sensitivity

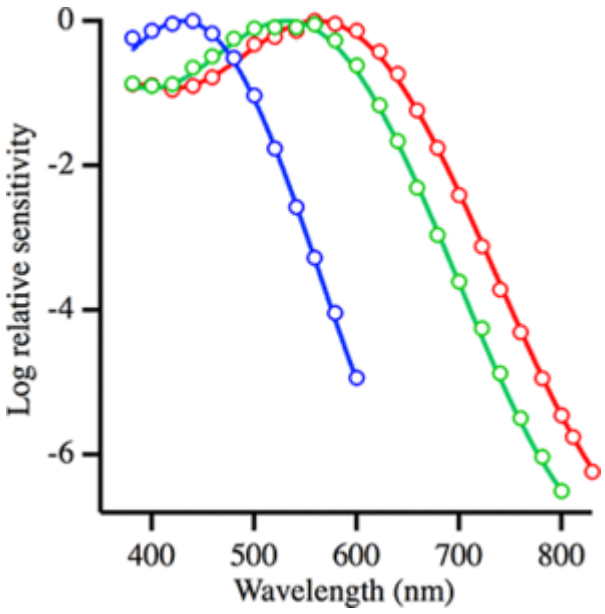

Figure 2. Cone spectral sensitivities. Average normalized spectral sensitivities versus wavelength of cones from Macaca fascicularis monkeys. S-cones are drawn in blue; M-cones in green; L-cones in red.

Data from Baylor, Nunn, and Schnapf (1987) re-drawn by Dr. Julie Schnapf.

As Old World primates, macaques have trichromatic vision based on cone photoreceptors with wavelength maxima near 430 nm (S cone), 531 nm (M cone), and 561 nm (L cone). The cones' spectral sensitivities were determined in experiments that employed suction electrodes to measure cone photocurrent directly (Baylor, Nunn, & Schnapf, 1987). Human cones studied in the same way behaved just like the monkey cones (Schnapf, Kraft, & Baylor, 1987). The spectral sensitivities of macaque cones are graphed in Figure 2, redrawn from the data in Baylor et al. (1987). One can observe the great overlap of M and L cones and the separation between S and both M and L cones. These direct measurements of photoreceptor spectral sensitivities are in generally good agreement with microspectrophotometric measurements of cone-pigment absorption spectra (Bowmaker & Dartnall, 1980; Bowmaker et al., 1980). The macaque cone data show no evidence for sub-types of S, M, or L cones but human psychophysical data on color matching suggested there are subtypes of L cones with slightly different peak wavelengths, 556 nm vs. 559 nm (Neitz & Jacobs, 1986). There seems to be much less diversity in the spectral peaks of human M cones than in L cones, as in the macaque data on M cones (Baylor et al., 1987). The genetics and spectral sensitivities of human S, M,

and L cones have been explained and discussed fully in the expert review by Neitz and Neitz (2011).

Univariance and Silent Substitution

One important property of cone photoreceptors is that each individual cone is color blind. Another way of expressing this thought is that the response of cones follows the principle of univariance (Naka & Rushton, 1966). Univariance means that the cone's response depends only on the quantum catch, not on the wavelength of the quantum caught. What univariance means in practice is that one can produce the same response in, for example, an L cone with two different colored lights. Say we use two monochromatic lights with wavelengths 500 nm and 659 nm that appear to us blue-green and deep red, respectively. The L cone's response will be the same for 500 nm and 659 nm as long as one adjusts the intensities so that the quantal catch is the same for both (Baylor et al., 1987). The univariance of cones is the basis of the technique of cone-isolation, or silent substitution (Forbes, Burleigh, & Neyland, 1955; Estevez & Spekreijse, 1982), which we and others exploited for studying color-signal-processing in retinal ganglion cells, LGN cells, and V1 cortical cells (Reid & Shapley, 1992, 2002; Johnson, Hawken, & Shapley, 2001, 2004, 2008; Lee, Kremers, & Yeh, 1998; Conway, 2001).

Retinal Ganglion Cells

Cone to Ganglion Cell Connectivity and Red-Green Color Specificity in P Ganglion Cells

Red-green color vision begins with the midget ganglion cells we previously termed P cells (Shapley & Perry, 1986) because of their connection to the Parvocellular layers of the LGN (Perry, Oehlcr, & Cowey, 1984). Near the fovea, each M or L cone provides input to one ON and one OFF midget bipolar cell, which in turn provide excitatory (sign-conserving) input respectively to ON and OFF P retinal ganglion cells. Again, near the fovea, each P ganglion cell receives direct input from a single midget bipolar cell (Calkins, Schein, Tsukamoto, & Sterling, 1994; Kolb & Dekorver, 1991). Consequently, near the fovea each of the P cell types receives direct (receptive field center) input from a single cone photoreceptor. The connectivity creates four receptive field types among the near foveal P cells: L ON-center, L OFF-center, M ON-center, and M OFF-center (Lee, Valberg, Tigwell, & Tryti, 1987).

The connectivity between cones and P cells varies with retinal eccentricity in two stages. Convergence from multiple midget bipolar to individual P ganglion cells begins at 10° eccentricity; by 30° to 40° eccentricity, a typical P cell gets convergent input from over 30 cone photoreceptors due to bipolar convergence (Lee, Martin, & Grünert, 2010). Also, eccentricity influences the convergence of cones onto midget bipolar cells. The one-to-one relationship between cones to midget bipolar cells persists to 40° eccentricity, beyond which some midget bipolar cells contact multiple cones (Wässle et al., 1994B). Thus, strict one-to-one, cone-to-ganglion cell connectivity is only possible for P cells within 10° of the fovea, and specificity of the cone connectivity to P cells deteriorates between 10°–40°. More specifics about the retinal connectivity of cones to P cells are given in the comprehensive review of Lee et al. (2010).

In central vision, 0°–10° from the fovea, P retinal ganglion cells behave visually like their cell targets in the Parvocellular LGN layers. They are color-opponent like the LGN cells (De Valois, 1960; DeMonasterio & Gouras, 1975). They have small receptive field centers driven by a single type of cone (DeMonasterio & Gouras, 1975). Also, there is evidence for cone specificity in wiring of P ganglion cells from results with cone-isolating stimuli (Lee et al., 1998; Martin et al., 2001; Lee, Shapley, Hawken, & Sun, 2012). It is most likely that all the detailed color properties of Parvocellular LGN neurons that are reviewed in the later section on the LGN are derived from the monosynaptic P-cell inputs to each Parvocellular neuron. This last assertion is based on data from recordings in the macaque LGN. Kaplan and Shapley (1984) were often able to record, simultaneously with recording LGN spikes, s-potentials known to represent the synaptic effect of each incoming afferent spike from retinal ganglion cells. Invariably in Parvocellular recordings,

the s-potentials were from a single P retinal ganglion cell, and they had the same color properties as the simultaneously recorded LGN spike.

