HUMAN COLOUR VISION: 1. COLOUR MIXTURE AND RETINO-GENICULATE PROCESSING

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ABSTRACT

Normal humans can match any wavelength of light with a combination of three other wavelengths. This capacity, called trichromacy, is due to the presence of three types of cone photopigment in the photoreceptors. These photopigments differ in their peak absorption at either short (S), middle (M), or long (L) wavelengths. A match between two fields of differing spectral distribution occurs when the relative intensities of lights in one field are adjusted so that the activity in each of the three classes of photoreceptor is identical for the two fields. This is possible because of the principle of univariance: light absorbed by a photopigment molecule produces the same response independent of wavelength.

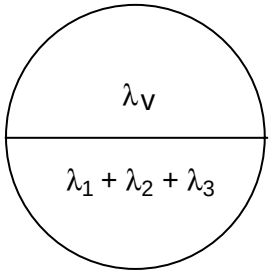
Bipolar cells separate the response of the photoreceptors, giving rise to parallel ON and OFF pathways from the retinal ganglion cells to cortex. The main projection site of the retinal ganglion cells for processing of chromatic signals is to the lateral geniculate nucleus (LGN). One class of retinal ganglion and LGN cell (parvocell) carries signals based on combinations of cone signals, L-M or S-(L+M). Although neural signals carrying colour information undergo additional transformations in the cortex, responses of these cells can account for many aspects of colour discrimination.

1. Trichromacy and its Representation

Colour is a sensation resulting from the activity in the nervous system that is initiated by light. Classically, colour is described by three dimensions: hue (chromatic sensations such as red, green, yellow or blue), saturation (proportion of chromatic content relative to achromatic or lightness-darkness content) and brightness (our subjective impression of stimulus intensity). These are not the only perceptual qualities of colour, however, as one can also speak of a colour's film or surface quality, its lustre, transparency and so forth.

Most objects that we experience in nature reflect a broad band of the spectrum, but in the laboratory it is useful to use narrow spectral bands, or monochromatic lights. With such stimuli, Maxwell (1860) and Helmholtz (1867) were able to demonstrate one of the most important characteristics of the human visual system: that it is normally trichromatic. Figure 1 illustrates what is meant by trichromacy.

If half of a bipartite field such as that shown in Figure 1 is filled with monochromatic light of variable visible wavelength (λ_v, 400-700 nm), or any other spectral composition, it is possible for an individual to adjust the amounts of three other monochromatic lights ($\lambda_1 + \lambda_2 + \lambda_3$) placed in the other half of the bipartite field so that the combination matches λ_v perfectly in hue, saturation and brightness. These two fields are said to be metameric (*i.e.*, the stimuli are physically different but perceptually identical). The matching stimuli ($\lambda_1 + \lambda_2 + \lambda_3$) are called primaries and any three wavelengths may serve as primaries provided only that no one of the primaries can be matched by a combination of the other two. Thus, the three primaries are typically selected from the short-, middle- and long-wavelength



f $\lambda_V \equiv \lambda_1 + \lambda_2 + \lambda_3$

$(\lambda_V) \equiv k(\lambda_1 + \lambda_2 + \lambda_3)$ (scalar invariance)

$\lambda_V + \lambda_a \equiv \lambda_1 + \lambda_2 + \lambda_3 + \lambda_a$ (additivity)

Fig. 1 Illustration of a bipartite stimulus used in a colour-mixture experiment. By varying the relative intensities of three primaries in the lower portion of the field, it is possible to obtain a metameric match (denoted by the symbol ≡) to the upper half of the bipartite field. The match can be specified by a colour-mixture equation and can be shown to obey the properties of scalar invariance and additivity.

regions of the spectrum. An individual who requires three primaries to match any other light is known as a trichromat.

Metameric matches can be described by a colour-mixture equation using the symbol "≡" to denote that the equality is perceptual, not physical. With appropriate specification of the units of the colour match, it is also possible to manipulate the match like an algebraic equation. Thus, for a trichromat, if one of the three lights in the mixture (*e.g.*, λ_1) in the bottom of the bipartite field is moved to the other half-field, the equation is written to express this by a negative sign: $\lambda_v - \lambda_1 \equiv \lambda_2 + \lambda_3$. In this case, the two half-fields would still match as implied by the ≡ symbol. This manipulation is necessary in normal colour matching when λ_v may take on any value in the visible spectrum. Two important characteristics of colour matching are described by Grassmann's (1853) laws of scalar invariance and additivity, as stated in Figure 1. Scalar invariance means that if the intensity of one half of the field is increased or decreased by a factor k, the match will be restored by increasing or decreasing the lights in the other half of the field by the same factor. Additivity means that if a light λ_a is added to one half-field, the match will be restored by adding the same light to the other half field.

While trichromacy characterises normal human vision, it should be noted that approximately 6% of males and 0.5% of females are anomalous trichromats. That is, they require three primaries for spectral colour-matching, but they combine them in proportions that differ systematically from those selected by most of the population. A more extreme departure from normal trichromacy is found in about 2% of males and about 0.03% of females. These individuals are said to be dichromatic because they require only two primaries for colour matching. Finally, while rare, some individuals are classified as cone monochromats because they can match any wavelength with any other simply by varying the intensity of one of the lights. Collectively, these departures from normal colour vision are referred to as (congenital) "colour vision deficiencies."

2. The Eye and Retina

Vision is initiated when light travels through the ocular media (cornea, anterior chamber, lens and vitreous humour) and retinal cells of the eye until it is absorbed by the photopigment contained in the photoreceptors (rods and cones). The left side of Figure 2 illustrates the human eye, while the right side shows an enlargement

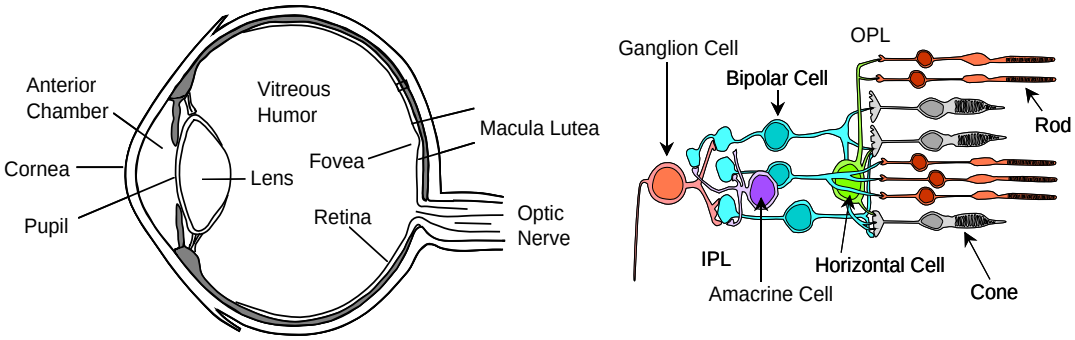


Fig. 2 A schematic of the globe of the eye is shown on the left, while an enlarged view of the retina is shown on the right. Each eye contains approximately 125 million rod and 6 million cone photoreceptors. Signals from the photoreceptors are processed by about 10 million horizontal, amacrine and bipolar cells and then leave the eye via the 1 million axons of ganglion cells comprising the optic nerve. Synapses between retinal cells occur in the outer plexiform layer (OPL) and inner plexiform layer (IPL).

of the thin layer (~0.4 mm) of cells in the back of the eye, the retina. There are five general cell types in the retina. Photoreceptors (rods under dim or scotopic illumination and cones in daylight or photopic illumination) initiate signals for vision upon absorption of light. These signals are transmitted in a pathway from the bipolar cells to the ganglion cells. The feedforward signals are modulated by lateral connections from the horizontal cells of the outer plexiform layer and the amacrine cells of the inner plexiform layer. The ganglion cell axons leave the eye in a bundle called the optic nerve.

