HUMAN COLOUR VISION: 2. COLOUR APPEARANCE AND CORTICAL TRANSFORMATIONS

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ABSTRACT

Four terms (red, green, yellow, and blue) are necessary and sufficient to describe the appearance of hue. Parvo cells of the LGN respond maximally to modulation along either an S-(L+M) or an L-M axis, but few cells are tuned to intermediate directions. These axes do not correspond to the colour appearance axes. Colour appearance requires at least one more stage of processing, a stage that transforms the second stage axes in the cortex. Receptive fields in area V1 vary much more in their colour tuning than in LGN, and they have significantly more S-cone input. Thus, a major accomplishment of striate cortex may be rotation of the colour axes by integrating S-cone input. Major differences in chromatic properties of receptive fields between area V1 and areas V2 and V3 have not been reported. V4 cells, however, have large receptive field surrounds that may support colour constancy and promote segregation of figure and ground by colour. It seems unlikely that V4 is a "colour centre" in the monkey brain, and it is equally unlikely to be homologous with those regions of human cortex associated with achromatopsia.

1. Perception of Hue

The previous chapter provided an introductory account of colour matching and discrimination based on the organisation of neural signals in the retina and LGN. An observer could, of course, say much more about these stimuli if asked to describe what they look like. While our understanding of the neural mechanisms mediating colour appearance is incomplete, it is clear from what is known that hue depends on cortical transformations of the retino-geniculate signals. Receptor/colour-matching space is sometimes considered a first (functional) stage of colour processing, while a second stage is described by the S-(L+M) and L-M axes that characterise ganglion and LGN cell receptive fields. Colour appearance requires at least one more stage of processing, a third stage that is the subject of this chapter. Consideration of evidence for third stage mechanisms of colour appearance in visual cortex leads to a discussion of the interaction of colour processing with form and motion processing, colour constancy, cerebral achromatopsia and the role of consciousness in behaviour dependent on colour vision.

Consider again the stimulus described in the first figure of the previous chapter. Once the two half-fields are matched metamerically, the match will be maintained with increases in intensity (scalar invariance), addition of another light (additivity) and changes in sensitivity due to moderate chromatic adaptation (persistence law). Note, however, that even though the two fields match under these stimulus manipulations, colour appearance is likely to change. If the observer is asked to describe these changes, a variety of words might be selected due to somewhat arbitrary cultural and linguistic conventions. If, however, we limit the number of terms that an observer is offered, only four hue names (red, green, yellow, and blue) are found necessary and sufficient to describe all aspects of colour appearance (Hering, 1878, 1920). Thus, as shown experimentally, what we call orange can be described just as well with the terms red and yellow, while purple can be described

just as well with the terms red and blue. Because these perceptual qualities are derived from the way our nervous system codes colour, they are largely independent of culture (Abramov & Gordon, 1994).

Figure 1 shows colour-naming data for 1° monochromatic fields presented as foveal 1-sec flashes. Observers scaled the hue in terms of percentages of red, green, blue or yellow. For example, a light at about 610 nm that appears orange, might be described as 60% red and 40% yellow. After a little practice, observers scale hues in this way with high reliability (Boynton & Gordon, 1965). Squares show red or green responses from 0 to 100% on the left ordinate, while blue or yellow responses are plotted from 100 to 0% on the right ordinate. The data could be plotted in this way because observers, despite being free to use the terms red and green simultaneously or the terms blue and yellow simultaneously, almost never did so. The arrows in Figure 1 denote the wavelengths of unique blue, green or yellow, *i.e.*, unitary hues that cannot be described by combinations of any other hue terms.[1]

Hering (1920) attached special significance to the observation that while we can experience red in combination with yellow or blue, we do not experience it in combination with green at the same time and place. Similarly, blue and yellow are not experienced cotemporally and cospatially. The experiences of red and green cancel each other, as do the sensations of blue and yellow. Hering, therefore, called these paired colours, opponent hues. He further proposed that opponent-hue sensations were due to corresponding opponent-physiological processes with two modes of response, corresponding to the modern terms, excitation and inhibition. Thus, according to Hering, red-green and blue-yellow opponent physiological processes account for all the hues experienced by human trichromats.

A complete description of colour appearance must also include the achromatic or black-white components of a colour. For example, pink and maroon look quite different even though both have red as the dominant hue component. The difference arises because pink also contains a prominent whiteness component while maroon has a prominent blackness component. To account for the achromatic dimensions,

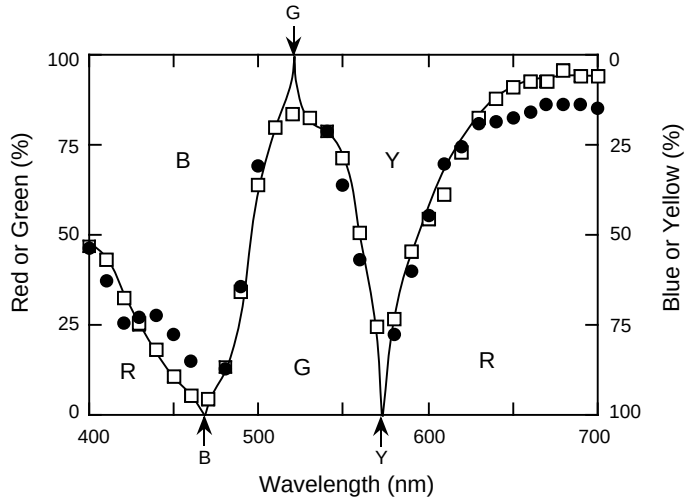

Fig. 1 Hue-naming data (squares) and modelled hue-naming data (circles) based on opponent-cancellation functions plotted as a function of wavelength. All data are means from the same three observers. Red or green percentages are plotted (left) opposite to the blue or yellow percentages (right). Unique hue wavelengths are denoted by arrows. (After Werner and Wooten, 1979.)

[1] Unique red is extraspectral, meaning that it must be produced by a mixture of two monochromatic lights such as a short-wave (reddish-blue) and a long-wave light (yellow-red). The yellow and blue components cancel each other, leaving only the red.

Hering postulated a black-white process. Thus, all colour experiences in Hering's theory are based on six elemental sensations (red, green, yellow, blue, black, white) coded in the nervous system by three opponent processes.[2]

Now consider what happens when the intensity of a chromatic field is increased. While colour matching obeys the scalar invariance law of Grassmann, hue appearance usually does not. Rather, as intensity is increased, the blue or yellow hue component increases relative to the red or green component. This change is known as the Bezold-Brücke hue shift. Hering took this as further evidence that yellow and blue are linked in the nervous system, as are red and green. One interpretation of the Bezold-Brücke effect is that the two opponent-physiological processes have different response vs. intensity functions, with a steeper slope for blue-yellow than for red-green (Hurvich & Jameson, 1958). Purdy (1931) quantified this effect and also reported that the wavelengths of the spectral unique hues were invariant with changes in intensity.