Because the one-to-one cone-to-ganglion cell connectivity in midget system of the macaque retina does not persist unaltered beyond 10° retinal eccentricity, one expects (and finds) deterioration of color vision in the peripheral retina, though not complete loss of peripheral color perception (Gordon & Abramov, 1977). In a complete study of cone-opponency as a function of retinal eccentricity, Solomon et al. (2005) studied temporal-chromatic properties of P cells, M (Magnocellular-projecting) cells, and K (Koniocellular-projecting) cells across the macaque retina. Their major results for the purposes of this article were that P cells were much more sensitive to low-frequency chromatic modulation in central vision (0–15°) but that at eccentricities > 20°, P cells were relatively more sensitive to achromatic modulation. Many more P cells in peripheral retina (about 30%) were classified as non-opponent, that is peripheral P cell responses were as if driven by a signal derived from the sum of L-cone and M-cone signals (i.e., L+M). All of the central P cells that they found acted as if driven by L−M (like Parvocellular LGN neurons that represent central vision) at low temporal frequency of stimulus modulation. This finding, that central P cells are all cone-opponent, is quite important because it confirms prior results on macaque Parvocellular LGN—that almost all Parvocellular neurons representing central vision are cone-opponent (Derrington, Krauskopf, & Lennie, 1984; Reid & Shapley, 2002).

A later study indicated the lack of specificity in cone-to-ganglion-cell connectivity in P ganglion cells of the peripheral macaque retina (Field et al., 2010). Field et al. (2010) provided direct evidence that the receptive field centers of peripheral P ganglion cells (eccentricity > 40°) often have mixed cone input. They also provided evidence that the receptive field surround often has mixed cone input in such peripheral P cells. While these results have been interpreted as evidence for mixed cone input to the surround of P cells in general, it seems to me that the results of Field et al. (2010) do not bear on the question of what is the cone input to receptive field mechanisms in the central retina. Rather, their results seem in generally good agreement with the findings of Solomon et al. (2005) indicating that color processing deteriorates in the peripheral retina.

Blue-Yellow Signals in the Retina and LGN

The color pathway that is found most often in most mammalian species is the neuronal channel that carries blue-yellow signals from eye to cortex (Casagrande, Yazar, Jones, & Ding, 2007; Jacobs, 2008). In primates, the Koniocellular pathway was proposed as the vehicle for blue-yellow signals to reach cortex (Martin et al., 1997; Hendry & Reid, 2000). Direct proof that S-(L+M) signals are carried by some of the Koniocellular cells was provided for the marmoset LGN by Martin et al. (1997) and for the macaque LGN by Roy et al. (2009).

There are two separate pathways for S-cone signals from the retina to LGN. S+ signals travel in the axons of Blue ON retinal ganglion cells. These are small bi-stratified ganglion cells that receive –(L+M) cone input in the upper (more distal) inner plexiform layer and S+ cone input in the lower inner plexiform layer (Dacey, Crook, & Packer, 2014). These bi-stratified ganglion cells project to a subset of Koniocellular LGN neurons. A separate (and sparser) group of midget ganglion cells receive L+ or M+ input and S- input and project to the LGN where they have been recorded, though rarely (Tailby, Solomon, & Lennie, 2008).

Lateral Geniculate Nucleus (LGN)

Parvocellular and Magnocellular Layers

Both humans and macaques have multilayered LGNs. In these species, the LGN is divided usually into six layers: four more dorsal, Parvocellular layers, and two more ventral Magnocellular layers (Polyak, 1957). Opponent color signals travel from retina in P cell axons to cortex through the Parvocellular neurons (De Valois, Abramov, & Jacobs, 1966; Wiesel & Hubel, 1966) and from K retinal ganglion cell axons to Koniocellular cells intercalated between the Parvo and Magno cell layers in the LGN (Hendry & Reid, 2000; Casagrande et al., 2007). Color opponent neurons in the Parvocellular layers take the difference between two opponent cone signals, for instance M-L, and therefore respond with opposite signs to different wavelengths (Derrington et al., 1984; Reid & Shapley, 1992, 2002). In this way, the Parvocellular cells simply follow their P-cell inputs from the retina (DeMonasterio & Gouras, 1975; Lee et al., 1998; Martin et al., 2001). Color-responsive Koniocellular cells receive S+ cone input and antagonistic input from a combination of M- and L-cone inputs. The color response properties of the various color-responsive neurons in the LGN have been studied in many ways. Here we focus on two main approaches that have been useful, namely (1) the study of how neuron responses are distributed in a specific color modulation space, DKL space, and (2) how the same populations of neurons respond to cone-isolating stimuli.

LGN Neurons

Modulation in Color Space

Chromatic opponency of LGN cells has been investigated using a technique similar to silent substitution, namely modulation in color space around a white point. This technique grew out of psychophysical investigations of chromatic opponent mechanisms.

The color space considered here is a re-mapping of cone excitation space. Any light source with any spectral distribution over the visible spectrum can be represented as a three-dimensional vector of cone excitations. The three coordinate axes in cone excitation space (or SML space) are S, M, L cone excitations by the light. Cone excitations can be calculated from the light source's energy spectrum and the spectral sensitivities of the cones (Baylor et al., 1987). Based on MacLeod and Boynton (1979), and Derrington et al. (1984) proposed a (linear) mapping of cone excitation space into a new color space (DKL space) that represented the degrees of activation of cone-opponent mechanisms rather than cones. The axes of DKL color space were luminance modulation, S-cone modulation (the so-called Constant R and G axis), and L-cone and M-cone modulation such that S-cone excitation was constant (the so-called Constant B axis). These axes would be preferred modulation directions for putative color-opponent mechanisms. Lights along the Constant B axis would stimulate only those cells that received +L−M or +M−L input, while the Constant R and G axis would isolate those lights that excited only cells that received excitation (or inhibition) from S cones. The Constant B and Constant R and G axes define a plane in DKL space, the Isoluminant Plane within which all lights have the same luminance as the white point. (Throughout the review of DKL space we have used the term "isoluminant" because that is the term used by Derrington et al., 1984. However, it is preferable to use the word "equiluminant" for the same concept. The remainder of the article will use that word instead.)

Krauskopf et al. (1982) demonstrated that the three axes of DKL space were preferred axes for habituation of the human perceptual response to chromatic flicker. Krauskopf et al. (1982) named these axes "cardinal directions of color space." It is important to note that the transformation from SML (cone activation) space to DKL (named after Derrington, Krauskopf, Lennie) space is a linear transformation, but angles are not preserved. Thus, the cone-isolating vectors that are orthogonal in SML space are not orthogonal in DKL space. The vectors for isolation of L and M cones are each about 45° from the S-cone vector in DKL space (Derrington et al., 1984). In DKL space, the L and M cone vectors are only 10 to 20° apart and are mapped close to the luminance axis (Derrington et al., 1984) (i.e., almost orthogonal to the isoluminant plane).

Derrington et al. (1984) used stimuli modulated along different vectors in DKL space to characterize macaque LGN neurons. Most of their experiments were done with large spots that covered the entire receptive fields of the LGN neurons. Such large stimuli were optimal for the LGN neurons studied.

