CORTICAL MECHANISMS OF COLOUR VISION

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The perception of colour is a central component of primate vision. Colour facilitates object perception and recognition, and has an important role in scene segmentation and visual memory. Moreover, it provides an aesthetic component to visual experiences that is fundamental to our perception of the world. Despite the long history of colour vision studies, much has still to be learned about the physiological basis of colour perception. Recent advances in our understanding of the early processing in the retina and thalamus have enabled us to take a fresh look at cortical processing of colour. These studies are beginning to indicate that colour is processed not in isolation, but together with information about luminance and visual form, by the same neural circuits, to achieve a unitary and robust representation of the visual world.

SENSORY SYSTEMS

It has been known since the nineteenth century that there are three types of photoreceptor for daylight vision in the human eye[1,2]. Since then, the shapes of the spectral absorption functions of the different cones have been determined with increasing precision in psychophysical experiments and verified using various electrophysiological methods[3]. On the molecular genetic level, the basis for the formation of the cone photopigments is known from the work of Nathans and colleagues (for review, see REF. 4). Whereas the rods and the short-wavelength (S)-cones are phylogenetically older, the long- (L) and middle-wavelength (M) cones evolved from a common ancestoral pigment only about 35 million years ago, probably for the benefit of an improved diet of ripe red fruit rather than green leaves[5].

Trichromacy implies that the effect of any light on the visual system can be described by three numbers — the excitation it produces in the L-, M- and S-cones. The cones act like photon counters; information about the wavelength of each individual photon is lost, a principle termed 'univariance' by Rushton[6]. For each individual cone, information about wavelength and intensity is confounded. At the next stage of processing, the visual system must compare the signals from the different cones to compute the colour of objects.

In the retinal ganglion cells, three channels convey information from the eye to the brain[7,8]. In the L + M or luminance channel, the signals from L- and M-cones are added to compute the intensity of a stimulus. In the L – M colour-opponent channel, the signals from L- and M-cones are subtracted from each other to compute the red–green component of a stimulus. Finally, in the S – (L + M) channel the sum of the L- and M-cone signals is subtracted from the S-cone signal to compute the blue–yellow variation in a stimulus. FIGURE 1 illustrates the transformation from the cone signals into colour-opponent signals. These three channels — the 'cardinal directions' of colour space[9] — are functionally independent and transmitted in anatomically distinct retino-geniculo-cortical pathways. Cells in the magnocellular layers of the lateral geniculate nucleus (LGN) are sensitive mostly to luminance information, cells in the parvocellular layers to red–green information, and cells in the koniocellular layers to blue–yellow information. Colour opponency has also been shown to be of computational importance, as it removes the inherently high correlations in the signals of the different cone types[10].

Although these early stages of processing have been explored in great detail, the later, cortical stages of visual processing are less well understood. We know