3. Phototransduction and the Univariance of the Photoreceptor Response

Figure 3a shows the three main portions of a human cone photoreceptor. Light travels through the inner segment containing the nucleus and cytoplasm and then reaches the outer segment with its many disk-like membranes containing photopigment molecules. The cone inner segment has appropriate dimensions and refractive index to act as a light funnel or waveguide. In the dark, there is a steady flow of current out of the inner segment, through the outer segment and then through the inner segment membranes. Inward flow of sodium and calcium ions in the outer segment is balanced by outward flow of potassium ions. The net effect is to maintain a resting potential of about -30 mV. Absorption of light interferes with the current flow by blocking some of the sodium ion channels and the intracellular potential becomes more negative (*i.e.*, the photoreceptor becomes hyperpolarized). The blockage of the current and intracellular negativity spreads electrotonically until it reaches the synaptic terminal. Transmission of the photoreceptor signal to bipolar and horizontal cells is due to a reduction in release of the neurotransmitter glutamate from the synapse (Baylor, 1987).

Photopigment molecules contain a protein or opsin bound to a chromophore (11-cis-retinal), the light absorbing region of the pigment. When a quantum of light is absorbed, the chromophore undergoes a cis-trans isomerization in one molecule (with a probability or quantum efficiency of about 0.67) and this initiates a cascade of chemical reactions that interfere with the dark current. Cis-trans isomerization is also called bleaching because the chromophore loses its intrinsic coloration and is unable to capture additional quanta until it returns to its original (cis) configuration

through a process called regeneration. The probability that a photopigment molecule will absorb a single quantum of light depends on the energy contained in the quantum, which is inversely proportional to its wavelength. However, once a quantum is absorbed and isomerization occurs, the response of the molecule is independent of the wavelength. This is known as the principle of univariance: once a quantum is absorbed, all information about the wavelength of the light is lost. The response of the receptor is based on the summed activity of all photopigment isomerizations, and it is impossible to discern the spectral content of the light stimulus from the receptor signal.

A great deal has been learned about the receptor response by recording the current change from single outer segments of monkey and human rods and cones while stimulating them with light. Each trace in Figure 3b shows the average of a large number of responses from a single cone to flashes of increasing intensity. The peak response occurs at about 55 msec for all different classes (to be described below) of primate cone. The response increases as a monotonic function of flash intensity up to a certain value; beyond this value there is no further increase in

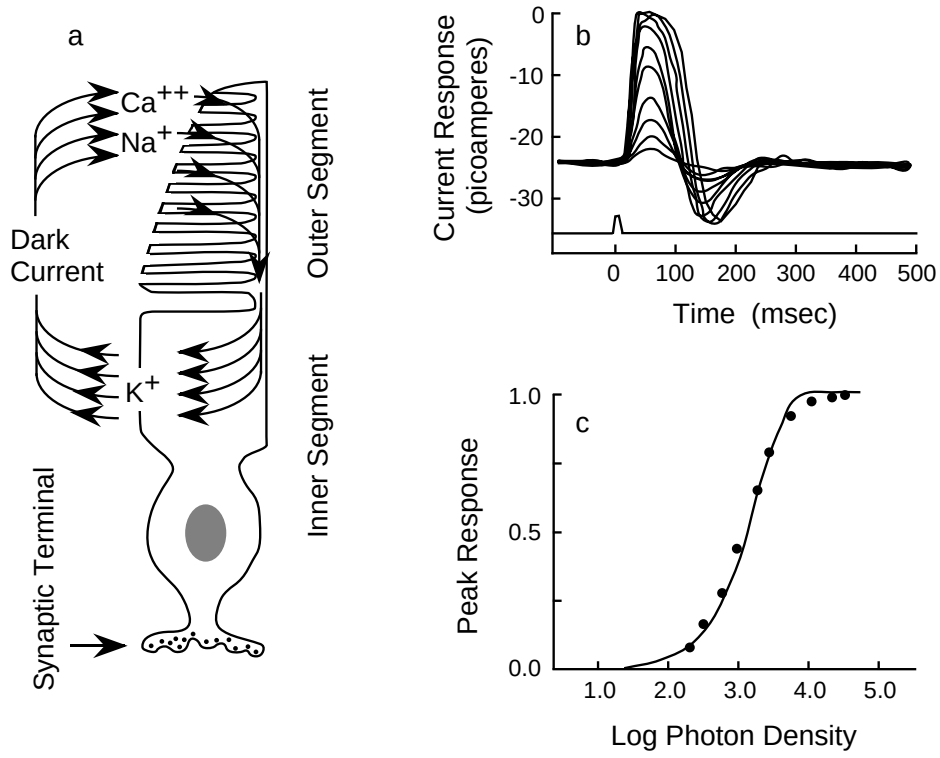


Fig. 3 Panel a illustrates a primate cone photoreceptor and the exchange of sodium (Na^+), calcium (Ca^{++}) and potassium (K^+) ions that produces current flow in the dark. Absorption of light causes a cis-trans isomerization of the photopigment and a disruption of the dark current owing to closure of sodium channels. This hyperpolarizes the membrane and decreases the release of neurotransmitter at the cone synapse. Panel b shows average responses of a cone to flashes of increasing light intensity. The bottom trace shows the temporal characteristics of the light flash. (After Schnapf & Baylor, 1987). Panel c plots the peak amplitude of the cone response (relative to the response maximum in picoamperes) as a function of log quanta of a flash.

amplitude, but duration of response may still increase. The response of cones (but not rods) is characterised by a prominent negative "rebound" in response before returning to the dark level.

Cone photopigments are not stable and therefore may produce random activity that is not different from activity due to absorption of light. This intrinsic noise is often called the "dark light" or *Eigengrau*, and it imposes a limit on the absolute sensitivity of the photoreceptor. Figure 3c shows the response vs. intensity function of a cone photoreceptor determined from current measurements of Schnapf and Baylor (1987). The dynamic range of a single cone is approximately 1.5-2.0 log units relative to the ambient illumination, but this range can be extended by adaptation mechanisms in order to mediate the 6-7 log unit range of photopic vision.

4. Spectral Sensitivity

Spectral sensitivity is measured by varying a monochromatic stimulus to determine the reciprocal number of quanta required for a criterion response (*e.g.*, detection 50% of the time). If the stimulus follows about 7 minutes of adaptation (the time in the dark required for cones to reach their maximal sensitivity) and is presented as a 1° spot[1] to the fovea (see Fig. 2), which contains only cones, the resultant spectral sensitivity curve has a peak (λ_{max}) at about 555 nm. It represents daylight (photopic) sensitivity. However, if the stimulus is imaged in the periphery where rod density exceeds cone density and follows about 30 min dark adaptation, the resultant spectral sensitivity function represents rod (scotopic) activity. This function is much higher on the sensitivity axis than the photopic spectral sensitivity function, and its λ_{max} is shifted to about 505 nm (see Figure 4).

4.1. Luminance

Because the sensitivity of the eye is not constant with wavelength, the effectiveness of a light as a stimulus for vision is not well specified by purely physical metrics. The International Commission on Illumination (Commission Internationale de l'Éclairage, CIE) has, therefore, developed a system for specifying the intensity of a stimulus as physical energy weighted by the spectral sensitivity of an average observer. This quantity is called the luminance and the spectral weighting functions are called the photopic (V_λ) and scotopic (V'_λ) luminosity functions. These functions are shown as smooth curves in Figure 4. The formal definition of luminance is thus, the amount of light energy at each wavelength interval weighted by the luminous efficiency function and summed across the visible spectrum: $k \int E_\lambda V_\lambda d_\lambda$, where E_λ is the radiant energy contained in wavelength interval d_λ, and V_λ is the relative photopic spectral sensitivity function for the standard observer. For scotopic conditions the same formula applies except that V'_λ is used instead of V_λ. Luminance applies to light reflected from a surface, and it is specified for a unit area of the surface. The value of k depends on the photometric unit used, the most common of which is the candela per square meter.