Modulation in the isoluminant plane should have been ineffective in stimulating luminance neurons that would have a spectral sensitivity like that of the photopic luminosity function. Each type of color-responsive neuron should have a null plane, like the isoluminant plane for luminance units, within which color modulation should be ineffective. The elevation of this null plane with respect to the isoluminant plane is a measure of the degree to which the neuron's response is determined by opponent mechanisms. The closer to zero the elevation of the null plane, the more nearly the neuron's response is completely determined by the luminance mechanism. Since the cone

vectors are pointing so close to the luminance direction in DKL space, neurons that are being driven by one cone only, either the L or the M cone, will have a null plane near the isoluminant plane, with a low elevation. Cells with null planes with higher elevations were the color responsive Parvocellular, and the blue-sensitive Koniocellular, neurons. Color-responsive neurons fell into two groups based on the azimuths of their null planes: L/M neurons and S-driven neurons.

Derrington et al. (1984) used the position of the null planes in DKL space for each neuron to calculate cone-weighting factors for each neuron studied. Derrington et al. (1984) found a most important result: that virtually every Parvocellular neuron was color-opponent. At least two cone-weights were of opposite sign. This major result about the scope of cone-opponency was probably a consequence of the fact that their recordings were from the part of the LGN that represented central visual field (0–15°). A second crucial finding was that with the large-area stimuli they used, the ratio of L to M cone input in Parvocellular neurons clustered tightly around 1:−1 or −1:1 (Derrington et al., 1984). That is, the L and M cone weights were approximately balanced so that one could approximate the Parvocellular signals as being L−M or M−L, with unit coefficients as the cone weights. Because the cone-weights were such an important part of the interpretation of results on DKL space, later workers instead used direct methods of cone-isolation to study cone-weights of receptive field mechanisms in Parvocellular neurons.

Cone-Isolating Stimuli

Cone-opponent input to Parvocellular neurons has been studied extensively with cone-isolating stimuli (Forbes et al., 1955; Estevez & Spekreijse, 1982) in order to understand the spatial pattern of cone inputs (Reid & Shapley, 1992, 2002; Lee et al., 1998, 2012). Reid and Shapley (1992, 2002) used reverse correlation (Sutter, 1992; Reid & Shapley, 1992; Reid et al., 1997) to characterize the conventional receptive field for achromatic stimuli. From these achromatic measurements Reid and Shapley estimated the spatial location and extent of the receptive field center and surround. Then the sign and magnitude of the cone signals to the center and surround of the Parvocellular cell's receptive field were estimated from cone maps. This was done by correlation of neuron responses with cone-isolating patterns (Reid & Shapley, 1992, 2002).

The experimental data were used to test hypotheses about the specificity of cone inputs to receptive field center and surround. Excitation and inhibition in almost all red–green opponent Parvocellular neurons (within 13° of the fovea) were both cone-specific. Mixed cone input to the surround would be observable experimentally as an antagonistic center/surround relationship for a single cone class, specifically the cone class of the center. Most red-green opponent Parvocellular neurons had center/surround antagonistic responses to a luminance stimulus (thus they were type I cells according to the classification scheme of Wiesel & Hubel, 1966), but the spatial map of cone-isolated responses was monophasic, with a profile resembling a bell-shaped curve. These results are what one would predict if the cone inputs to each Parvocellular cell were sign-specific (Reid & Shapley, 2002). Across the population sampled, not all the cone inputs to the receptive field surrounds were completely specific, but almost all were. Reid and Shapley's results on specificity of cone inputs to the receptive field surround in Parvocellular neurons were consistent with the results of Lee et al. (1998) on responses of P retinal ganglion cells in finding mostly cone-specific inputs to the receptive field surround. They also were consistent with results on the spatial frequency responses to cone-isolating grating patterns (Lee et al., 2012). However, the results of Lee et al. (2012) about cone-specific surrounds obtained in central P ganglion cells and Parvocellular LGN cells are not in agreement with data obtained on peripheral P cells by Solomon et al. (2005), and Field et al. (2010), but the main difference is likely to be the different retinal eccentricities sampled. This conjecture is based in part on the results of Solomon et al. (2005) who reported that the specificity of cone weights to center and surround varied with retinal eccentricity, consistent with greater cone specificity nearer to the fovea.

Single-Opponent LGN Cells

Mixed or Cone-Specific Surrounds

The experimental data on the cone spatial receptive fields of Parvocellular neurons and their P cell inputs from the retina is summarized in the concept of the single-opponent cell. Parvocellular neurons are called single-opponent because there are two (opponent) receptive field mechanisms of opposite sign, each driven by a single type of cone, and

each cone input is of one sign (Reid & Shapley, 1992). The sensitivity profiles of an ideal single-opponent cell are plotted in Figure 3; the drawing there is for a Parvo cell that would be +M-L. There are also cells that are +L−M. Such single-opponent neurons will compute the color modulation of a local region compared to its local adaptation level, and as such could be useful in signaling the color of a spatially extended region. Lee et al. (2012) reported that single-opponent cells (both P retinal ganglion cells and Parvocellular LGN neurons) were spatially low-pass for equiluminant color grating patterns. This means that the best stimulus for P cells and their Parvocellular target neurons is color modulation of the full visual field—or in practice a large region that is large enough to cover completely the receptive field of the neuron. Parvocellular neurons that are low-pass for color gratings may or may not be spatially band-pass for luminance grating patterns (Solomon et al., 2005; Crook et al., 2011; Lee et al., 2012), depending on how much the opponent inputs from the opponent cones overlap in space. The LGN cells excited by S− all were low-pass for S-cone-isolating color stimuli (Tailby et al., 2008). That is, the S− (L+M)+ cells appear also to be of the single-opponent type. The S+ LGN cells studied by Tailby et al. (2008) were weakly bandpass on average. The population studies of spatial frequency tuning of Parvocellular and color-responsive Koniocellular cells indicate that most are single-opponent, which is an important result consistent with cone-isolation and color-space-modulation studies (Derrington et al., 1984; Reid & Shapley, 1992, 2002).

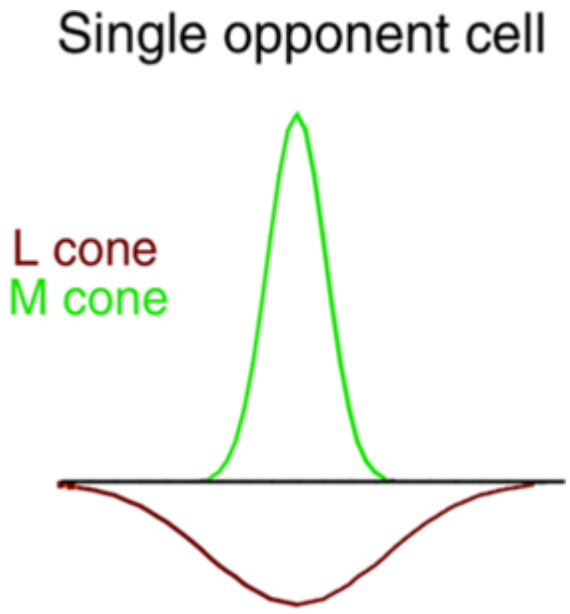

Figure 3. Diagram of the sensitivity profile of an idealized M+L− single-opponent cell.