Hues can also be subjected to tests of additivity; as described in Chapter 1. These tests are carried out by superimposing a light on each half of a bipartite field to demonstrate the linearity of colour matching. Although the two fields continue to match with the added light, colour appearance is usually altered. The changes in colour appearance are predictable from the opponent coding of hue. This was quantified by Jameson and Hurvich (1955), who measured the amount of a cancelling light required to just eliminate its opponent hue in a series of monochromatic lights. For example, by adding a stimulus that looks green to a light that has a reddish component, one can find an amount of green that just cancels the red and the resultant appearance is neither red nor green. It can be assumed that the strength of the red response (or valence) at a particular wavelength and luminance level is proportional to the energy or number of quanta in the superposed green light required to reach this equilibrium state. Similarly, Jameson and Hurvich measured the energy of a yellow (or blue) light necessary to cancel blueness (or yellowness). Figure 2 shows data obtained for wavelengths between 400 and 700 nm using this hue-cancellation method. Blue and yellow are plotted with opposite signs, as are red and green; note that red occurs at both short and long wavelengths. This convention in plotting is in keeping with the cancellation operation, and also with Hering's idea that opponent hues are based on antagonistic modes of neural response.

If these opponent-cancellation functions are based on physiological processes mediating the perception of hue, then they ought to be related to hue naming. Following an equation of Hurvich and Jameson (1955), circles in Figure 1 show the ratio of red-green or blue-yellow chromatic response, at each wavelength, relative to total chromatic activity (red-green plus blue-yellow) using the same data presented in Figure 2. The two sets of measures agree quite well, consistent with the idea that these opponent processes are part of a neural network involved in colour appearance.

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[2] Space limitations preclude a more complete discussion of the achromatic colours, but see a recent review by Volbrecht and Kliegl (1998).

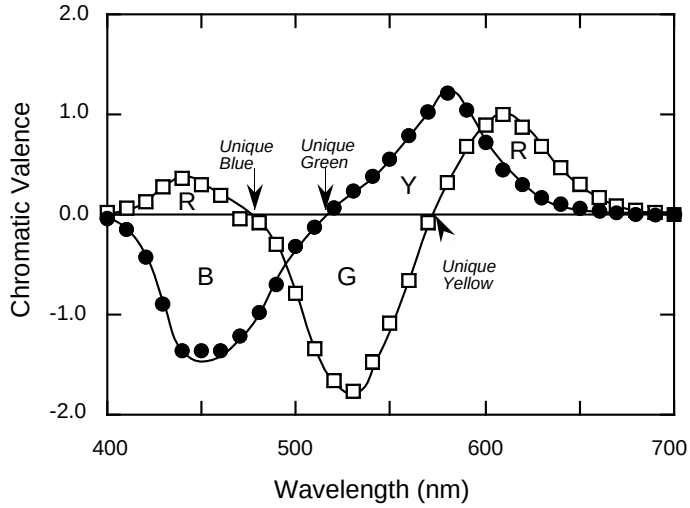

Fig. 2 Opponent-hue valence determined by cancellation is plotted as a function of wavelength. Red-green is shown by squares, with red as positive and green as negative. Blue-yellow is shown by circles, with blue as negative and yellow as positive. (After Werner & Wooten, 1979.)

The cancellation functions shown in Figure 2 resemble responses of some single cells in the LGN (see Figure 11 of the previous chapter). These cells are not, however, good candidates for the physiological substrate of perceived hue. One line of evidence that the cancellation functions are not mediated by the LGN cells is that LGN cells combine inputs from photoreceptors linearly. In contrast, the blue-yellow process has been shown to combine cone signals nonlinearly (Larimer, Krantz & Cicerone, 1975; Werner & Wooten, 1979). This is why unique green is often not scalar invariant (Schefrin & Werner, 1990); that is, the wavelength that appears unique green at low intensities is not the wavelength that appears unique green at high intensities. The red-green process does not exhibit striking nonlinear behaviour, but some examples may be found in the literature (Ayama *et al.*, 1987). Furthermore, the loci of LGN-cell neutral points (Derrington *et al.*, 1984) differ from the crossover points of the cancellation functions (which agree with the unique hue wavelengths). An additional complication is that a triphasic spectral response function, with substantial amplitude at short wavelengths, is required to explain the redness of short-wave lights (Wooten & Werner, 1979), but is seldom observed in ganglion and LGN cells (DeMonasterio & Gouras, 1977). Indeed, electrophysiological studies have not identified cells at any anatomical level that produce response functions that resemble the cancellation functions in detail. This is perhaps because the cancellation functions are due to the combined activity of a cell network.

The mismatch between the retina/LGN axes and hue mechanisms was demonstrated with a colour-naming study by DeValois *et al.* (1997b). They presented stimuli that were modulated from a white point along the colour axes shown in Figure 3. As described in the previous chapter, Derrington *et al.* (1984) used this colour space when mapping LGN receptive fields and found that these cells respond best to modulation along either the S-(L+M) or L-M axes. Along the former axis, only the S-cone stimulation varies, while along the latter axis there is covariation of L- and M-cone stimulation in a way that keeps their sum constant. DeValois *et al.* selected their stimuli from the same isoluminant plane (the S+M+L axis) so responses would be based on chromatic, not luminance, differences. If mechanisms mediating hue perception are Stage 2 mechanisms found in the LGN,

one might expect maximal hue responses, or unique hues, for red, yellow, green and blue to coincide with the 0°, 90°, 180° and 270° positions, respectively. Instead, they found that the coordinates defining hue naming were shifted by varying amounts from the retinal/LGN axes.

Based on the results shown in Figure 3, hue coding appears to require at least one more stage of processing beyond the LGN. Put another way, between the LGN and the sites mediating colour perception, the colour axes must be rotated. The next sections will explore physiological processes that may account for this neural transformation.

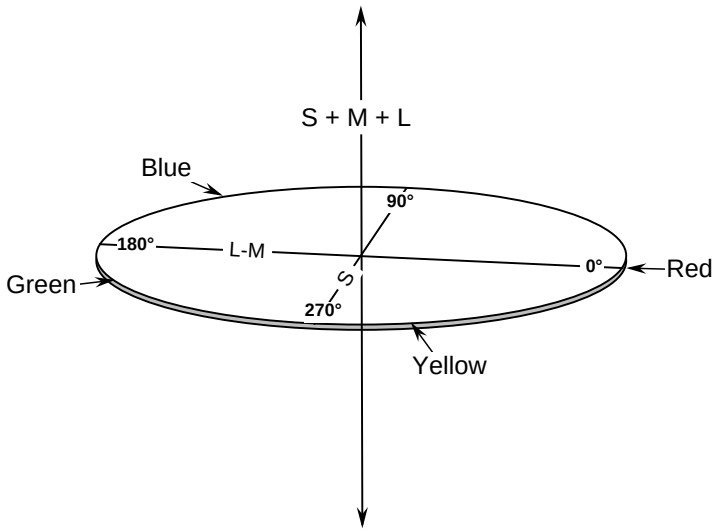

Fig. 3 Colour space of Derrington *et al*. (1984) having three orthogonal planes defined by mechanisms that combine cone inputs linearly (Stage 2): an axis of differential L-M stimulation (constant S-cone stimulation) between 0° and 180°, an axis of S-cone modulation (constant L- and M-cone stimulation) between 90° and 270°, and an achromatic axis (S+M+L). Arrows denote colour vectors receiving maximum hue responses by DeValois *et al*. (1997b). Note that the hue positions are off the Stage 2 axes.

2. Projections from LGN to Cortex

Thirty-two anatomically distinct visual areas have been identified in monkey cortex, of which 25 are primarily visual and 7 are polysensory or involved in visually-guided motor control. Fortunately, the initial cortical connections have been accessible to study by experimental methods. Figure 4 shows the cortical areas in the macaque monkey that will be discussed in this chapter. There are important differences in the cortical circuitry of the visual areas among primate brains (Casagrande, 1994), and it is not yet clear how well the macaque will ultimately serve as a model of human vision. However, given the similarity between macaque and human colour vision shown by psychophysical experiments (DeValois *et al.*, 1974), the macaque seems to provide an excellent starting point.