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[1] The retinal image subtense of a stimulus can be specified as a visual angle, $\alpha = 2 \arctan (s/2d)$, where s is the size of the stimulus and d is the distance of the stimulus to the nodal point of the eye (*i.e.*, the distance between the stimulus and the front surface of the eye or cornea plus 7 mm). 1° (deg) = 60' (min); 1' = 60" (sec). A useful "rule of thumb" is that the size of the thumbnail at arms length is about 2 deg.

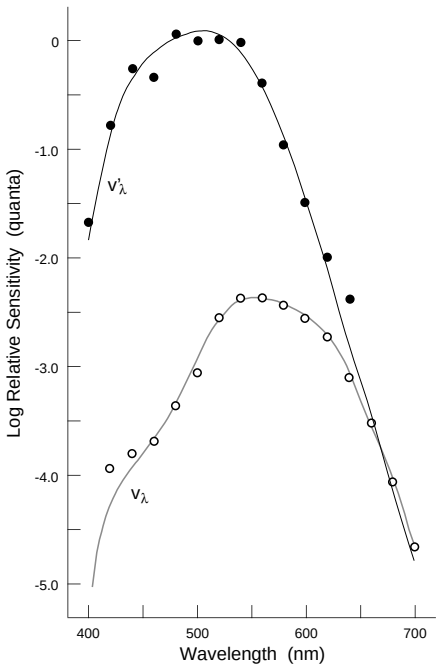

Fig. 4 Log relative quantal sensitivity for one observer plotted as a function of wavelength. Filled circles represent scotopic conditions: detection of 2 Hz (5° diameter) flicker following 30 min dark adaptation. The solid upper curve shows the CIE V'λ function, vertically translated to fit the data. Open circles represent photopic conditions: minimising flicker between monochromatic lights and a 2,000 troland broadband stimulus presented in square-wave counterphase at 14 Hz (1° diameter stimulus). This method is called heterochromatic flicker photometry. The grey curve is the CIE Vλ function, vertically translated to fit the data. (After Volbrecht & Werner, unpublished.)

A few caveats about luminance specifications may be in order. First, luminance is not a subjective quality. It can be determined from the above equation if one knows the energy at each wavelength in a stimulus. Alternatively, luminance can be measured directly with a meter that has a filter that transmits light according to the V_λ function. Second, luminance should not be confused with brightness, a psychological dimension of colour that varies with many factors; stimuli of equal luminance may not be equally bright. The term luminance should be reserved for specifying light intensity (in photometric terms), and the term brightness should be reserved for describing one aspect of colour appearance.

4.2. Two-Colour Increment Thresholds

The trichromacy of human vision implies the existence of three classes of photoreceptor or photoreceptor channels. A number of investigators have attempted to identify these "fundamentals" of human colour vision by measuring spectral sensitivity under conditions of chromatic adaptation (Stiles, 1953; Wald, 1964). The inset in Figure 5 illustrates the stimulus configuration used by Stiles and others to measure foveal two-colour increment thresholds; a 1° incremental test flash is presented on a continuously-viewed adapting field. The wavelength of the background is chosen to adapt or reduce the sensitivity of two of the three classes of cone, leaving the third class more likely to detect a superimposed test flash. Rigorous tests, however, resulted in isolation of seven rather than the expected three colour mechanisms which Stiles called π-mechanisms.

There are several criteria by which one may reject the hypothesis that mechanisms isolated in this way are candidates for the cone fundamentals (*i.e.*, the mechanisms underlying colour mixture). One of the most important criteria is that the cone fundamentals must be expressible as linear combinations of colour-matching data since the latter must be linearly related to the cone photopigment

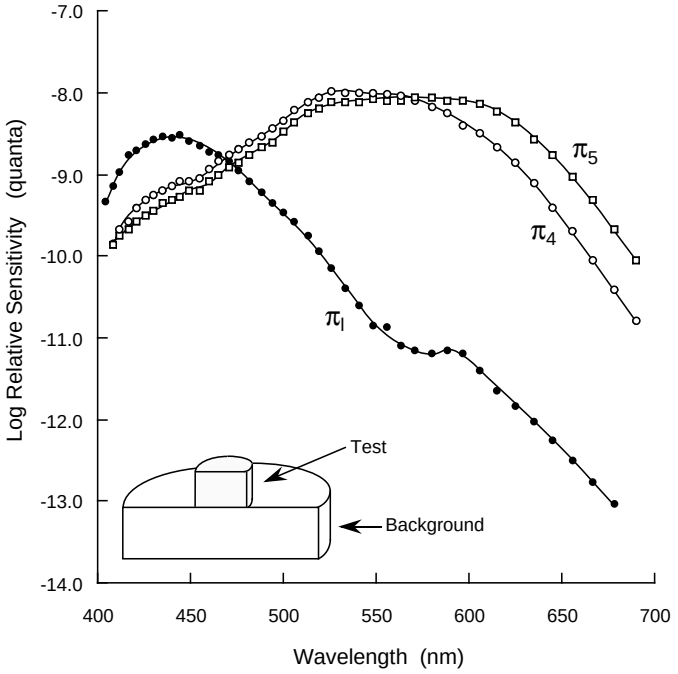

Fig. 5 Three of the foveal colour mechanisms isolated by Stiles (1953) by determining the reciprocal intensity of the background required to raise the test threshold by 1 log unit from absolute threshold. The test flash (1°, 200 msec) was superimposed on steady (10°) chromatic backgrounds as illustrated by the inset. The graph presents three mechanisms having peak sensitivity at short- (π_1, filled circles), middle (π_4, open circles) and long- (π_5, open squares) wavelengths.

absorptions. On the basis of this criterion, Stiles (1953) rejected the hypothesis that these mechanisms are due to the response of single classes of cone (but see Estévez & Cavonius, 1977). The π-mechanisms can also be rejected as cone fundamentals because they fail to obey Grassmann's law of scalar invariance (Figure 1). The physiological basis of scalar invariance is described in the next section.

The two-colour increment threshold method remains important even though it does not permit perfect isolation of the cone fundamentals. For many purposes the method may reveal sufficient information about cone contributions to allow strong inferences about physiological mechanisms. In addition, the small discrepancies between π-mechanisms and cone fundamentals may be important for revealing properties of visual mechanisms related to adaptation.

5. Cone Photopigments and Receptor Spectral Sensitivities

Several different methods have been used to measure the absorption spectra of the photoreceptors in the primate retina. The first approach was based on reflection densitometry whereby the amount of light reflected from the eye of a dichromat or monochromat was measured as a function of wavelength before and after exposure to an intense bleaching light (Weale, 1959). The difference in spectral reflectance before and after bleaching is assumed to represent the bleachable photopigment contained in the retina. Another approach, called microspectrophotometry (MSP), involves the measurement of spectral absorption of single cones from excised retinas (Bowmaker, 1984). In both cases, the results are consistent with three classes of human cone photopigment having λ_{max} at the short-, middle- or long-wavelengths, referred to as S, M and L cones, respectively.