The view of Parvocellular cells as single-opponent has excited much opposition, mainly based on theoretical calculations. Long ago it was proposed that mixed cone input to the receptive field surround in macaque P cells and Parvocellular LGN cells could generate a cone-opponent signal because the receptive field center was cone-specific: the so-called hit or miss hypothesis (Paulus & Kröger-Paulus, 1983; Shapley & Perry, 1986). Computer simulations (Lennie et al., 1991) provided quantitative support for the mixed surround hypothesis. However, direct physiological measurements of the cone input to the surround did not support the mixed surround hypothesis for P cells and Parvocellular cells with receptive fields not more than 0–15° from the center of gaze (Reid & Shapley,

1992, 2002; Lee et al., 1998, 2012). The cone-specificity of the surround appears to disappear in P cells and Parvocellular cells that have receptive fields in the peripheral retina (Solomon et al., 2005; Field et al., 2010).

The results of Crook Manoonkin, Packer, and Dacey (2011) provide support for the mixed surround hypothesis. Using almost identical stimulus methods to those used by Lee et al. (2012), they reported very different results for a population of P cells recorded at retinal eccentricities of 10–20°. Lee et al. (2012) found most spatial frequency responses to cone-isolating gratings to be low-pass, consistent with a cone-specific surround. However, Crook et al. (2011) reported that many P cells responded in a band-pass manner to cone-isolating gratings, a result that suggests that one cone provided input to the receptive field center and also antagonistic input to the receptive field surround (i.e., that the surround had mixed cone input). The reasons for the discrepancies between datasets have not been resolved. Lee et al. (2012) did experiments on anesthetized monkeys in vivo. They presented visual stimuli through the natural optics of the eye, at background luminances in the range of 1,000 trolands retinal illumination, and recorded ganglion cell activity extracellularly. Crook et al. (2011) worked on isolated retinas from excised eyes, and imaged stimulus patterns on the retinal surface with microscope lenses. They used high background illumination of ~100,000 trolands. They recorded ganglion cell activity with intracellular electrodes. It is possible that some of the many differences in experimental details led to the reported differences in data.

L/M Cone Balance

The first important result of the study by Derrington et al. (1984) was the prevalence of cone opponency in Parvocellular neurons as reviewed above. The second important result of Derrington et al. (1984) was the roughly equal and opposite L and M cone-weights in Parvocellular neurons in their responses to stimuli of large area that cover the receptive field. This rough balance of L and M cone input is likely to be inherited from each cell’s P cell input (though there is not a definitive study of central P retinal ganglion cells to parallel the Parvocellular LGN on this important point; it would be a study worth doing). Other studies have confirmed the fact of L/M cone balance on Parvocellular neurons (Reid & Shapley, 2002; Tailby et al., 2008; Lee et al., 2012). Thus, for stimuli of large area we can conceive of Parvocellular cells (those representing the central visual field, 0–15°) as sending signals to the cortex that indicate the strength of L-M or M-L cone signals.

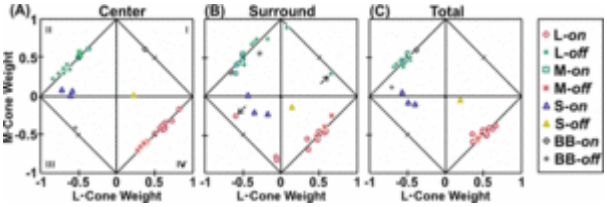

Figure 4. Cone weights of macaque LGN cells derived from experiments with cone-isolating stimuli. Scatter plots of normalized cone weights are plotted for all neurons recorded in Parvocellular layers. M-cone weights plotted on the ordinates versus L-cone weights on the abscissa. Points near the diagonal lines are from cells that received negligible S-cone input. Opponent L-cone versus M-cone responses are plotted in the second and fourth quadrants. Cone non-opponent (additive) responses are plotted in the first and third quadrants. The two Parvocellular cells that had mixed surrounds are indicated with arrows in (B). Putative Konicellular (S+) cells are plotted as blue triangles.

From Reid and Shapley (2002).

It is important to realize that roughly 1:–1 (or –1:1) L/M cone balance in Parvocellular LGN neurons only applies for stimuli that cover the entire receptive field. The cone-weight ratio is much more variable for stimuli that drive only the receptive field center or only the receptive field surround. This point is illustrated in Figure 4 from Reid and Shapley (2002). In that figure, there are three diamonds that are cone-weight plots of the kind introduced in Derrington et al. (1984). Most of the points represent cone-weights of Parvocellular cells. Also data are drawn for three S+ neurons and one S- neuron. Please notice in Figure 4A that the cone-weights for stimuli presented to the receptive field center were distributed widely for the different types of Parvocellular neuron (with L+ or L– centers or M+ or M– centers). For the same population of neurons in panel B, the cone-weights again were distributed widely for stimuli that only stimulated the receptive field surround. In the right-hand panel C are drawn the cone-weights for stimuli that covered the entire receptive field (labeled “Total”). Here the M:L cone-weights are highly clustered around 0.5: –0.5 or –0.5:0.5. What these cone-weight plots signify is that it is correct to say that Parvocellular cells carry a signal proportional to M−L or L−M when the stimulus is a large uniform field. But when the stimulus is not a large area but instead is a sharp visual edge, or a fine spatial grating pattern, the signals the Parvocellular cells are carrying are not proportional to M−L or L−M but rather to gL−M or hM−L where g and h are weighting coefficients that may vary widely (between 0–2 or more) from cell to cell as in Figure 4A. Thus, when one views a colored spatial pattern, the color signal from LGN to cortex is not simple and low-dimensional but complicated and high dimensional and needs to be decoded by the cortex (see Shapley & Hawken, 2011; Shapley et al., 2014).

Parvocellular LGN color signals are relayed to V1 in the Parvocellular-recipient layer 4Cβ, and from 4C to upper and lower cortical layers (Lund, 1988; Yoshioka, Levitt, & Lund, 1994; Callaway, 1998).

Koniocellular Cells in LGN

There are also neurons within the LGN intercalated between the main cell layers (Casagrande, 1994). Some of the intercalated cells carry signals derived from S+L– color-opponent ganglion cells (Hendry & Yoshioka, 1994; Martin et al., 1997; Hendry & Reid, 2000; Tailby et al., 2008). Some of the “blue-yellow” intercalated (or Koniocellular) neurons have direct input to cortical cells in the zone of layer 3B or 4A (Chatterjee & Callaway, 2003; Casagrande et al., 2007). Unlike Parvocellular and Koniocellular neurons, cells in the Magnocellular layers are to a first approximation “color blind.” Their receptive field centers sum signals from the L and M-cones (Lee et al., 1987; Reid & Shapley, 2002).