Most LGN fibres project to cortical area V1 which maps the LGN input retinotopically; each point on the retina is represented by a discrete region of LGN which projects to a discrete region of cortex. Neighbouring points on the retina are thus represented by neighbouring regions of cortex. There is, however, a magnified representation of central retina (the central 7° of retina is represented by about 50% of striate cortex). This retinotopy is preserved in a second map of visual space in area V2. A third retinotopic map is split between two areas, ventral posterior (VP) which represents the upper visual field and V3 which represents the lower visual

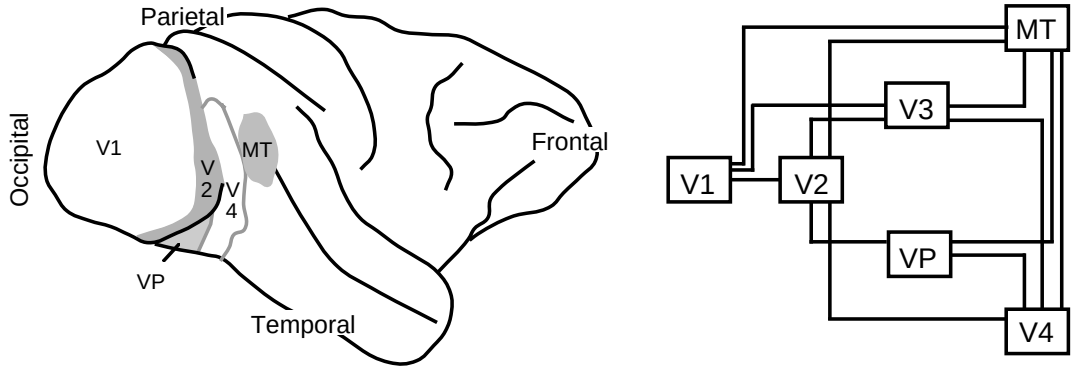

Fig. 4 Location of select visual areas and the major lobes (occipital, parietal, temporal, frontal) of the right hemisphere of macaque cortex. Cortical area V3 cannot be seen from this view. The schematic on the right shows some of the major connecting circuits among visual areas that are of concern in this chapter; visual areas 1 (V1), 2 (V2), 3 (V3), 4 (V4), ventral posterior (VP) and medial temporal (MT). (Modified after Maunsell, 1995.)

field.[3] The fourth visual area (V4) and the medial temporal (MT) area, also called V5, may be specialised to analyse visual information in a way that is less dependent on spatial position, and the retinotopy here is not as strict as in the other visual areas. The difficulty in discerning the retinotopic organisation in these higher areas is based, in part, on their large receptive fields. As a general rule, receptive fields become larger at higher levels in the pathways.

The size of the cortical regions generally becomes smaller at higher levels, although it is not possible to see this without flattening the convoluted cortex. Using data from Felleman and Van Essen (1991), the sizes of the areas shown in Figure 4 may be expressed relative to V1: V1 = 1.0, V2 = 1.06, V3 = 0.11, VP = 0.08, V4 = 0.48 and MT = 0.05.

The functional organisation of the cortex is partially determined by the segregation of inputs from the LGN to area V1. This area is also called striate cortex because of the distinctive white stripe (the stripe of Gennari) created by the myelinated fibres that enter layer IV of the six cell layers of cortex. Layer IV is a large layer within area V1 and has distinct subdivisions as illustrated in Figure 5. Parvocellular (P) axons from the LGN project to the lower half of layer IV (IVCβ) and to IVA. They also have a minor projection to layer I and the upper region of layer VI. Layer IVCβ has output projections to the lower part of layer III and from there to layer II and the upper sublaminae of III. Magnocellular (M) axons from the LGN project to layer IVCα and have a minor projection to the lower region of layer VI. From layer IVCα there is a projection to layer IVB. The main outputs of this layer are to the second (V2) and third (V3) visual areas and then to the middle temporal (MT) area, which appears to be specialised for processing motion signals. Beyond layer IV, most of the M and P circuits interconnect different striate layers and then different cortical regions, although the M and P streams are less strictly segregated than at the level of the LGN.

[3] VP cortex is sometimes included with V3 so that the latter area includes one complete visual field representation. The VP term was introduced to recognise that the superior field representation does not receive direct V1 input and because there are other anatomical differences between these two regions.

All cells contain mitochondria which can be stained by the mitochondrial enzyme, cytochrome oxidase (CO). Higher CO staining is associated with cells having higher metabolic activity. Throughout V1, there are regularly-spaced regions that stain densely for CO; they appear as slightly irregular ovoids, about 150 x 200 μm, called blobs (Livingstone & Hubel, 1984). They are most apparent in cortical layers 2 and 3, but are aligned with fainter CO regions in layers V and VI, as illustrated by Figure 5. It is worth noting that blob borders are not sharp, nor are they clearly delineated by changes in axonal or dendritic patterns. The koniocellular (K) layers of LGN project directly to layer III blobs (Fitzpatrick *et al.,* 1983; Calkins *et al.*, 1998). There are also intracortical connections to the blobs that provide inputs from both M and P pathways. It has been suggested that blob cells may be specialised for the analysis of colour, although we shall consider alternative interpretations.

The output of striate cortex is primarily from the upper layers (II, III, IVA and IVB) to extrastriate cortex, most prominently to cortical area V2. Outputs from the lower layers go to deep structures; layer V projects to the superior colliculus, while layer VI projects back to the LGN.[4]

3. Chromatic Properties of V1 Receptive Fields

In addition to the laminar organisation of cortex, there is also a columnar organisation such that cells above and below each other often have common properties such as the representation of overlapping positions in visual space. A large proportion of cells in striate cortex have receptive fields with spatially antagonistic excitatory and inhibitory regions elongated along a particular orientation, making them selective in their response to bars or edges with a particular orientation. A vertical electrode penetration reveals that cells within a column have receptive fields with similar preferred orientations. In addition to these

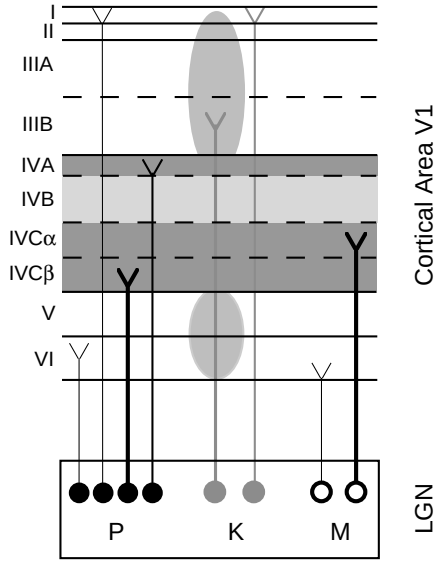

Fig. 5 Some of the main geniculo-striate connections of the macaque monkey. P, K and M represent parvo-, konio- and magno-cellular layers of the LGN, respectively. Divisions and subdivisions of cortical area V1 (also known as area 17 or striate cortex) follow the terminology of Brodmann. Grey levels are related approximately to the density of staining obtained with the mitochondrial enzyme cytochrome oxidase. The differential staining in layers I-III and V-VI define the "blobs."