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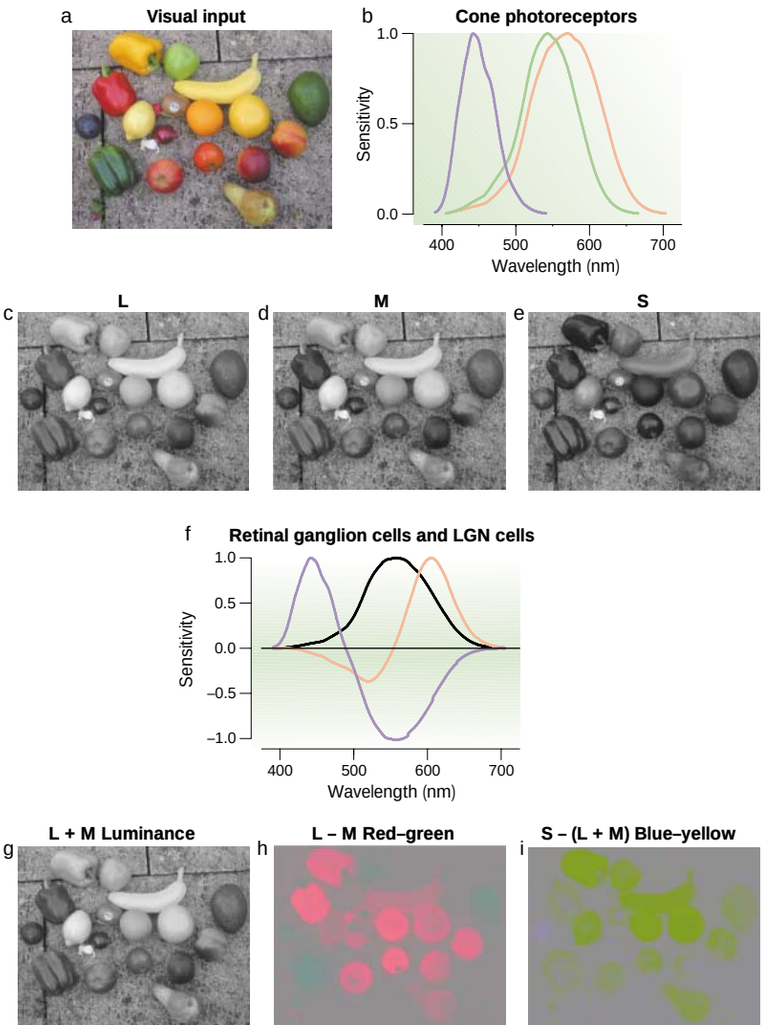

Figure 1 | **Early stages of colour processing.** Colour vision (for example, of the picture shown in **a**) starts with the absorption of light by three types of cone photoreceptor (L, M and S) in the eye (**b**). The three black and white pictures (**c**–**e**) show how the three cone types are excited by the image in **a**. The L- and M-cone images are similar. The electrical signals generated by these photoreceptors go through complicated circuitry (**f**) that transforms the signals into three channels— one carrying luminance and the other two being colour-opponent, red–green and blue–yellow (**g**–**i**). These colour-opponent signals are sent to the visual cortex by way of the thalamic lateral geniculate nucleus (LGN).

that many cells in the visual cortex respond to colour. The main questions are whether these cells respond to other visual attributes as well, what kind of computations these cells perform on their visual input, and how these signals contribute to our perceptual experience of colour.

Psychophysics

Before discussing the cortical mechanisms of colour vision, it is helpful to summarize briefly the psychophysics of colour perception, which constitute the behavioural, perceptual and experiential measures that we try to explain in terms of neural events. Many psychophysical studies were motivated by the idea that colour, form and motion were processed separately in the visual cortex[11,12]. Rediscovering a principle first used by Liebmann[13], Ramachandran and Gregory[14] created displays that were designed to activate the colour vision system selectively. They used knowledge about human luminance perception to construct stimuli in which every point was assigned the same luminance (FIG. 1h,i). These stimuli, which vary only in hue and saturation, are called iso- or equiluminant. A number of early studies concluded that processing of features such as form and motion was severely impaired at isoluminance (for an overview, see REF. 15). Interestingly, 25 years later, and after many experiments, it can be said that for most functions the colour vision system is just as efficient as the luminance system (for reviews, see REFS 16,17). Why did the early studies lead to such mistaken conclusions?