Color in V1 Cortex

How Important is Color in Primate V1 Cortex? Red-Green Cells

Many investigators have reported that there are many color-responsive neurons in V1 cortex. Victor et al. (1994), recording local field potentials (LFPs) in macaque V1 with multi-electrode probes, found LFP responses to equiluminant red-green modulation both in upper and lower cortical layers at most recording sites. Leventhal, Thompson, Liu, Zhou, and Ault (1995) found that a large fraction of cells in the upper layers of macaque V1 were color-sensitive. In our own studies, we estimated that about 40% of all macaque V1 cells were color-selective, but this percentage rose to 60% in layer 2/3 (Johnson et al., 2001, 2004, 2008). Friedman et al. (2003) found a similar percentage, 64%, of color-selective cells in layer 2/3 of macaque V1. Most of the color-responsive neurons respond to both color and luminance (Thorell et al., 1984; Lennie, Krauskopf, & Sclar, 1990; Johnson et al., 2001), and, as we found out later, they are almost all double-opponent (Johnson et al., 2004, 2008) in that they are more responsive to color patterns than to large fields of uniform color (Livingstone & Hubel, 1984).

Studies of human V1 with fMRI changed how scientists think about color processing in V1. Many fMRI results comparing color and luminance responses in human V1 indicated that color responses were comparable to or larger than achromatic responses. For instance, Engel, Zhang, and Wandell (1997) found that the strongest fMRI modulation in human V1 was caused by red-green modulation that evoked opposite-signed L and M cone responses. These results supported the concept that human V1 was strongly responsive for color, a result that was replicated often (Kleinschmidt, Lee, Requardt, & Frahm, 1996; McKeefry & Zeki, 1997; Hadjikhani et al., 1998; Beauchamp, Haxby, Jennings, & DeYoe, 1999; Brewer, Liu, Wade, & Wandell, 2005; Mullen et al., 2007; Wade, Augat, Logothetis, & Wandell, 2008).

Color Modules

The most widely publicized view about color in the cortex used to be that color, motion, and form were analyzed in parallel by separate visual cortical modules. The ideas behind the modular view originated with Hering and were transmitted by Hurvich and Jameson (1957). Krauskopf and colleagues adopted a modular approach in their work on cardinal directions in color space (Krauskopf et al., 1982; Derrington et al., 1984). The modular view was also thought to be supported by the shape of the contrast sensitivity function for red-green equiluminant patterns, which is low-pass, as opposed to the spatial band-pass contrast sensitivity function for luminance patterns (Mullen, 1985). Thus, it was thought that the distinct contrast sensitivity functions meant that color was being analyzed separately, in parallel to achromatic patterns.

Semir Zeki emphasized the modular view in his research on the functions of extra-striate visual cortex (Zeki, 1973, 1978A, 1978B). He reported that different extra-striate regions in the macaque monkey cortex were specialized for different visual features. For instance, V5 (called by others MT) cortex was reported to be comprised of neurons almost all of which were directionally selective. Thus, Zeki thought of V5 as the motion area, the region of the visual cortex designed to respond to motion in the visual scene. On the other hand, he believed that another extra-striate cortical area, V4, contained mainly neurons that were color-responsive.

Zeki's later work also supported the special role of V4 in color perception by analyzing the differences between V4 and V1 (Zeki, 1983A, 1983B) in responses to color patterns, comparing color responses in the monkey cortex with human color perception. In his experiments, wavelength distribution and perceived color were dissociated by surrounding context. Zeki (1983A, 1983B) reported that while a fraction of V1 cells responded to the wavelength distribution of a target in their receptive fields, only V4 cells responded to the color of the target perceived by a human observer. Based on these results and his earlier work on the functional specificity of anatomical areas, Zeki proposed that V4 was a color center in the monkey brain.

The famous studies of V1 and V2 cortex in monkeys by Livingstone and Hubel (1984, 1988; Hubel & Livingstone, 1987) supported Zeki's modular concept and linked it to parallel processing of color and form in the LGN. Livingstone and Hubel (1984, 1987, 1988) proposed a tripartite model in which signals from Parvocellular LGN were subdivided in V1 cortex into two separate streams, one for color processing, localized in the cytochrome oxidase (CO) patches or "blobs" in layers 2–3, (Wong-Riley, 1979; Horton & Hubel, 1981), and one for form processing in the interpatch regions of layers 2–3. Layers 2–3 are important as output layers of V1 to extra-striate visual areas.

Livingstone and Hubel also proposed that a specific cell-type found in layers 2–3 of V1 was important for color perception: the double-opponent cell. Such a hypothesized neuron is double-opponent because it adds up opposite-signed inputs from different cones (cone-opponency) and also opposite-signed inputs from different locations in the cell's

receptive field (spatial opponency). The defining characteristic of a double-opponent cell is that it is strongly responsive to color patterns but weakly or non-responsive to full-field color stimuli or shallow gradients of color. Subsequent work, reviewed in the next section, showed that a large fraction of color-responsive neurons in V1 are double-opponent but that their receptive field organization is different from what Livingstone and Hubel thought in the 1980s. But there is much experimental support for Livingstone and Hubel's proposal that double-opponent cells are important for color perception.

Integrated Color and Form: Single- and Double-Opponent Cells

An alternative to the modular view is the idea that neural signals about color are integrated with form-related neural signals in the cerebral cortex (Lennie, 1999; Shapley & Hawken, 2002, 2011; Gegenfurtner, 2003; Kiper & Gegenfurtner, 2014). Neurophysiological investigations of this problem were strongly influenced by color phenomenology, reviewed at the beginning of this article, about the links between color and form in perception.

It is important to state our working hypothesis, namely that V1 plays an important role in color perception through the combined activity of two kinds of color-sensitive cortical neurons, single- and double-opponent cells. The cortical single-opponent cells have a receptive field organization that is like that of the LGN single-opponent cells diagrammed in Figure 3, but usually the cortical single-opponent receptive fields are larger than those of the LGN cells, as deduced from their lower spatial frequency resolution for equiluminant color patterns (Johnson et al., 2001; Schluppeck & Engel, 2002). The visual properties of single-opponent receptive fields make cortical single-opponent cells responsive to color illumination that spreads widely in space, and changes only gradually across a scene.

The double-opponent cells comprise a large population of neurons in V1. These cells respond to color boundaries and color patterns much better than to large fields of color (Livingstone & Hubel, 1984; Thorell, De Valois, & Albrecht, 1984; Lennie et al., 1990; Johnson et al., 2001, 2004, 2008; Solomon & Lennie, 2005). A diagram of the sensitivity profile of a V1 double-opponent cell is given in Figure 5. Color perception of objects probably depends on signals that originate in the double-opponent cells. Color contrast is an aspect of perception that likely depends on double-opponent cells. Double-opponent cells in V1 are likely to be the neural substrate of the spatially-tuned chromatic channels that were revealed in psychophysical masking and adaptation experiments with equiluminant patterns (Switkes et al., 1988; Bradley et al., 1988; Losada & Mullen, 1994; Pandey-Vimal, 1997; Beaudot & Mullen, 2005).