[4] There are as many fibres feeding back from cortex to LGN as there are feedforward connections. The function of cortical feedback on the LGN is not understood.

orientation columns, there are columns that are dominated in their response by input from one eye, called ocular dominance columns. LGN cells entering layer IV are segregated by eye of origin so that there are alternating inputs from each eye. The CO blobs are centred within the ocular dominance columns. The orientation and ocular dominance columns are thought to be arranged orthogonally to each other so that each region of visual space is subserved by a module in area V1 that has a set of two ocular dominance columns (one from each eye and each having a CO blob) and about 18 orientation columns. Hubel and Wiesel (1977) called these cortical modules, hypercolumns.

A hypercolumn contains all the machinery necessary to subserve vision for a small region of visual space. In addition to providing a functional architecture for orientation and ocular dominance, cells within these hypercolumns encode other dimensions such as colour, size, and motion. The receptive field profile for one stimulus dimension sometimes depends on the characteristics of the other dimensions, making it difficult to characterise fully a cell's precise tuning characteristics. When they tested striate cortex cells with chromatic stimuli, Hubel and Wiesel (1968) found less than ten percent to be colour selective. Several other early studies (*e.g.,* Dow, 1974) also reported low proportions, but more recent studies (Thorell *et al.*, 1984; Lennie *et al.,* 1990) find the percentage of V1 cells that are colour selective to be much larger, perhaps more than 80%. The exact proportion depends critically on the criteria used to classify cells, and these criteria have varied in a number of important ways across investigators.

As in the LGN, receptive fields in area V1 can be classified as opponent or broad-band nonopponent when responses are measured with spectral lights. The polarity of the response varies in terms of ON and OFF as in the geniculate, but inhibition is more difficult to see in cortical cells due to their lower rate of maintained discharge. Action spectra reveal some important differences between cells in layer IVC compared to cells in the upper layers of area V1. For example, Dow (1974) and Gouras (1974) report that while IVC cells typically receive inputs from only M and L cones, the upper layer cells often have inputs from all three cone types. Ts'o and Gilbert (1988) also report more cortical cells with S-cone input than would be expected from P-cell recordings in the LGN. It is not clear whether the various recording methods were insensitive to S-cone input near the input layers or whether the S-cone inputs arrive by another path (K-projections?) and converge in the upper layers, which appear to contain a higher proportion of colour-selective cells. Dow also notes that some of the cells in the upper layers are further distinguished by the presence of triphasic receptive fields, having inputs from S and L cones opposed by M cones, as might be anticipated from opponent-cancellation functions for a red-green process (Figure 2).

3.1. Spatial Tuning

Hubel and Wiesel (1968) classified cells as simple or complex based on their response to broadband light (Hubel & Wiesel, 1968). Simple cells respond optimally to lines or bars having a particular orientation and position in the visual field. Complex cells also respond optimally to lines or edges of a particular orientation, but are insensitive to the location or phase of the light-dark areas. The responses of some complex cells are enhanced by motion in a particular direction and speed.

While the simple and complex distinction remains useful for some purposes, it is not the most general way to describe the pattern response of the visual system or of individual cells. From linear systems theory, it is known that any pattern can be described in terms of a set of sinusoidal basis functions (Fourier's theorem), with each sinusoidal component specified in terms of its frequency, amplitude and phase.[5] From this point of view, the visual system's or a cell's overall response to spatial-temporal patterns can be better characterised in terms of a contrast sensitivity function, the reciprocal of the amount of contrast required to produce a criterion response as a function of spatial or temporal frequency.

Figure 6 shows human spatial contrast sensitivity functions (CSF). The achromatic CSF has a band-pass shape; the visual system is most sensitive to a band of spatial frequencies in the middle of the range. The chromatic CSF is low pass; sensitivity is highest at low frequencies and decreases at middle and high frequencies. The high frequency limit for red-green chromatic modulation is similar to that for achromatic modulation, but that limit is typically much lower for blue-yellow chromatic modulation. This means that resolution of black-white and red-green equiluminant stimuli exceeds that with equiluminant blue-yellow stimuli. The sensitivity and poor resolution for blue-yellow stimuli are likely due to the sparse distribution of S cones.

Thorell *et al.* (1984) found that V1 receptive fields are selective for both luminance and chromatic modulation, with only a small proportion of cells responding exclusively to luminance (20%) or colour (1%). Most cells respond over

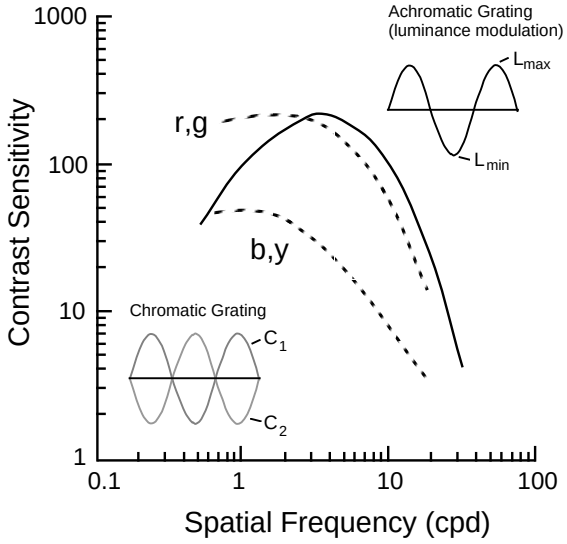

Fig. 6 Human spatial contrast sensitivity for achromatic gratings is shown by the black curve (after Robson, 1966) and by dashed curves for equiluminant red-green and blue-yellow chromatic gratings (after Van der Horst and Bouman, 1969). Insets illustrate achromatic (L refers to luminance) and chromatic (C refers to chromaticity) modulation as a function of spatial position.

[5] In the space domain, frequency is specified by the number of cycles per degree of visual angle, while in the temporal domain it is specified as cycles per second (Hz). Amplitude of an achromatic sine wave is described in terms of (Michelson) contrast, defined as: L_{max} - L_{min} / L_{max} + L_{max}, where L_{max} is the maximum luminance and L_{min} is the minimum luminance. (See inset in Figure 6.) For chromatic gratings, contrast is defined differently because luminance is held constant and the chromatic modulation is between two out-of-phase sinusoids that differ along a line in colour space. Different results might be expected depending on whether the stimuli are defined by a colour space based on individual cone mechanisms or a particular postreceptoral combination of cones. Phase is important for specifying the positional relations between sine-waves.

a narrow range of spatial frequencies for both chromatic and luminance modulation. When a cell differs in its tuning for the two types of modulation, the peak sensitivity for that cell is generally lower for chromatic than for luminance modulation. Such cells are simple cells with band-pass spatial tuning for luminance modulation and low-pass tuning for chromatic modulation. The population of striate cells as a whole obtained by Thorell *et al.* had tuning that was consistent with chromatic and achromatic spatial contrast sensitivities measured psychophysically in humans. Simple and complex cells are rather different in their spatial-chromatic tuning, however. Some complex cells respond in an opponent manner for chromatic and luminance modulation, but depart from classical opponency in being tuned to multiple spectral regions. For example, a particular cell might respond in the same manner to red, blue, or white bars. Thus, the responses of such individual complex cells do not discriminate colour or the sign of colour contrast.

3.2. Double-Opponent Receptive Fields

Hubel and Wiesel (1968) described cells with concentric centres and surrounds, in which each zone has a double-opponent organisation (Figure 7). For example, a cell may have a -M+L centre surrounded by an antagonistic zone of opposite polarity, +M-L. Such a cell responds selectively to the size and chromaticity of a stimulus, but it does not respond well to uniform chromatic or achromatic stimuli. A cell with the receptive field profile shown in Figure 7 should respond to long-wave stimulation of the centre in a way that resembles its response to middle-wave stimulation in the surround. It should be maximally excited by long-wave light falling on the centre *and* middle-wave light falling on the surround of its receptive field. Cells with double-opponent receptive fields seem to require significant transformations of LGN inputs at the cortical level.