Most low-level visual functions depend heavily on the magnitude of the input signal, as nearly all neurons in the visual system give faster and stronger responses to stimuli of higher contrast[18]. Contrast C is typically defined as $C = (L - L_0)/L_0$, where L is the luminance of an object and L_0 is the luminance of the background. As isoluminant stimuli are defined as having no luminance difference from the background, we need to find another way to specify the strength of the input signal. We already know the spectral absorption functions of the cones, so we can calculate the effect any input stimulus has on the cones and then calculate the resulting cone contrasts ($C_{S,M,L}$), for example $C_S = (A_S - A_{S0})/A_{S0}$ where A_S specifies the S-cone activation caused by an object and A_{S0} the S-cone activation caused by the background. Luminance stimuli, by definition, activate all three types of cone equally, and their excitations vary between 0 (black) to full (bright white). The contrasts are equal in all three types of cones (FIG. 2a). For isoluminant stimuli, the luminance at each point in a stimulus has to be kept constant. Every increase in excitation of the L-cones, for example, has to be balanced by an equally large decrease in M-cone excitation. Although we could still, in principle, achieve high contrasts, under realistic conditions the maximal achievable contrast is limited by the large overlap between the absorption spectra of L- and M-cones (FIG. 1b) to a value of about 0.3. So, to achieve a 'fair' comparison between the visual capabilities of luminance and colour vision, we first have to normalize the input so that it leads to stimulation of equal magnitude in the cones. When contrasts are normalized in this way, there is no longer an obvious impairment of visual perception at isoluminance. The efficiency of processing is equal for colour vision and luminance vision for most tasks, even though in some cases there are performance advantages for luminance stimuli because higher input contrasts can be achieved[16,17].

In addition to functioning as well as the luminance system in many tasks, the colour vision system excels at visual detection. Stromeyer and colleagues[19] determined the visual stimulus with the least physical energy that can be detected on a neutral grey background. They found that the visual system is most sensitive to small red spots of light. FIGURE 2b shows thresholds for detecting small patches of sinewave gratings that are modulated in various directions of colour space[20]. The thresholds are

METAMERIC
Two stimuli with different spectral light distributions are called metameric if they lead to the same activation patterns in the three cones.

V_λ
The human luminous efficiency function V_λ specifies the effectiveness with which stimuli of different wavelength activate the visual system.

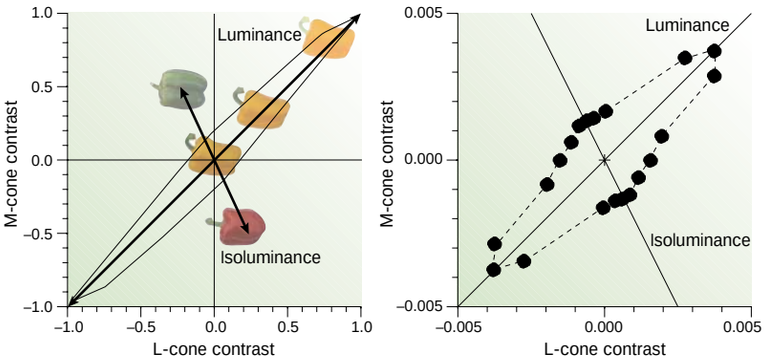

Figure 2 | **High sensitivity of the colour vision system.** Left, cone contrasts produced by green, yellow and red peppers. The positive and negative diagonal lines represent the luminance axis and the red–green isoluminant axis, respectively. The axes show the contrasts produced in the L- and M-cones when comparing other objects to the yellow pepper, which excites L- and M-cones equally well. The red pepper and the green pepper produce changes in L- and M-cone excitations of opponent signs with respect to the yellow pepper. The magnitude of these changes is on the order of 10%. An equally large change along the luminance direction, indicated by the slightly brighter yellow pepper, is hardly noticeable. Right, detection thresholds for slowly modulated (1 Hz), low-spatial-frequency (1 cycle deg.$^{-1}$ visual angle) sinewave gratings of various colours consisting of L- and M-cone modulations[20]. Thresholds are indicated by the distance from the origin and are much smaller for isoluminant stimuli (negative diagonal) than for luminance stimuli (positive diagonal).

smallest when L- and M-cones are modulated with opponent signs (isoluminance) rather than with the same sign (luminance). FIGURE 2a also illustrates the high sensitivity of the colour-opponent system. Although small changes in luminance are hardly noticeable, colour changes with equally large cone contrasts can appear quite marked. We can conclude from these experiments that colour is what the eye sees best.