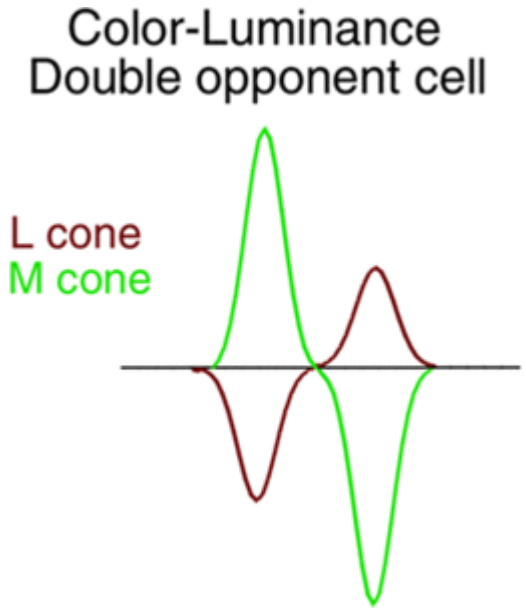

Figure 5. Diagram of the sensitivity profile of an idealized double-opponent V1 cell.

The modular viewpoint predicts that color perception should depend only on cells that are highly specialized for color detection (reviewed in Gegenfurtner & Kiper, 2003). An exemplar of this approach is the study of color responses in V1 and V4 neurons by Zeki (1983A, 1983B). Zeki selected from the population of V1 neurons only those cells that gave a bigger response to color than to black-white stimuli. Then he found that the population of cells he sampled were not really color-selective but rather wavelength-selective. This is the line of reasoning that led Zeki to conclude that V1 was not really responding to color. But his selection procedure led Zeki not to study the color responses of double-opponent cells because most of them respond to luminance as well as color.

Later studies also have selected for study those V1 cells that respond much more to color than to achromatic stimuli (Conway, 2001; Conway et al., 2002). Here, as in Zeki's (1983A, 1983B) studies, neurons were selected for investigation. The criterion used to screen cells for color selectivity in Conway and Livingstone (2006) was opposite signs of response to the same type of cone-isolating stimuli used in the main experiments: so-called sparse noise (Reid et al., 1997), that is, spots flashed briefly at random locations in the visual field.

In opposition to the modular viewpoint, there is extensive evidence for other types of color-responsive neurons in V1. For example, investigators found many color-responsive neurons in V1 that were also selective for spatial patterns of color. Thorell et al. (1984) reported the existence of many V1 neurons that were responsive to equiluminant color stimuli and also tuned for spatial frequency. Lennie et al. (1990) replicated and extended the results of Thorell and colleagues, studying V1 neuron responses to sinusoidal grating patterns that were formed by achromatic or color contrast. Their stimuli varied over a range of directions in DKL color space (Derrington et al., 1984). Lennie and colleagues studied the properties of all neurons they could record in V1 cortex. They found many neurons that were spatial-frequency-tuned and that also responded to both chromatic and achromatic stimuli. Most of the neurons they studied that were spatial frequency tuned were classified as cone-opponent. They found a small population that responded much more strongly to equiluminant color than to black-white grating patterns, and these were most responsive at low spatial frequencies. Lennie et al. (1990) hypothesized, like Zeki

(1983A, 1983B), that the only V1 neurons that were important for color perception were the small percentage of neurons that strongly preferred equiluminant stimuli. The idea that color perception depends on the minority of strongly color-preferring neurons was inconsistent with the integrated-color viewpoint articulated by Lennie (1999) though it resurfaced later in the review by Lennie and Movshon (2005).

Leventhal et al. (1995) unambiguously put forward an integrated-color-form point of view. They tested all neurons they encountered in the upper layers (2–4) of macaque V1 with a battery of visual stimuli to test sensitivity for color, direction of motion, orientation, and spatial selectivity. They also compared orientation selectivity and direction selectivity between neurons that were color responsive or color blind. This aspect of their study seems a logical requirement for understanding how specialized the “color” neurons were. Leventhal and colleagues found that the neurons they studied were selective on many dimensions. Cells sensitive to color were approximately as orientation selective as the cells that were color blind. Their results were not consistent with modular color processing in V1 cortex.

Friedman et al. (2003) in their study of color-coding in awake macaque monkeys also found that V1 and V2 neurons responded both to color and spatial pattern. Their stimuli were flashed, uniformly colored, geometric figures. Using several different indices of color selectivity, Friedman et al. (2003) found a substantial proportion (64%) of color-selective neurons in the upper layers of V1, and most of these were edge sensitive. Friedman et al. (2003) also found a smaller proportion of color-surface-responsive cells, so-called because these cells were not especially sensitive to edges. The edge-responsive color cells were mostly orientation selective, while the surface-responding cells were not selective for orientation. They wrote, “the vast majority of colour-coded cells are orientation tuned.”

Johnson et al. (2001) studied color responses in macaque V1, and their findings were consistent with and complementary to those of Thorell et al. (1984), Leventhal et al. (1995), and Friedman et al. (2003). Johnson et al. (2001) included all visually responsive single neurons that they recorded in macaque monkey V1, that is, they did not select neurons for study. Each neuron was assigned a single number, I, its sensitivity index, defined as:

$$I = \max\{\text{equilumresponse}\} / \max\{\text{lumresponse}\}.$$

High values of I indicated preference for colored stimuli compared to achromatic. All of the V1 population was divided into three groups: luminance-preferring ($I<0.5$); color-luminance cells ($0.5<I<2$); and color-preferring ($I>2$). A majority (60%=100/167) of V1 cells sampled were luminance preferring. Color-luminance cells were 29% of the total. The color-preferring cells were only a small fraction (11%) of the cells recorded, in agreement with previous studies. The percentage of color-luminance cells was higher in layers 2/3 where more than 50% of the cells we recorded were color-luminance cells.

Color-luminance cells were spatially tuned for equiluminant and also for black-white grating patterns (Johnson et al., 2001, replicating Thorell et al., 1984). The spatial frequency preference and bandwidth for a color-luminance cell was approximately the same for black-white or red-green equiluminant patterns (again replicating Thorell et al., 1984). Most color-preferring cells were not spatially tuned for equiluminant grating patterns; they preferred the lowest spatial frequency (Lennie et al., 1990, replicated in Solomon & Lennie, 2005). Color-preferring cells did not respond to red-green grating patterns at higher spatial frequencies (> 3 c/deg). The spatial frequency tuning of luminance-preferring cells was similar to that of color-luminance cells in preference and bandwidth (Schluppeck & Engel, 2002). Color-luminance cells responded poorly to color (and luminance) patterns at low spatial frequencies (<0.5 cy/deg), acting like double-opponent cells. Later Johnson et al. (2004, 2008) introduced a new conceptualization of the double-opponent receptive field, in which the opponent mechanisms were spatially side by side and elongated. They then proposed that the receptive fields of most color-luminance cells in fact conformed to the newly defined double- opponent organization they had introduced. This idea was subsequently adopted by others (Solomon & Lennie, 2007; Kiper, & Gegenfurtner, 2014).