A number of other authors have described double-opponent cells in macaque striate cortex (*e.g.*, Gouras, 1974; Dow, 1974; Michael, 1978). Ts'o and Gilbert (1988) found very few "true" double-opponent cells but a number of variations on the prototype shown by Figure 7. For example, some double-opponent centres had "silent surrounds" (*i.e.*, surround stimulation by itself did not affect the activity of the cell, but, when the centre was stimulated, the surround strongly inhibited the cell's response). Some investigators classify such cells as double opponent, but others do not. Perhaps this is why Lennie *et al.* (1990), using different methods that should nevertheless be sensitive to the identification of double-opponent cells, found none. This fundamental inconsistency in the literature remains to be resolved.

Double-opponent cells provide a means for explaining simultaneous colour contrast, the experience that the hue of a colour is strongly affected by surrounding hues. For example, a red spot will appear more red when surrounded by green and a

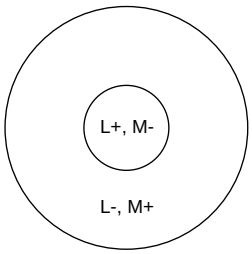

Fig. 7 Schematic of a double-opponent receptive field found in area V1. In this example, light falling on the centre causes excitation through the L cones and inhibition through the M cones. Light falling on the surround has an opposite effect on the response of the cell.

spot that appears white when viewed on a neutral ground may appear reddish when surrounded by green. Hering (1920) noted that such effects are expected if red-green and blue-yellow opponent processes are reciprocally linked across space. Through such linkage, neural activity in one region of the visual field tends to induce its opposite in surrounding regions. Thus, a link between double-opponent cells and simultaneous hue contrast seems straightforward.[6]

3.3. Blobs

Livingstone and Hubel (1984) reported that a large proportion of cells within blobs had concentrically-organised receptive fields with high chromatic selectivity. More than half these cells had chromatically-opponent receptive fields, while only about 17% of cells outside the blobs were chromatically opponent. Outside the blobs, cells had higher selectivity to orientation and size. Using different methods, Lennie *et al.* (1990) reported a great deal of chromatic selectivity in blob cells, but not more so than in cells of interblob regions.

Tootell *et al.* (1988) combined CO staining with radiolabeling by ^{14}C-2-deoxy-d-glucose (2DG), a marker for short-term metabolic activity. In this way, cells that are active when a stimulus is being presented can be compared anatomically to the distribution of CO blobs. As expected, when a monkey viewed diffuse chromatically-varying stimuli, 2DG uptake was high in the cells contained within blobs. However, this result could occur because blob cells have larger receptive field sizes; it may not be indicative of response to hue. Cells within blobs were also labelled by activity elicited by both chromatic and achromatic stimuli of low spatial frequency. For chromatic modulation of higher spatial frequencies, 2DG uptake was found in the blob and interblob regions. Taken together, these findings suggest that blobs may be organised around spatial frequency tuning rather than hue. If so, identification of colour selectivity with blobs follows because chromatic information is carried better by cells with low spatial frequency tuning, as shown by the low-pass chromatic CSF. Cells in the interblob regions are tuned to higher spatial frequencies and have lower chromatic selectivity.

3.4. Colour Axis Rotation Revealed by Silent Substitution

Most of the aforementioned studies involved receptive field mapping with monochromatic or broadband stimuli presented as flashes on a dark background. Such stimuli necessarily have both chromatic and achromatic (or luminance) components. When luminance and chromaticity are confounded in the test stimuli, they are likely to be confounded in the cell's receptive field profile. A few recent studies have modulated chromaticity around a white point while holding luminance constant. Lennie *et al.* (1990) used the methods of Derrington *et al.* (1984), described in the previous chapter, to map receptive fields of V1 cells within the colour space illustrated by Figure 3. Their results were similar to those of Derrington *et al.* with some important quantitative differences. Most simple cells

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[6] One additional complication often overlooked in this explanation is that simultaneous contrast is experienced over areas of the visual field that are at least an order of magnitude larger than double-opponent receptive field centres. Receptive fields in cortical Area V1 thus signal a change in colour only at the edge of many natural stimuli, even though we obviously experience uniform hue across larger extended areas. Long-range mechanisms are needed to "fill-in" the colour of the area between the stimulus borders (see Spillmann & Werner, 1996).

combined cone signals linearly as was true for complex cells, but this occurred in complex cells following half-wave rectification. Compared with P cells of the LGN, which fall into well-defined groups, cortical cells were quite varied in their chromatic tuning.

More recently, Cottaris and DeValois (1998) have studied cortical receptive fields using methods similar to those of Lennie *et al.* In contrast to earlier work, they identified many cells that combine cone signals in a nonlinear manner, as might be expected from opponent-hue cancellation functions (Figure 2). One difference in the response of V1 cells and the cancellation functions is that single cells do not show a linkage between opponent-hue pairs. This might, however, be due to the rectification in cortex that masks inhibitory activity from an opponent hue. DeValois *et al.* (1997a) also reported that nearly every neurone in area V1 from which they recorded had inputs from all three cone types. This observation has an important consequence; it rotates the geniculate axes in the direction of the unique hue axes as illustrated by Figure 3. It is not yet clear how this rotation can occur when S cones are so few in number compared to M and L cones. In a physiologically-based theory of colour vision advanced before obtaining these results from cortical cells, DeValois and DeValois (1993) proposed that this rotation might occur in cortex by combining S-cone signals with M- and L-cone signals, as suggested by Müller (1930). Recent evidence supports this view. It is tempting to speculate that the mechanism may be in the upper layers of area V1, possibly in the blobs, where K-cell fibres carrying S-cone signals are combined with signals from P cells.

4. Chromatic Properties of V2 and V3 Receptive Fields

One view of striate cortex function is that it segregates information about colour, form, motion and depth and distributes that information to extrastriate regions for specialised processing. The CO staining patterns have been thought to reveal parallel pathways that originate from retinal M and P streams, forming the inputs to posterior parietal cortex through area MT and to inferior temporal cortex through area V4. Their streams are shown schematically in Figure 8. It has been suggested that the temporal stream is specialised for identification of objects based on form and colour, while the parietal cortical path is specialised for motion perception, spatial localisation and control of attention. Ungerleider and Mishkin (1982) call the temporal and parietal streams the "what" vs. "where" paths, respectively.