Given the principle of univariance, one would expect the photopigment absorption spectrum to agree with the receptor's sensitivity measured electrophysiologically (the receptor's action spectrum). A possible complication is that MSP measurements are obtained with light passing through the outer segment transversely and therefore through a lower density of photopigment[2] than in the living eye where the light passes longitudinally through the outer segment and hence through more photopigment. Increasing the amount of photopigment by increasing either the concentration of pigment or the path length will increase the quantal capture and broaden the photopigment absorption spectrum. Measurements of the current response of outer segments from macaque and human cones have produced the most reliable physiological data on cone action spectra to date (Schnapf *et al.*, 1987). These results yield three types of action spectra with peak sensitivities at approximately 430, 530 and 560 nm, in close agreement with data obtained from human psychophysics.

Absorption spectra and action spectra obtained with these various methods have been compared with data from two-colour increment threshold experiments and from the measurement of photopic spectral sensitivity in human dichromats and cone monochromats. One complication in evaluating estimates of cone sensitivities from human psychophysical experiments is that some of the light delivered to the cornea is lost before it reaches the retina owing to absorption or scatter by the ocular media within the eye. This absorption is usually highest at short wavelengths and mostly due to the lens. As shown in Figure 6, the lens acts as a cut-off filter for wavelengths below about 400 nm, and absorbs significantly in the visible short wavelengths. In addition, there are large individual differences in optical density for persons of the same age (on the order of 1.0 at 400 nm) and increases in mean optical density as a function of age (Werner, 1991). Additional attenuation of short-wave light also occurs at the fovea by the yellow macular pigment which lies in the receptor fibre layer and inner plexiform layer. This

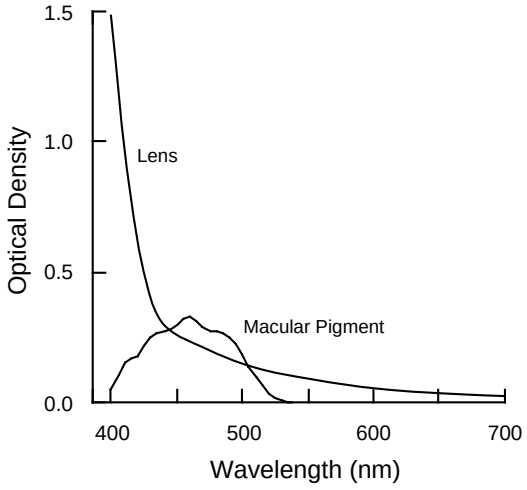

Fig. 6 Optical density (log of the reciprocal of transmission) is plotted as a function of wavelength for the lens (after Norren & Vos, 1974) and macular pigment (after Vos, 1978a). These functions are for an average young adult but there are substantial individual differences in peak density.

[2] The amount of photopigment contained in a receptor is usually described in terms of the pigment's density (D): D= log (1/t), where t is the transmittance, or fraction of incident light that passes through the pigment.

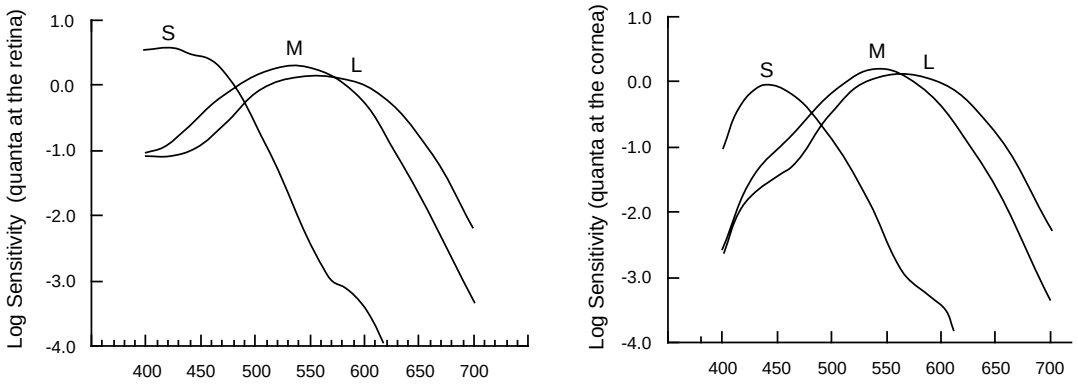

Fig. 7 Spectral sensitivity of the human cone fundamentals of Vos and Walraven (1971) specified in terms of quanta arriving at the retina (left) or delivered to the cornea (right). Sensitivity at the cornea was calculated from retinal cone sensitivities corrected for light losses due to absorption by the ocular media and macular pigment using data presented in Figure 6.

pigment reaches its highest density in the foveal centre and declines exponentially as an increasing function of retinal eccentricity, reaching its asymptotic minimum at about 7-8° (Bieber & Werner, 1998). The macular pigment varies in its peak density (at 460 nm) from about 0-1.0 across individuals although it does not change much as a function of age.

Deriving the "true" cone action spectra, or cone fundamentals, has been a major preoccupation of several generations of colour vision researchers. Modern estimates by Vos and Walraven (1971) are shown in Figure 7, with and without corrections for ocular media and macular pigment. These curves are consistent with the spectral sensitivities of individuals who have only S and M *or* L cones (dichromats) tested under conditions in which S-cones are assumed to be "silent" (Smith & Pokorny, 1975). Moreover, these functions are linearly related to the colour-matching functions of a standard observer (Vos, 1978b).

The absorption spectrum of a photopigment is determined by the opsin molecule. The molecular codes for different opsins have been identified and the gene sequences largely determined (Nathans *et al.*, 1986, Neitz & Neitz, 1998). It is known that chromosome 3 codes the rod photopigment, while chromosome 7 codes the S-cone photopigment. M- and L-cone photopigments are coded by the X chromosomes. Not all individuals with normal trichromatic vision have the same gene sequences or the same cone photopigments. Multiple genes code for the M- and L-cone photopigments, and there is normal variation in the number of these genes. As a result, the peak absorption of these photopigments varies in small increments of about 6 nm for M- and L-cones (Neitz *et al.*, 1991). Certain females (heterozygous carriers) possess genes for anomalous colour vision on one X-chromosome and genes for normal colour vision on their other X-chromosome. Their colour matches are trichromatic at different light levels, but they violate Grassmann's law of scalar invariance because the proportions of the primaries are not the same across light levels (MacLeod, 1985). This implies that they have more than three photopigments and that their trichromacy is due to photoreceptor-channel trivariance rather than photopigment trivariance.

5.1. Silent Substitution

Photopigment polymorphism notwithstanding, it is possible to describe the photoreceptor spectral sensitivities of most normal trichromats by the cone fundamentals shown in Figure 7. An important implication of this fact is that it is possible to create pairs of stimuli, both of which produce the same quantal catch in two of the three cone classes (and in rods), but differential quantal catch in the remaining cone type. When these lights are temporally alternated, flicker can only be detected by an individual possessing the cone type being modulated. This is a powerful technique for classifying an observer's colour vision and for isolating the contributions of different cone types to postreceptoral cells or mechanisms (Estévez & Spekreijse, 1982).

6. Distribution of Cones Across the Retina

The retinal distribution of rods and cones is shown in Figure 8 (left). A careful examination of the cone distribution reveals asymmetries; at any given eccentricity, the nasal retina has a higher density of cones than the temporal retina. The inferior retina may also contain about 5% more cones than the superior retina.

Vos and Walraven's (1971) derivation of cone fundamentals from psychophysical data led them to conclude that the ratio of L, M and S cones was 32:16:1 for the central 2° of human retina. These cone ratios are consistent with psychophysical estimates (Williams *et al.*, 1981; Cicerone & Nerger, 1989), while anatomical methods have suggested a somewhat higher proportion of S cones.