The double-opponent cells in V1 cortex were almost all color-luminance cells. Spatial frequency analysis with cone-isolating stimuli, and with equiluminant stimuli, revealed that most color-luminance cells were tuned for spatial frequency in the 1–3 c/deg range (Johnson et al., 2001; Schluppeck & Engel, 2002). Furthermore, when the receptive fields of V1 double-opponent cells were mapped with cone-isolating stimuli, the receptive field subregions were elliptical, and the receptive field was not circularly symmetric (Johnson et al., 2008). The L- and M-cone inputs each had elongated, spatially separated increment-excitatory and decrement-excitatory sub-regions and there was cone opponency within each receptive field sub-region. That is, the cone inputs were of opposite sign at each receptive field location—therefore cone-opponent everywhere. In these ways, the double-opponent cells' receptive fields conformed well to the receptive field diagram in Figure 5. Conway reported results on mapping the receptive fields of double-opponent cells in awake Macaque V1 (Conway, 2001; Conway & Livingstone, 2006) that were not consistent with the results of Johnson et al. (2001) on double-opponent responses to gratings. Conway found maps that were approximately even symmetric and circular. Possible reasons for the different results were different sampling of cell populations and different visual stimuli. Conway's maps were derived from reverse correlation of neuronal responses with so-called sparse noise while Johnson et al.'s experiments employed drifting or flashed grating patterns. Future research will be needed to reconcile the different results.

Most double-opponent cells were orientation-selective for both achromatic and chromatic stimuli (Johnson et al., 2008). There were a few color-preferring, double-opponent cells that responded only weakly to achromatic stimuli (Johnson et al., 2004, 2008) though most had a significant response to achromatic patterns. Johnson et al. (2008) reported

the orientation selectivity distributions of double-opponent, single-opponent, and non-opponent cells in V1; the double-opponent cells as a population were orientation-selective but not as selective as the non-opponent cells. Single-opponent cells were not orientation-selective.

The big question is to what extent do single-and double-opponent populations contribute to color vision? While the topic is controversial, several studies agree that double-opponent cells are important for color perception. Schluppeck and Engel (2002) showed that human V1 signals measured by fMRI could be understood as a sum of signals from single- and double-opponent cells. Hass and Horwitz (2013) found that chromatic detection could be explained by the combined activity of single- and double-opponent cells. Recently, it was reported that supra-threshold color perception was predominantly controlled by double-opponent cells (Nunez et al., 2018), and the perceptual results were consistent with cVEP results described in the following section.

cVEP: The Color Visual Evoked Potential in Humans

The study of human visual evoked potentials (VEPs) also implied that color responses in human V1 cortex were produced by color-sensitive neural mechanisms that were spatially tuned like those measured in monkey V1. Many scientists have measured and studied the human cVEP, the color Visual Evoked Potential (cVEP, Murray et al., 1987; Rabin et al., 1994; Crognale, 2002; Souza et al., 2008; Crognale et al., 2013). cVEPs are evoked by equiluminant color modulation; therefore, they must be driven only from the subpopulations of cortical neurons that are responsive to color (Schluppeck & Engel, 2002). Evidence that the cVEP reflects color-evoked activity in the early visual cortex is as follows. The cVEP does not vary with attention, a result that strongly suggests it is evoked early in cortical visual processing (Highsmith & Crognale, 2010). Furthermore, normal cVEPs have been recorded in cases of cerebral achromatopsia where color appearance was lost and lesions were observed in ventromedial extrastriate cortex, but V1 responses to color were unaffected by the lesion (Victor et al., 1989; Crognale et al., 2013). The combined evidence from source localization, lack of attentional effects, and cerebral achromatopsia indicates that the cVEP is an index of early cortical responses to color.

The spatial tuning of the human cVEP is consistent with measurements of spatial tuning in V1 double-opponent cells in macaque monkeys. The cVEP amplitude is much smaller for lower spatial frequencies than at its peak between 1–2 c/deg, a result replicated in many different laboratories (Murray et al., 1987; Rabin et al., 1994; Tobimatsu et al., 1995; Porciatti & Sartucci, 1996). The spatial frequency tuning of the population of double-opponent cells in macaque V1 (Schluppeck & Engel, 2002) is consistent with the cVEP spatial frequency tuning data in humans. Another VEP experiment yielded the important result that color-responsive neurons should have the same diversity of spatial symmetry in their receptive fields as non-color responsive cells. In particular, there ought to be some odd-symmetric color-responsive cells (Girard & Morrone, 1995). This

prediction from VEP experiments on human observers is consistent with data obtained in studies of spatial symmetry of the receptive fields of V1 single- and double-opponent cells (Johnson et al., 2004).

Blue-Yellow Signals in V1 Cortex

The research on color processing in V1 reviewed so far was on studies of red-green color vision that is salient in Old World primates. However, the neuronal channel that carries blue-yellow signals from eye to cortex is the color pathway that is present in most mammalian species (see Jacobs, 2008). Chatterjee and Callaway (2003) found B/Y input to layer 4A/3B in their study of the laminar pattern of afferent LGN input to V1 in macaques. Buzas et al. (2008) studied the laminar distribution of S-cone driven responses in marmoset V1 and found no evidence for clustering of S-cone driven cells in layer 3 CO patches; rather the spatial distribution was uniform throughout layer 3.

There is no consensus on how strong the S-cone driven signals in V1 are. Single-unit studies in macaque V1 report relatively weak S-cone input, in agreement with the relative frequency of recording S-cone single-opponent cells in the LGN (Johnson et al., 2004; Solomon & Lennie, 2005) but De Valois et al. (2000) reported enhanced S-cone input in V1. fMRI studies of human V1 also disagree on this point, with Liu and Wandell (2005) reporting relatively weak S-cone driven responses while Mullen et al. (2007) reported stronger S-cone activity that was approximately as strong as the L-M signal in V1.

Johnson et al. (2010) reported studies of S-cone driven signals in the V1 cortex of tree shrews with the technique of intrinsic signal optical imaging and the higher resolution method of two-photon imaging. Johnson et al. (2010) found that tree shrew V1 cells that received S-cone input could be color-opponent or not and could be orientation-selective or not. The degree of color selectivity was not correlated with orientation selectivity, consistent with the results of Friedman et al. (2003). The results of Johnson et al. (2010) are consistent with the idea that S-cone opponent signals are combined with achromatic signals in cortical double-opponent cells. The relative contributions of the different cones can be gauged from cone-weight plots for V1 analogous to those presented earlier for the LGN (Figure 4). That is what we consider next.

Cone-Weights in V1 Cells

The weighting of cone signals by post-receptoral neurons is a strong clue to how signals about color are being handled by the visual system. We have already seen how useful the study of cone-weights is, in our review of the cone-weights of LGN neurons. Cone-weights are also useful in understanding cortical processing of color. Lennie et al. (1990) calculated cone weights for their sample of V1 cells based on their measurements of neuron’s responses in DKL space when optimal spatial grating patterns were used as stimuli. Conway and Livingstone (2006) also employed cone-isolating stimuli when they

performed sparse-noise mapping in awake Macaque V1 and also estimated cone weights across the population; in general, they found more S-cone weights than Lennie et al. (1990). They also found the L:M cone-weight ratio more tightly clustered around 0.5: –0.5 than Lennie et al. (1990) had reported. Horwitz and Hass (2012) also calculated cone weights from their experiments in awake Macaque V1. Their results are in broad agreement with those of Johnson et al. (2004) in particular the broad distribution of L:M ratio in color-responsive neurons. The sophisticated analysis of Horwitz and Hass (2012) revealed that many V1 cells combined cone signals in a nonlinear manner.