4.1. Area V2

Like V1, V2 is organised retinotopically, has ocular dominance and orientation columns, and contains anatomical subdivisions revealed by patterns of CO staining. These CO staining patterns suggest three anatomical divisions or "compartments" in V2: densely stained thin and thick stripes separated by pale stripes. These stripes are visible in horizontal sections throughout all V2 layers (Tootell *et al.*, 1988), but the difference between thin and thick stripes is often subtle in the macaque brain. The thick stripes can be identified more clearly by counterstaining with the monoclonal antibody Cat-301 (DeYoe & Van Essen, 1985). The thin stripes receive much of their input from blobs in the upper layers of area V1, while the pale stripes seem to receive their main inputs from P cells of the interblobs. M-

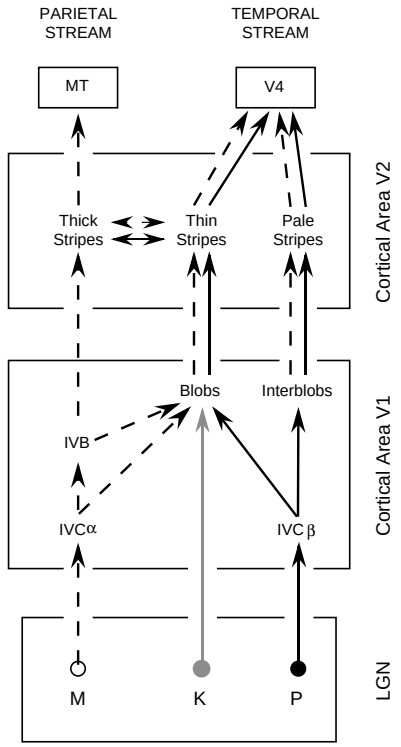

Fig. 8 A schematic of some of the major feedforward connections that give rise to serial, hierarchical processing from the LGN through extrastriate cortex. This schematic shows the parallel pathways through cortical areas V1 and V2 that have been identified by cytochrome oxidase (CO) staining. V3/VP connections between V2 and MT and V4 are not shown here because CO staining has not revealed divisions there. Magnocellular (M) pathways are shown by dashed lines, parvocellular (P) pathways by solid lines, and a koniocellular (K) pathway by a grey line. It is not known whether there is a parallel K path beyond the blobs that is separate from the P paths, nor is it clear how M inputs reach the interblobs, although there are numerous lateral connections within cortex.

cell signals are relayed from layer IVB in V1 to the thick stripes in V2. Livingstone and Hubel (1984) initially suggested that M paths are linked to thick stripes, orientation-selective P paths to thin stripes, and colour-coding P-paths to pale stripes. However, intracortical connections to V1 blobs come from both M and P pathways, as noted in the previous section, and it seems unlikely that M and P segregation could be any better when the signals arrive in area V2. The connections shown in Figure 8 reflect this mixed input, but do not rule out the possibility of some bias in strength of inputs along the lines suggested by Livingstone and Hubel.

In one of the few quantitative studies of V2 receptive fields, Levitt *et al.* (1994) studied chromatic and achromatic receptive field properties using diffuse stimuli and gratings adjusted to the optimal spatial frequency for each cell. The results were quite similar to those obtained for striate cortex. Most V2 cells were more responsive to luminance modulation than chromatic modulation. Cells that were highly colour-selective tended to have poorer orientation selectivity, although some cells tuned to orientation also responded to chromatic modulation. Consistent with results of Cottaris and DeValois (1998) for V1, Levitt *et al.* reported that many V2 cells combined cone signals nonlinearly.

Cells that were selective in their response to colour, size and motion were found mainly, but not entirely, in different V2 stripes. The relation between colour selectivity and V2 locations is presented quantitatively in Figure 9. Most colour-selective cells were found in the upper layers of V2. Within the various layers, there were more colour-selective cells in the thin and pale stripes, consistent with greater P than M input. Cells more responsive to chromatic than luminance modulation were found only in the thin stripes. Gegenfurtner *et al.* (1996) reached

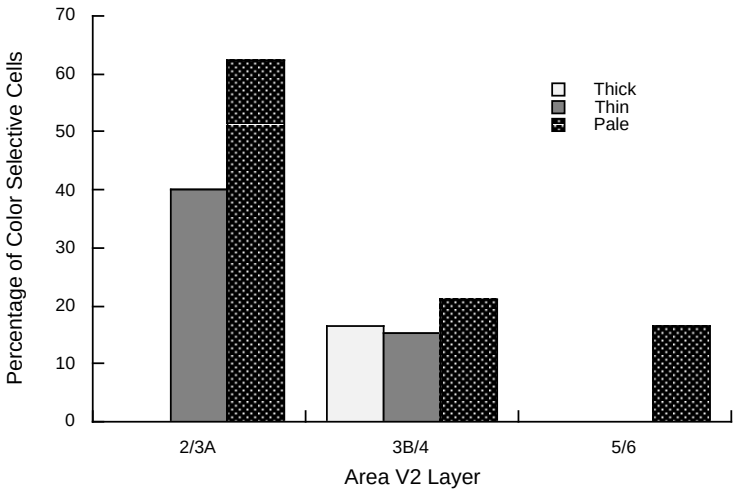

Fig. 9 Percentage of colour-selective cells in area V2 laminae and CO compartments. (Data from Levitt *et al.*, 1994.)

similar conclusions after mapping V2 receptive fields with probes for colour, motion and form. However, many cells were selective to more than one of these types of stimuli, regardless of their CO compartment. Kiper *et al.* (1997) concluded that one of the major differences between V1 and V2 is in the higher proportion of cells in V2 (~ 35%) with narrow colour tuning, a property that some researchers suggested to be characteristic of cells in area V4. They found very few cells with double-opponent receptive fields.

4.2. Area V3/VP

Cortical area V3 receives input from layer IVB (dominated by M cells) of striate cortex as well as from area V2. This cortical area does not have regions that stain selectively for CO, but it is known to have inputs from both M and P pathways. The major efferent projections from V3 are to areas MT and V4. Most of the cells in V3 respond selectively to orientation, direction of motion, or binocular disparity. While earlier studies suggested that cells in area V3 have little or no selectivity to colour, several more recent studies reach different conclusions (Felleman & Van Essen, 1987). Gegenfurtner *et al.* (1997), using the same methods described previously for their studies of area V2, verified that directional selectivity was an important characteristic of cells in V3. These and other data support the conjecture that this area is important in processing higher-level motion signals. In addition, they found about half of V3 cells to be colour selective, essentially the same proportion previously found in area V2 using the same criteria. Many of these cells had more S-cone input than these researchers observed in cells of area V2, but the input was weak and combined with strong additive signals from M and L cones. Achromatic contrast sensitivity was generally higher for V3 than V2 cells. Finally, some of the directionally-selective cells responded to isoluminant chromatic gratings, leading Gegenfurtner *et al.* to conclude that there is significant interaction between colour and motion signals in this extrastriate area.

5. Cortical Area V4

The topographic mapping of visual space is generally less strict at those higher cortical levels where specialised processing has been suggested to occur. The concept of specialised cortical modules fits our experience, in the sense that we can think of perceptual dimensions (colour, motion, form) that are not localised in space. The existence of specialised cortical regions is also reasonable from computational considerations; breaking a task into components is an efficient way to cope with complex tasks. Such specialisation within cortex would also minimise the number and length of connections needed to link neural representations of common properties across widely separated portions of a stimulus (Barlow, 1986). However, this specialisation might only be advantageous after earlier stages have accomplished preparatory steps common to all specialised modules; otherwise, there would be unnecessary duplication of circuitry. Zeki (1975) suggested that area V4 is a cortical module specialised for processing of colour and area MT for motion. This role of MT has been largely sustained; the hypothesis about V4 has been less completely supported. Zeki's hypothesis has nevertheless engendered a great deal of research regarding the properties of cells in these areas, their anatomical connections, and the consequences of circumscribed cortical damage.

Area V4 has a topographical organisation, but the mapping is considerably more complicated than that found in V1. The topography is difficult to discern because it is in a highly convoluted region of cortex and because the receptive fields are so large. While the representation in V1 is concerned with the contralateral visual field, V4 is concerned primarily with only about 30°, mostly of the central visual field. Part of this field (~5°) is ipsilateral, indicating that it has major inputs via the corpus collosum from the opposite hemisphere, presumably from the corresponding area V4.