M and L cones have not been differentiated morphologically, but S cones appear to have slightly larger inner segments than M and L cones, and they can also be identified by selective uptake of various chemical labels. Results obtained by labelling with an antibody (antiopsin blue) are presented in Figure 8 (right). Although the highest density of cones is in the fovea, there are very few S-cones there, and perhaps none in the very centre (~15'). S-cones as a proportion of the overall cone population increases from the fovea to about 2° where they reach an asymptotic value of about 7-8%. The small number of S cones has important

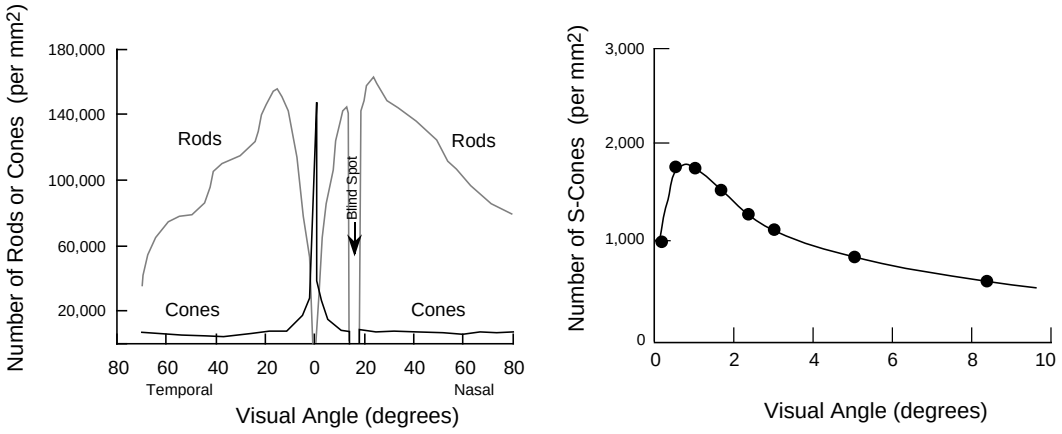

Fig. 8 Distribution of photoreceptors along the horizontal meridian for a human eye is shown on the left (after Østerberg,1935) while that for S-cones only is shown on the right (after Curcio *et al.*, 1991).

implications for visual perception. Partly because of their numbers and uniform distribution within a subregion of retina, and partly because of their neural connections, the S cones make a negligible contribution to high spatial acuity (Kelly, 1974), and they do not support the perception of chromatic borders (Tansley & Boynton, 1976). The S cones are very important for colour discrimination, although colour discriminations that depend on S cones will be impaired for very small stimuli due to their more or less uniform separation of about 10', and for stimuli falling on the centre of the fovea where S-cones are absent. In these cases, one can experience small-field tritanopia, colour vision comparable to that of a person lacking S cones (called a tritanope).

7. Adaptation and the von Kries Coefficient Rule

Our visual system has evolved to respond to an enormous range of light intensities, on the order of 12 log units. The number of quanta reaching the photoreceptors varies from about 10^2 per sec on a dark night to about 10^{14} per sec on a bright afternoon at the beach (Barlow, 1982). Two mechanisms of coping with this enormous range are clearly identifiable: (1) we have two photoreceptor systems, rods for scotopic illumination and cones for photopic illumination, with both rods and cones active at mesopic illumination; and, (2) the eye pupil adjusts its size to admit more light when the illumination is low and less light when the illumination is high. The pupil can only account for about 1.0 log unit of the light range control, leaving rods and cones to cover about 6 or 7 decades of variation each. As we have seen, the dynamic range of single cones is only about 1.5-2.0 log units (Figure 3c), so the system must have other mechanisms of extending its range. This is what is meant by adaptation. Several mechanisms of receptor adaptation have been identified by mass response recordings (Valeton & Van Norren, 1983). It is also clear that adaptation occurs at postreceptoral levels (Shapley & Enroth-Cugell, 1984).

Of particular importance for colour vision is a type of gain control postulated by von Kries (1905) whereby sensitivity of the receptors is reduced in proportion to their activity. This is sometimes called the Coefficient Rule because it implies a separate multiplicative scaling of each class of receptors. While this adaptive effect strongly alters colour appearance, it has no effect on colour matching.[3] This is because a colour match occurs only when the quantal absorptions of all the photoreceptor types are equated for both fields; after adaptation there may be a reduction in quantal catch by one or more classes of photoreceptors, but it has the same effect on both fields in the match. This fact is known as the persistence rule.

Von Kries adaptation is believed to be important in maintaining colour constancy, our experience that colours appear more or less the same under different illuminants. Consider, for example, a scene that is illuminated by a (white) light that emits quanta at all visible wavelengths and then by a (red) light that emits relatively

[3] This generalization holds only at light levels that are not sufficiently high that they cause significant photopigment bleaching. Under this condition, the adaptive effect can be thought of as logarithmic scaling of the receptor's sensitivity function without a change in the shape of the function. If the adapting light is intense enough to cause substantial bleaching of photopigment, the shape of the receptor sensitivity functions will be changed. Color matches are not upset by changes in the photoreceptor heights, but they are affected by changes in their shapes. As a result, adapting levels that cause significant bleaching lead to a violation of scalar invariance of color matching.

more quanta at long wavelengths. In the latter case, objects will necessarily reflect more long-wave light to the eye and the activity of the L cones will increase. This increase in quantal absorption will, however, lead to a decrease in L-cone sensitivity following the von Kries Coefficient Rule. The ratio of signals from the three cone classes under the two illuminants will, therefore, be similar following adaptation and the colours of objects will necessarily also be similar. Much data on colour constancy can be modelled quite well by von Kries adaptation (Brainard & Wandell, 1992; Lucassen & Walraven, 1993), although higher-level mechanisms are also needed under some conditions (D'Zmura & Lennie, 1986).

Von Kries adaptation is a cornerstone of all models of chromatic adaptation, although it must be combined with additional processes to explain colour appearance completely. A second process is a subtractive process (Jameson & Hurvich, 1972; Walraven, 1976) that transmits the signal from the test flash but removes the additive contribution of the background, or at least much of it (Shevell, 1978). Horizontal cells (described in the next section) are reasonable candidates for carrying out this subtractive process.

8. Horizontal Cells and the Importance of Spatio-Temporal Contrast

The synaptic terminals of cones contain a number of invaginations where contact is made not only with bipolar cells, but also with horizontal cells. Each human horizontal cell makes approximately 7-36 cone contacts, depending on retinal eccentricity (Anhelt & Kolb, 1994). Hyperpolarization of cones causes the horizontal cells to hyperpolarize; these cells provide feedback onto the cone in a sign-inverted manner (depolarization). Such negative feedback onto a single cone may be based on inputs from 3-4 horizontal cells. Weak signals from only a few cones will not be sufficient to hyperpolarize the horizontal cell, so inhibitory influences on cones will be evident only when many adjacent cones are activated. The net effect is that a small, brief flash of light will produce a stronger response from a cone than a diffuse flash or a small increment on a background. Thus, the horizontal cell shapes the cone response to emphasise spatial and temporal contrast while diminishing the response to uniform and continuous stimulation.

Two types of horizontal cell have been identified physiologically in the primate retina.[4] H1 cells receive input from both L and M cones, but not S cones. H2 cells connect all three cone types (Dacey *et al.*, 1996). Because there are so many more M and L than S cones, the net effect is that H2 cells have a strong influence on S cones, but S cones influence M and L cones much less. Cells of each class form a separate mosaic via gap junctions with like-type horizontal cells across wide regions of the retina. In addition to contacting cones near their cell bodies, the horizontal cells have a long axonal branch that terminates diffusely on several hundreds of rods. Because the horizontal cell responds only with graded potentials, and because the axon is so long, the rod and cone inputs to horizontal cells are believed to remain functionally separated.

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[4] Anatomical criteria suggest three horizontal cell classes (Ahnelt & Kolb, 1994). The H1 (little S-cone input) and H3 (no S-cone input) anatomical types correspond to H1 identified physiologically. The H2 type is classified both physiologically and anatomically as the only type receiving strong S-cone input.