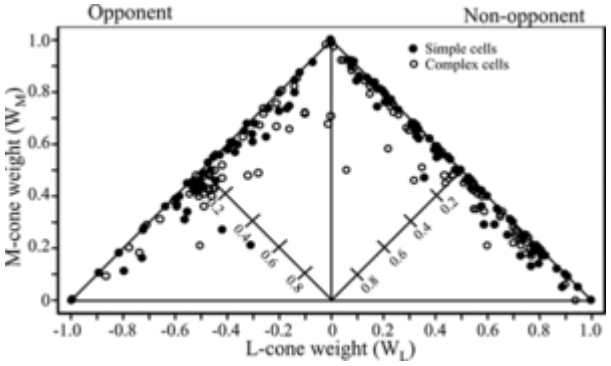

Figure 6. Cone weights of V1 cortical cells in macaque monkeys. The plot shows the relative weights of cone input for all simple and complex cells in the population studied by Johnson et al. (2004) (n=247). The sign of L-cone input was multiplied by –1 for cells that were found to be cone-opponent in responses to cone-isolating grating patterns. From Johnson et al. (2004).

Johnson et al. (2004) used cone-isolating stimuli to estimate cone weights for optimal sine-grating stimuli in anesthetized Macaque monkeys. A summary figure of the V1 cone weights measured in that study is provided in Figure 6. Each dot is one cell's cone weight. The cone weights are plotted in a triangle rather than the diamond format of Figure 4 because V1 cells usually have receptive fields with multiple sub-regions and therefore one cannot assign a single sign to the neuron's response as one can for LGN cells. Therefore, cone weights for cortical cells must be plotted in "Opponent" and "Non-Opponent" quadrants in the upper half plane, as in Figure 6. In the format of Figure 6, as in the cone-weight diamonds in Figure 4, the S, M, and L cone weights for each point add up to 1. So if there were S-cone input, its strength could be estimated by how much distance there is between the cone-weight point and the closest outer diagonal boundary of the cone-weight triangle. The cone-weight point for a neuron that gets no S-cone input will lie on one of the diagonal lines that bound the triangle. Points off the line indicate some S-cone input. The cells plotted in the non-opponent quadrant of Figure 6 received cone input that could be written a_L*L + a_M *M where a_L and a_M are the weights and sign (a_L) = sign (a_M). The non-opponent cells are mostly luminance-preferring neurons. Cells plotted in the opponent quadrant received cone input that could be written a_L*L + a_M*M where sign (a_L) = - sign (a_M). The distribution of the ratio a_L/a_M was very broad, meaning there was a wide variation in the relative cone weights of L and M cones in the V1 population. The distribution also was very broad for double-opponent, color-luminance cells, but was tightly clustered near ($a_{M,}$ $a_{L)}$ = (0.5, −0.5) for single-opponent color-preferring cells (Johnson et al., 2004). Cone-weights for 247 cells are plotted in Figure 6, and only a small fraction of that population had a large S-cone weight as judged by distance of the points

from the bounding lines of the triangle. The relative proportions of opponent and non-opponent cone weights in Figure 6 were confirmed in the study of Solomon and Lennie (2005) in distinction to an earlier study that reported the large majority of neurons were cone-opponent (Lennie et al., 1990). The wide distribution of the ratio a_L/a_M for the opponent cells in Figure 6 (replicated both by Solomon & Lennie, 2005; Horwitz & Hass, 2012) could be a result of the nature of the LGN input when spatial patterns are used (as discussed above in the section on single-opponent LGN cells), or could be generated by intra-cortical processing, or both. Figure 6 indicates heterogeneity in the population of color-responsive neurons in V1 and therefore implies that there must be population-coding of color in V1, as suggested by Wachtler et al. (2003).

V1 Population Responses: The Hole in the Middle of a Red Square

All of the research above suggests that the V1 population representation of colored objects should be a filtered version of the visual image. Zweig, Zurawel, Shapley, and Slovin (2015) asked the questions: what is the representation in V1 cortical population responses of surfaces defined only by color? And is there a uniform filled-in representation of the perceived visual image in V1? They studied surface representations in awake, behaving monkeys with voltage-sensitive dye imaging (VSDI) in V1 cortex. VSDI measures the neuronal population activity in upper layers of V1 as an average of the slow membrane potential and nerve impulses in thousands of cortical cells (Slovin et al., 2002).

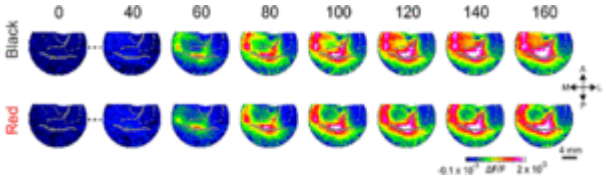

Figure 7. Voltage-sensitive-dye-images of Macaque fascicularis V1. Plotted is a sequence of Voltage-Sensitive-Dye (VSD) maps in V1, from an example session in which a macaque monkey was presented with 2° black (top) or red (bottom) squares as visual stimuli while the monkey was doing a fixation task. The region of the cortex in the imaging window contained the cortical projection of the squares. Stimulus duration was 300 ms. The time when each image was taken, written above the image in ms, was relative to stimulus onset. Each map is averaged over 20 ms of time.

Re-drawn from Zweig et al. (2015).

Figure 7 shows the spatiotemporal VSDI response (fluorescence change, dF/F) evoked by 2° X 2° squares in an example recording session. Each image is of a region of V1 approximately matched to the position of the stimulus in the visual field. The magnitude of heightened evoked activity is indicated in the color scale keyed in the figure. Responses to black squares on a gray background are presented in the upper row and responses to red squares in the lower row. What is strikingly evident in the VSDI images is a hole in the middle of the cortical representation of a square both for black squares and for equiluminant red squares. Though perceptually the red square appeared uniform, in fact the V1 representation of the square remained a filtered version throughout stimulus presentation. The results with

pure color squares in Figure 7 support the conclusion that an isomorphic representation in V1 is not required for uniform color surface perception, consistent with the results of Friedman et al. (2003).

Conclusions

Color perception in macaque monkeys and humans depends on the visually evoked activity in three cone photoreceptors and on the neuronal post-processing of the cone signals. In the retina there is specific connectivity designed to enhance color processing of red-green and blue-yellow signals. The result is the population of single-opponent retinal ganglion cells as well as their targets in the LGN, the Parvocellular and Koniocellular neurons. In the retinal periphery, single-opponency deteriorates as the specific wiring of the retinal receptive fields is lost. There is a second stage of post-receptoral cone signal processing in the primary visual cortex, V1. Here there is wide diversity of color properties and spatial tuning of the single-opponent and double-opponent color neurons. Single opponent cells, in LGN and in V1, respond to color modulation over their receptive fields and respond best to color modulation over a large area in the visual field. Single-opponent cells could provide neuronal signals that could be used for estimating the color of the illumination. Cortical double-opponent cells subtract cone signals across space as well as between cones. Double-opponent cells have visual properties that explain most of the phenomenology of color perception of surface colors. For instance, they respond to color edges and spatial patterns of color. There is a wide diversity of cone-weights in double-opponent cells indicating that color-coding may be done by population-coding in the cortex.

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Robert Shapley

Center for Neural Science, New York University