The main inputs to area V4 are from the thin and pale stripes of V2 (DeYoe & Van Essen, 1985) and from V3/VP. The latter input may be one of the sources of the M-cell signals that it receives. These M-cell inputs are apparently not appreciably segregated from those from P-cell dominated paths (Ferrera *et al.*, 1994). V4 provides a major source of input to inferotemporal cortex, which is known to be essential for visual recognition of objects.

It is generally agreed that a high percentage of cells in area V4 respond selectively to colour, although the exact proportion clearly depends on the criteria used. The spectral bandwidth of V4 cells is similar to that found in LGN P-cells using similar methods, suggesting that V4 responses are based on colour opponency established at a prior level, but half-wave rectified in the cortex (deMonasterio & Schein, 1982). Receptive fields of area V4 are 4-6 times as large as those at comparable eccentricity in V1, and an individual receptive field may receive input from thousands of ganglion cells (Schein & Desimone, 1990). Despite their large receptive fields, some V4 cells appear to be as selective as cells in striate cortex in their tuning for size, spatial frequency, and orientation (Desimone *et al*., 1985; Desimone & Schein, 1987). Finding the optimal stimulus for a V4 cell can be difficult due to the complexity of their receptive fields.

Many cells in V4 have a silent suppressive surround for stimulation outside the conventionally-mapped receptive field. Schein and Desimone (1990) reported that the effectiveness of the surround inhibition was maximal when it was close to the wavelength that most effectively drove the centre, while the surround had a

facilitatory effect when it approached the complementary colour to the centre. Such cells may be useful in figure-ground segregation based on colour.

5.1. Colour Constancy

Our ability to label colours with the same terms despite changes in the spectral distribution of the illumination is called colour constancy. The requirements for a colour constant visual system have been much studied (*e.g.*, Maloney & Wandell, 1986). This work provides useful guidance for evaluating neural and psychophysical data, but it is also worth noting that the human visual system is not completely colour constant under most conditions. The previous chapter described a role of von Kries adaptation in supporting colour constancy, but other aspects of constancy may require a comparison of illumination across wide regions of space. These effects have been tested with spatially-complex patterns consisting of patchworks of different colours called "Mondrians" because of their resemblance to paintings by Piet Mondrian. Using such patterns, Land (1959) showed that while a focal region changes its appearance when the illumination is altered in spectral composition, these changes are less evident when the same surface is viewed in the context of the whole pattern.

Some surprisingly long-range interactions related to colour contrast demonstrated in the retina and in the LGN may contribute to integration and comparisons of colour across large areas of the visual field. Zeki (1983) has suggested, however, that V4 cells have a special role in colour constancy. Such a role might arise from the large surrounds in many V4 receptive fields that could permit a comparison of the light within one spectral band in a focal region to light of the same spectral band covering a large portion of the scene. If changes in illumination affect the centre and surround identically, cell responses will be invariant with illumination. Cells with these properties would be consistent with those that Zeki (1980) described as colour constant when tested with a Mondrian stimulus.

The input to V4 from lower cortical areas carries a representation of the contralateral visual field; yet, the large surrounds of V4 cells representing the fovea include areas of the ipsilateral visual field. Inputs to cells in V4 from the ipsilateral field are believed to be provided by the corresponding V4 area in the opposite hemisphere through connecting fibres of the corpus collosum. As a result, the ipsilateral portion of receptive field surrounds in V4 is eliminated when the corpus collosum is transected. In a patient whose corpus collosum was severed to prevent the spread of epileptic seizures, colour constancy across the midline was disrupted. Colour constancy was not disrupted, however, for the patient when all parts of a Mondrian stimulus were placed in the same visual hemifield (Land *et al.,* 1983). These findings support the belief that V4 cells provide an important part of the network for colour constancy.

5.2. Attentional Modulation of V4 Receptive Fields

Desimone *et al.* (1985) estimate that the receptive fields of area V4 are about 4-6 times larger in diameter than those cells representing the same spatial locations in V1. These cells show high selectivity for spatial dimensions of stimuli falling within their receptive fields, as though their receptive fields are composed of a number of

spatial subunits. *If* these subunits were separately addressable, it might be possible to enhance responses to important information and filter out irrelevant information.

Many cells in area V4 are difficult to drive with visual stimuli, and some appear to use input from outside the geniculostriate path in the control of eye movements (Fischer *et al.*, 1981). Studies of alert behaving monkeys demonstrate that the response properties of area V4 cells are not fixed but can be modulated by attention (Haenny *et al.*, 1988). For example, after an animal was trained to respond to one of two stimuli, the response of a V4 cell to the unattended stimulus presented inside its receptive field was dramatically reduced; the response to the attended stimulus was enhanced, as though the receptive field effectively "contracted" around the attended stimulus (Moran & Desimone, 1985). Such modulation of receptive fields was not observed for cells in area V1, but it was observed for cells in inferotemporal cortex, an area receiving input from V4 and known to be important for visual recognition and memory.

5.3. Effects of V4 Lesions

Wild *et al.* (1985) reported that colour constancy was disturbed in monkeys following a V4 lesion, while chromatic discrimination was unaffected. Others have reported relatively modest effects of V4 lesions on chromatic discrimination.

Heywood and Cowey (1987) conducted psychophysical tests of colour discrimination with macaque monkeys following bilateral lesions of area V4. Monkeys were presented with three stimuli and reinforced for picking the one that was different from the other two. The lightness of the stimuli was systematically varied. Following a V4 lesion, hue discrimination was significantly impaired at lower lightness values. Unoperated controls and animals with a different extrastriate lesion showed no impairment. Experimental and control animals did not differ in an achromatic lightness discrimination task. In addition to a loss of colour discrimination, V4-lesioned animals suffered a severe loss of pattern discrimination. Thus, behavioural studies confirm the evidence from single-unit studies that area V4 is involved in the analysis of both colour and form information. Similar results were described by Schiller and Lee (1991), who ablated portions of area V4 in monkeys and then tested them on a variety of tasks with stimuli presented in affected or unaffected regions of the visual field. Deficits in the affected region were found for both colour and pattern discrimination, but even more profound losses were observed when the animal had to choose stimuli requiring comparisons within an array based on lower contrast, smaller size, etc. It thus appears that area V4 is involved in much more than the analysis of colour. Nevertheless, with respect to colour, it seems reasonable to conclude that V4 is involved in a high level of analysis of chromatic properties of images which supports constancy and figure-ground segregation by hue.