The horizontal cells provide gain control of cone signals to bipolar cells, reducing the effect of the cone signal by a factor that is proportional to the overall stimulation of the horizontal cell. Note that because the horizontal cells receive input from more than one cone type, the activity of a particular cone class would be expected to affect the gain not only in that cone type but in other cone types with which it is connected. This may be why the π-mechanism sensitivities are broader than cone action spectra. Werner and Walraven (1982) have proposed that the von Kries coefficients that describe receptor scaling in chromatic adaptation are controlled at an adaptation site having the sensitivity of the π-mechanisms. Horizontal cell circuits provide a candidate mechanism to explain two-colour increment thresholds but a link with π-mechanisms may seem unlikely in view of the fact that the sensitivity of the π-mechanisms involves cone-opponent interactions (Pugh & Mollon, 1979). Note, however, that while the inputs to the horizontal cell are all of the same sign, the output of the H2 cell is subtractive or divisive such that the S-cone signal is reduced in part on the basis of the sum of M- and L-cone signals.

9. Bipolar Cells and the Coding of Light Increments and Decrements

Bipolar cells extend from the outer plexiform layer where they receive their inputs from photoreceptors to the inner plexiform layer where they contact ganglion cells. Of the variety of bipolar cells that have been identified, three general classes are of special interest for colour vision: (a) The midget bipolar which in the fovea receives input from a single M or L cone; (b) the diffuse bipolar which receives input from 5-10 foveal cones, contacted indiscriminately with respect to cone type; and (c) the S-cone bipolar that receives input from one or more S cones (Mariani, 1984).

Although the cones have only one type of neurotransmitter (glutamate), they can make contact with bipolars through two types of synapses (Boycott & Wässle, 1991), one which is sign-conserving and one which is sign-inverting, as illustrated by Figure 9. Thus, an increase in photon absorption by the receptor will depolarize one bipolar (the ON type) and hyperpolarize the other bipolar (the OFF type). The ON- and OFF-bipolar cells make synaptic contact with ganglion cells in separate sublaminae of the inner plexiform layer; this stratification provides both functional and anatomical segregation of incremental and decremental neural signals resulting from the same photopigment isomerizations. Because there is no further

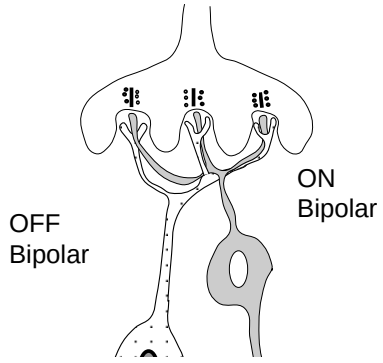

Fig. 9 Synaptic contacts are made between a single cone and two midget bipolar cells. One bipolar has central invaginating processes that contact the cone pedicle below the synaptic ribbon. Semi-invaginating processes from the second bipolar have branches that surround the first bipolar's central process. These two types of contact result in parallel signals of opposite polarity when the receptor responds to light.

sign inversion, ganglion cells receiving input from ON bipolars become ON ganglion cells and those receiving input from OFF bipolars become OFF ganglion cells. The terms ON and OFF are misleading in that these cells respond to *changes* in light level across space and time, not just the presence or absence of light. The ON and OFF cells code the local contrast, incremental and decremental changes in light, respectively.

The bipolar cell signal to the ganglion cell can be modulated by lateral connections from amacrine cells. There are many types of amacrine cells, and each type has numerous branches. When bipolars activate an amacrine cell, it may send a sign-inverted signal to the ganglion cell and sometimes back to a bipolar cell. In its simplest form, such a circuit would work much like a horizontal cell circuit to make the cell more selective in its response to stimuli with greater spatio-temporal contrast. Amacrine cells may provide other types of feedback circuits as well, but they are poorly understood, especially in the primate retina.

The functional segregation of ON and OFF signals at the bipolar level is preserved in the visual pathways through subsequent stages up to the visual cortex. Animals can be tested using psychophysical methods to show how their behaviour changes when the ON pathways are suppressed as the result of intraocular infusion of a chemical, 2-amino-4-phosphonobutyrate (APB). Schiller (1995) has used this approach with monkeys that are trained to fixate a central light and then to shift their gaze toward a spot that is turned on or off in some peripheral location. Normally, the accuracy and the response times (latencies) are about the same for detecting increments and decrements of light relative to a background. During APB infusion, latencies increase for detection of incremental stimuli and the proportion of those incremental stimuli detected is reduced. There is no significant change in the detection of decremental stimuli. Thus, the parallel pathways formed in the outer layers of the retina and preserved to the cortex appear to have a direct influence on perception of brightness and darkness.

10. Retinal Ganglion Cells and their Projections to the Thalamus

The input to ganglion cells is based on graded potentials from bipolar and amacrine cells. The midget ganglion cells, which constitute about 70% of the ganglion cell population, receive their input from midget bipolar cells. In the fovea, one midget ganglion cell will contact one midget bipolar, but in the periphery a midget ganglion cell receives input from more than one midget bipolar. Diffuse bipolars contact parasol ganglion cells which make up about 10% of the ganglion cell population. There seems to be extensive coupling between the parasol cells, most likely via amacrine cell connections. Finally, S-cone bipolar cells contact small bistratified ganglion cells with a distinctive morphology (Dacey & Lee, 1994; Calkins *et al.*, 1998b). Because of their different classes of inputs, there are several classes of ganglion cell which provide the output from the retina. These signals are coded in terms of action potentials, a digital code carried by the axons of each ganglion cell.

The dendritic field size of all ganglion cells increases with retinal eccentricity, implying that the more peripheral ganglion cells collect their inputs from wider regions of retina. The spread of the dendritic fields for parasol and midget ganglion cells is sufficient to form overlapping networks, or mosaics, that cover the entire retina. It has been shown in the cat that the main ganglion cell types form separate

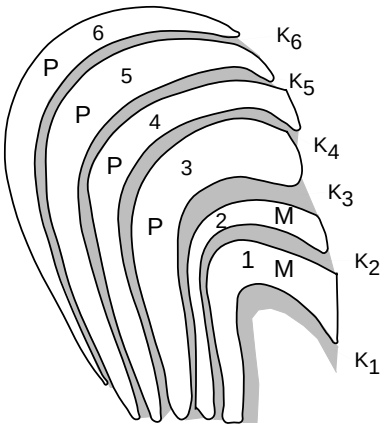

Fig. 10 Cross section of the macaque LGN. The principle LGN layers contain parvo- (P-) and magno- (M-) cells. Koniocellular (K) layers contain tiny cells. Layers M2, P3, P5, K2, K3 and K5 receive input exclusively from the ipsilateral eye; layers M1, P4, P6, K1, K4 and K6 are innervated only by the contralateral eye. Each layer contains a retinotopic map of one hemifield from one eye. The retinotopic maps of each layer are in register such that a vertical penetration through the LGN would encounter cells representing the same point in the visual field. (After Hendry & Calkins, 1998)

ON and OFF mosaics, each of which has sufficient dendritic spread to sample the entire retina (Wässle *et al.*, 1983).

Axons of retinal ganglion cells form the optic nerve which sends retinal signals to several regions of the brain. The main projection site of the retinal ganglion cells for processing of chromatic signals is to the two lateral geniculate nuclei (LGN) of the thalamus. The LGNs are multilayered structures that segregate their inputs according to cell type and according to the eye of origin, as shown in Figure 10. Midget ganglion cells send their axons to dorsal layers of the lateral geniculate nucleus (P3-P6), while parasol ganglion cells send their axons to ventral layers (M1- M2). The small cells in the dorsal layers are called parvo (P cells), while the larger cells in the ventral layers are called magno (M cells). The P- and M-cell distinction also applies to the retinal ganglion cells, but note that P designates the midget ganglion cells, while M designates the parasol ganglion cells.