6. Human Cerebral Achromatopsia and Consciousness of Colour

Lesions of human striate cortex result in complete blindness; partial lesions will result in circumscribed regions of the visual field that are blind. These patients lack phenomenal vision; they are not aware of visual stimuli presented in the blind field. They can nevertheless detect and discriminate some luminance changes, as well as orientation and size changes, when tested with forced-choice procedures. This capacity to "see" without awareness is called blindsight. Both the normal and

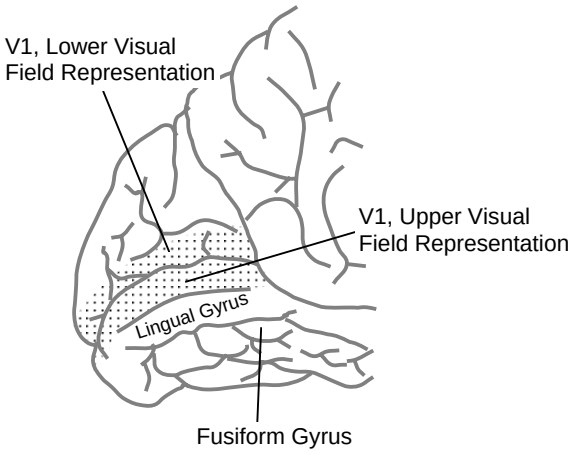

Fig. 10 Posterior region of the left hemisphere of the human brain, viewed medially to expose the lingual and fusiform gyri. Filled pattern shows the approximate region of V1 (Modified after Zeki, 1993.)

cortically-blind visual fields may show normal photopic spectral sensitivity under conditions that produce a V_λ-like function or conditions that reveal the presence of colour-opponent mechanisms (Jaeger *et al.,* 1988). The spectral sensitivity curves from one patient were identical for the two regions of the visual field except that the sensitivity of the cortically-blind field was approximately 0.5 log unit lower. In addition, blind-sight patients are able to discriminate wavelength when tested by a forced-choice procedure (Stoerig, 1998). Because opponent-chromatic responses are not found in midbrain pathways, these results suggest that unconscious colour discrimination in these patients is mediated by the rather sparse connections from LGN to extrastriate cortical regions (Fries, 1981).

In contrast to patients with striate lesions, patients with lesions in certain extrastriate areas are lacking only in phenomenal colour vision, a condition called achromatopsia.[7] These patients describe images or scenes as appearing grey, similar to watching a black-and-white television. Even objects identified as white, such as snow, are usually described as appearing drab, dirty and grey. Postmortem evaluations and computerised tomography of the living brain identify the sites of damage as being in the lingual and fusiform (occipitotemporal) gyri, located in the ventral portions of the occipital lobes (Damasio *et al.*, 1980), as shown in Figure 10. The achromatopsia may encompass half (hemiachromatopsia) or all of the visual field depending on whether the damage to these gyri is unilateral or bilateral. More often than not, there is a peripheral defect in the upper visual field due to its representation in a region of striate cortex near the lingual gyrus. Damage extending anteriorly into other extrastriate areas may produce additional deficits of perception, most commonly prosopagnosia (the inability to recognise faces).

Much has been learned from an extensively studied patient, M.S., who suffered complete cortical blindness in one half of his visual field and achromatopsia in the other half due to damage to his lingual and fusiform gyri. Tests were conducted with stimuli placed in the achromatopsic region of his visual field. His two-colour increment thresholds showed him to have normal S-, M- and L-cones (Mollon *et*

[7] Achromatopsia should be distinguished from dyschromatopsia, a condition associated with colour confusions and in which hues are experienced but are highly desaturated. It is also different from colour anomia, a loss in the ability to name colours but not other objects or stimulus dimensions, attributed to severed connections between language and visual areas.

al., 1980) although he was unable to use differences in cone signals to reliably discriminate colours on standard tests of colour vision. He could, however, read colour plates from the Ishihara test (made up of coloured dots of different size and reflectances) if they were placed farther away (2 m), and he was able to detect some isoluminant chromatic borders even though he described the colours as appearing the same shade of grey. This ability was quite limited, though, for he could not pick out a chromatic square when imbedded in a field of grey squares of varying lightness (Heywood *et al*., 1994). This patient, like a patient described by Victor *et al.* (1989), had normal chromatic contrast sensitivity, mediated perhaps by cells in area V1. Thus, signals about chromatic properties were available to extract information about form even though they were not available for the conscious appreciation of hue.

On the basis of achromatopsia, Zeki (1990) has argued that there is a colour centre in the human brain, and that this centre is homologous with area V4 in the macaque monkey. Cases of achromatopsia that are apparently uncomplicated by any other visual defect provide support for the existence of a colour centre. Conversely, there are many visual deficits that completely spare colour vision. One striking example was the patient described by Rovamo *et al.* (1982) who had abnormally low achromatic contrast sensitivity, but normal chromatic contrast sensitivity.

A cortical area that shows selectivity for colour has been identified through positron emission tomography (PET), a method that reveals brain regions activated by a particular stimulus on the basis of increased blood flow. PET scans showed enhanced activity in two cortical regions when subjects viewed a multicoloured Mondrian pattern (Lueck *et al.,* 1989). One region was V1 (and perhaps V2), and the other was in the lingual and fusiform gyri. Then, when subjects viewed a grey Mondrian with patches having the same luminances as the coloured version, activity in V1 remained high, but activity was reduced in the other area(s). Although studies from PET scans and achromatopsia support Zeki's idea that there is a colour centre in the human brain, it seems unlikely that the sites of damage associated with human achromatopsia are homologous to area V4 in the macaque monkey. Lesions of V4 invariably produce deficits in both pattern and colour discrimination, but this is not invariably the case with achromatopsia.

These clinical studies raise questions about what it means to have conscious colour vision. Since patients with achromatopsia can detect light through chromatic mechanisms, discriminate wavelengths, and locate isoluminant chromatic borders, clearly one does not require consciousness to make reliable decisions about colour. It is evident that phenomenal representations of colour are not always necessary for biological systems that discriminate colour. On the other hand, it is possible that the absence of conscious colour experience might limit learning and memory for chromatic information, although this seems not to have been studied experimentally.

7. Summary

P- and M-cell inputs from the LGN are segregated upon their arrival in layer IV of cortical area V1, but they are less strictly segregated in other V1 layers and higher cortical areas. Colour selectivity is significantly different in layers of V1 that integrate the layer IV inputs. Receptive fields in these regions vary much more in

their colour selectivity than in LGN, and they have significantly more S-cone input than is observed for LGN P cells. The source of this S-cone "amplification" has not been determined, but it may be derived from the K-cell input projecting from the LGN directly to the V1 blobs. Thus, a major accomplishment of striate cortex may be rotation of the colour axes by integrating S-cone input, as illustrated in Figure 3 and suggested by DeValois and DeValois (1993).

The CO compartments provide for parallel pathways within the context of a topographic representation in the first visual areas. These pathways appear to provide inputs to posterior parietal cortex through area MT and to inferior temporal cortex through area V4. Regions of V4 and MT may be functionally specialised to identify what something is (temporal path) and where something is (parietal path). While the CO compartments do provide some specialisation, their segregation of signals derived from P and M pathways is not complete. There is overlap between P and M inputs to the blob and interblob regions of V1 and to the thick, thin and pale stripes of V2.

Receptive fields in area V1 differ from LGN cells in their spatial organisation, with many carrying multiplexed information about luminance, chromaticity, orientation and movement. It is usually assumed that this information must be separated for perception of separate perceptual dimensions. How this might be accomplished is a major unsolved problem. Remarkable differences in chromatic properties of receptive fields between V1 and areas V2 and V3 have not been reported yet. V4, however, contains cells that appear quite different from the other prestriate areas. They are selective in their response to colour, and they have large receptive field surrounds; perhaps these large surrounds support colour constancy and promote segregation of figure and ground by colour. At the same time, cells in V4 have complex spatial properties and are subject to top-down influences modulating their receptive fields on the basis of motivation and attention. Therefore, it seems unlikely that V4 is a "colour centre" in the monkey brain, and it is equally unlikely to be homologous with those regions of human cortex (lingual and fusiform gyri) associated with achromatopsia, since this colour deficit sometimes occurs unaccompanied by other visual deficits.

Acknowledgements

The helpful comments of Russell L. DeValois, James Kraft, Celeste McCullough, Janice L. Nerger, Kathleen A. Schelble, Brooke Schefrin and Vicki J. Volbrecht are gratefully acknowledged. The author's research is supported by the National Institute on Aging (AG04058).

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