Ventral to the principal layers of the LGN (M and P layers) are tiny cells that form the koniocellular (K cell) layers. Cells in these layers do not stain well by conventional techniques, but they express two proteins not found in M and P cells (αCaM II kinase and calbindin), making it possible to label them (Hendry & Yoshioka, 1994). The small bistratified ganglion cells carrying S-cone signals are believed to project to K3 (Calkins *et al.*, 1998a).

11. Receptive Fields of Retinal Ganglion and LGN Cells

The functional properties of cells in the visual pathway can be measured by recording their electrical activity when light is placed on different regions of the retina. That area of the retina influencing the activity of a cell defines that cell's receptive field. Ganglion and LGN cell receptive fields are essentially identical, although LGN recordings have the advantage of easier identification by layers and, therefore, identification by cell type.

DeValois *et al.* (1958) reported the first evidence concerning the chromatic properties of receptive fields of single cells in the primate visual system. In the "middle laminae" of the LGN, cells were found with ON or OFF responses depending on the wavelength of the stimulus. An example of such a cell is shown in Figure 11. This cell is said to have an opponent chromatic organisation because it is

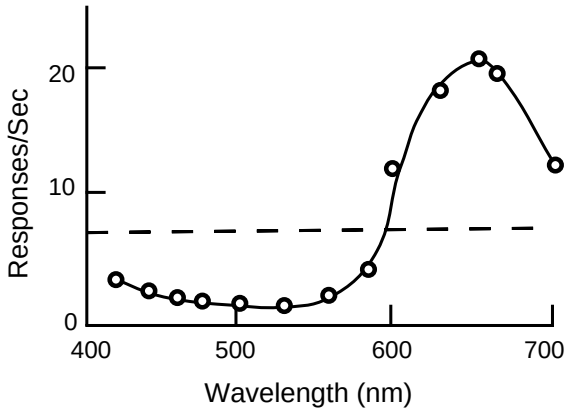

Fig. 11 Mean number of responses (action potentials) per second from a macaque monkey LGN cell as a function of wavelength. The dashed line shows the spontaneous discharge of the cell in the dark. Points above the dashed line are due to excitatory inputs and points below the dashed line are due to inhibition. The wavelength of crossover between excitation and inhibition is often called the cell's neutral point. (After DeValois *et al.*, 1966.)

excited by some wavelengths, inhibited by others, and produces only the baseline level of responding for the neutral point or crossover point. This cell is also opponent in the sense that a mixture of short and long wavelength light (or a broadband, white light) falling on its receptive field causes no change in maintained discharge (*i.e.*, the inhibition from the short-wave light cancels the excitation from the long-wave light). About 70% of the LGN cells encountered by DeValois *et al.* (1966) could be described as opponent, although they differed in the location of their neutral point or had regions of excitation and inhibition opposite to those shown in Figure 11. The remaining 30% of the cells were described as nonopponent or broadband; these cells showed varying degrees of excitation (or inhibition) to different wavelengths, yielding an action spectrum similar to the V_λ function (or its inverse). Ganglion cell receptive fields with similar chromatic opponency were later described by Gouras (1968).

Comparison of the cone sensitivity functions (Figure 7, right) and the cell response in Figure 11 suggests that the latter combines inhibitory input from S and/or M cones and excitatory input from L cones. In many cases, however, the cone inputs to a cell are not clear because one particular cone type may have a strong input that obscures the weaker input from another cone type. Chromatic adaptation was also used by DeValois *et al.* to characterise the cone inputs more fully using the same logic as psychophysical experiments with two-colour increment thresholds. As in psychophysics, this approach can provide useful information but does not always reveal the full identity of the cone inputs.

If small spots are used to map the receptive fields of ganglion or LGN cells, a spatially antagonistic organisation such as that shown in Figure 12 (left) is often found (Wiesel & Hubel, 1966). In this example, a spot of light in the receptive field centre leads to an increase in firing of the cell, while light in the surround leads to a decrease in firing. The cell is said to display a centre-surround antagonism, and the response profile (shown by a bold curve) can be modelled as the difference of two Gaussian distributions with different space constants and opposite polarity (excitation from the centre and inhibition from the surround). Note that both Gaussian curves peak in the same spatial position, implying that some of the same photoreceptor(s) contribute to the centre and the surround. As would be predicted from the summing of its excitatory and inhibitory inputs, the cell responds poorly to uniform illumination. The centre and surround of the receptive field are derived

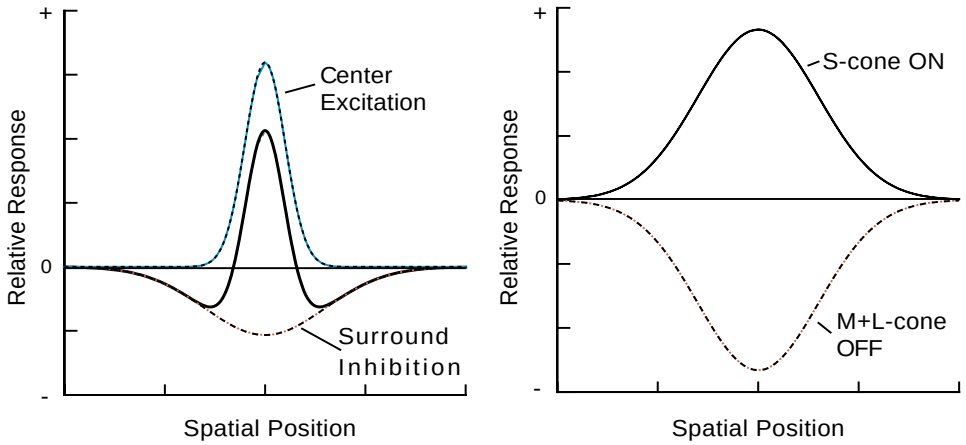

Fig. 12 Two-dimensional receptive field profiles for a midget ganglion cell (left) and a small bistratified ganglion cell (right).

from bipolar inputs; in this illustration the centre is driven by an ON cell, but about half the ganglion cells receive inputs from OFF bipolars and therefore have OFF-centre receptive fields (inverse profiles to that shown in Figure 12 left).

Both P and M cells have a centre-surround organisation. In the fovea, where midget bipolar cells receive input from a single cone, the ganglion cells that they contact have receptive fields that are driven by a single M or L cone. The surround receives inputs from M and L cones; S cones may also be involved but their numbers are so low that their effective input is difficult to measure. The radii of receptive field centres of M cells are about 2-3 times as large as those of P cells at any given eccentricity (Croner & Kaplan, 1995) because their input from diffuse bipolars carries combined signals from more cone photoreceptors. In addition, the antagonism from their surrounds is weaker than for P cells.

Coextensive excitatory and inhibitory regions have been found for many cells receiving input from S-cones (Wiesel & Hubel, 1966), as illustrated by Figure 12 (right). The response of such a cell increases when short-wave light falls within its receptive field (due to input from S-cones via ON bipolars) and decreases for middle and long-wave light (due to inputs from M- and L-cones via OFF bipolars). These receptive fields are associated with the small bistratified ganglion cell (Dacey & Lee, 1994). Their receptive fields are relatively large (~0.3-to 2.0^{o}) compared with L/M- opponent cells (~0.08^{o} to 0.25^{o}) at comparable retinal eccentricity.

11.1. Koniocellular Layers

Both Wiesel and Hubel (1966) and Malpeli and Schiller (1978) noted that cells with S-cone input were typically found in the lower parvocellular sublaminae close to what are now identified as the koniocellular layers. Martin *et al.* (1997) recorded chromatic responses in LGN cells of the marmoset using chromatic and luminance modulation. Their data suggest that S-ON cells are found primarily, if not exclusively, in the K-layers. As expected, M/L opponent cells were identified in the parvocellular layers.

11.2. Cone Inputs to Receptive Fields Deduced from Silent Substitution

The silent substitution method described for isolating the response of a single class of cones can be extended to postreceptoral combinations of cone inputs. Derrington *et al.* (1984) used modulation of a field in specific directions of colour space that include: (a) an axis in which luminance varies and chromaticity is constant, (b) an axis of constant S-cone stimulation and (c) an axis of constant M/L-cone stimulation (tritan line). For a cell that combines cone inputs linearly, there is an axis of maximal response and a plane orthogonal to it containing all lights that can be exchanged silently. This null plane provides the cell's "signature" and can be used to deduce the input from the various cone classes.

Derrington *et al.* described two groups of P cells: cells that received opposed, but not equally balanced inputs from M and L cones, and cells with S-cone input that had almost equally opposed input from varying combinations of M and L cones. Subgroups of each of these classes imply the following groups of P cells:

+M -L	-M +L
+L -M	-L +M
+S -(ML)	-S +(ML)

P cells fit remarkably well into these categories and the range of "neutral points" (see Figure 11) was narrow compared to what has been reported for receptive fields mapped with monochromatic lights. Surprisingly, Derrington *et al.* could not identify a group of cells that might form the substrate for a 'luminance' mechanism (*i.e.*, M+L), for all P cells were opponent in nature. This suggests that chromatic and achromatic signals are multiplexed for transmission along the same P-cell fibres (DeValois & DeValois, 1993; Lennie *et al.*, 1993).

Derrington *et al.* found that M-cells were opponent both spatially and (weakly) chromatically. At higher temporal frequencies, however, Lee *et al.* (1988) found null-responses for equal-luminance stimuli (Lee *et al.*, 1988). This has been interpreted as evidence that M-cells provide the substrate for a V_λ-type function measured by heterochromatic flicker photometry (see Figure 4).

12. Chromatic Detection and Discrimination

This chapter began with a consideration of colour mixture. Trichromatic mixtures are based on the trivariance of cone photoreceptors. If two physically different patches of light produce indistinguishable responses in the photoreceptors, no later stage can recover the differences. The nearly converse situation occurs when two physically different fields of light produce different signals in the cone photoreceptors. If these differences are not lost in signals at subsequent stages of neural coding, discrimination is possible. Although neural signals carrying colour information undergo additional transformations in the cortex (to be considered in the next chapter), postreceptoral mechanisms in the retina and LGN appear to account for many aspects of colour discrimination.[5]

Krauskopf *et al.* (1982) measured chromatic detection thresholds following adaptation to stimulus modulation around a white point along either an axis of S-cone modulation (with constant M+L cone stimulation) or an axis of M/L-cone

[5] When psychophysical performance is modeled in terms of low-level (subcortical) mechanisms, decision processes (presumed to be cortical) are not considered.

modulation (with constant S-cone stimulation). Adaptation to one axis of chromatic modulation increased colour detection thresholds for stimuli on that axis, but not to stimuli on the orthogonal axis. This would seem to imply that threshold elevation under these conditions (but not necessarily all conditions; see Webster & Mollon, 1994; D'Zmura & Knoblauch, 1998) is due to independent mechanisms similar in sensitivity to the P cells described by Derrington *et al.* (1984).

Studying colour discrimination with stimuli that isolate physiological mechanisms is a relatively new approach. Historically, colour discrimination has been quantified in terms of the physical (wavelength) difference between two monochromatic lights that is just detectable, when the brightness of the two lights is equated (Wright & Pitt, 1935). Figure 13 presents hue discrimination data measured in this way but analysed in terms of postreceptoral mechanism sensitivities. Note that the threshold for discrimination of wavelength varies from about 1 nm at 580 nm to about 23 nm at 650 nm. Reference to Figure 7 suggests a reason for these large differences in threshold. A mechanism comparing M and L cone signals (L-M) will be stimulated about equally by a 1 nm change around 580 nm as a 23 nm change around 650 nm. Contours in Figure 13 represent the wavelength difference required for different postreceptoral mechanisms to produce a criterion change in response as a function of wavelength.[6] It is assumed that a difference between two wavelengths will be detected when one of those mechanisms reaches threshold. As a result, an observer's data should be fit by the lower envelope of the combined discrimination contours (*i.e.*, performance in a particular spectral region will be due to the most sensitive mechanism in that spectral region). As the figure shows, mechanisms with properties similar to those of P cells in the retina and LGN can account reasonably well for wavelength discrimination.

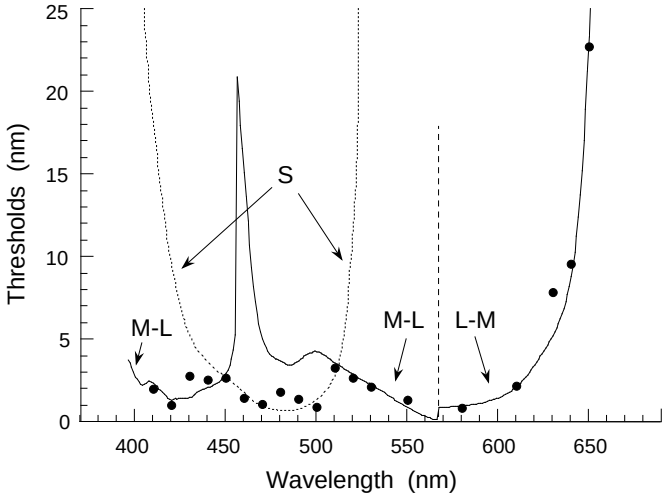

Fig. 13 The minimum difference between two wavelengths required for discrimination is plotted as a function of the wavelength of the standard. Smooth curves are model fits to the data using the mechanisms indicated in the figure. (After Shinomori & Werner, 1997).

[6] A simplification made here is to treat the absolute sensitivity of mechanisms as a free parameter. This is because we do not know how groups of cells combine to mediate psychophysical performance. Lennie (1993) has estimated that psychophysically-measured chromatic discrimination is about a factor of seven better than that of single P cells, but this factor is likely to depend on the specific stimulus conditions.

13. Summary

A person with normal colour vision can match any spectral distribution of light with an appropriate mixture of three primaries. The match with such a mixture occurs because the activity in each of the three classes of photoreceptor is identical for the two different fields. This is possible because of the principle of univariance: once a quantum of light produces an isomerization of a photopigment molecule, all information about the wavelength causing the reaction is lost. If the signals at this level are indistinguishable, the match will hold through subsequent processing by later (even nonlinear) stages; signals from the matched fields pass through the same stages making the match transparent to further transformations.

Bipolar cells separate the response of the photoreceptors, giving rise to parallel ON and OFF pathways. Details were discussed in relation to the midget bipolar system, but the diffuse bipolars also have ON and OFF circuitry. As a result, within the ON and OFF division, there is further parallel processing according to the M and P pathways. These pathways are segregated functionally and anatomically up to the cortex.

Ganglion and LGN cell receptive fields have varying degrees of centre-surround antagonistic organisation, shaped by their direct connections from photoreceptors via bipolar cells and by lateral influences from horizontal and amacrine cells. The S-cone ON signals are processed by a specialised ganglion cell, the small bistratified cell with a coextensive receptive field arising from M+L OFF signals. These various postreceptoral circuits, in particular those of the P pathway, account for a wide range of psychophysical data concerning the detection and discrimination of colour.

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