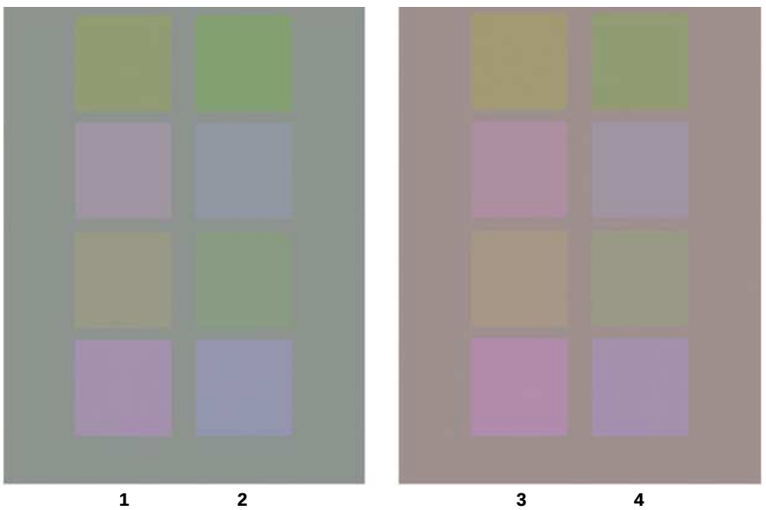

Figure 3 | **Colour constancy and induction.** Eight squares were cut from coloured papers and were illuminated with a greenish light to the left and a reddish light to the right. The mechanisms of colour constancy make the eight squares on the left seem identical to the eight squares on the right. In fact, the four squares in column 1 were printed with the same ink as the four squares in column 4. They appear different because they are printed on a slightly different background. The change in background was due to the illumination change from green to red light. The four squares in the first column appear similar to those in the third column because the local colour difference to the background is identical for these two columns (1 and 3), as it is for columns 2 and 4.

In natural vision, isoluminant stimuli arise rarely, if ever. It would be wasteful to devote many cortical resources to the analysis of such stimuli. In evolutionary terms, the red–green colour vision system evolved after the luminance system was already in place. Therefore, the natural question to ask is what colour information can add to existing luminance information, rather than what it can do on its own. In other words, why did trichromatic colour vision evolve? The answer that is frequently favoured indicates a very specific purpose. Detecting ripe red fruit against green foliage is improved when using the L – M colour-opponent channel[5]. However, psychophysical data indicate that colour vision might have a more general role. Using various natural images as stimuli, we showed that colour vision significantly improves the speed with which images can be recognized, and also significantly improves memory for the same scenes[21,22]. But how does the brain do it?

Chromatic properties of cortical cells

Before talking about 'colour' cells, it is helpful to ask what requirements are necessary for a cell to be useful in the analysis of colour. The simplest answer would be that a colour cell should respond to a single colour only, or to a limited range of colours. However, colour is a sensation, rather than a physical property of the stimulus. On the one hand, two stimuli that reflect the same spectral distributions of light, and which will therefore lead to the same local pattern of cone excitations, can lead to different colour percepts when embedded in different surrounding stimuli — a process often called chromatic induction[23]. On the other hand, different spectral distributions of light can lead to the same colour percept, either because the stimuli are METAMERIC and therefore lead to the same excitation pattern in the cones, or because they are interpreted as being caused by the same object — a concept called colour constancy[24] (FIG. 3). So we have to differentiate between two types of colour-selective neuronal response, one correlating with the ratios of retinal cone excitations and the other with our percept.

We will start with cells that respond to a fixed pattern of cone excitation. Several definitions of colour selectivity have been adopted in recent years. The early studies of primary visual cortex adopted a stringent criterion. Cells responding at all to luminance stimuli were classified as 'luminance' cells, and only the few cells that responded exclusively to chromatic stimuli were classified as 'colour' cells. In this way, the proportion of chromatically responsive cells was estimated to be as low as 10% (REF. 25). Some authors still adopt this strong criterion for the classification of colour cells[26]. Luminance responses mean that cells sum the L- and M-cone input in the 2:1 ratio that is compatible with the human luminous efficiency function, V_λ. Most neurons in the magnocellular layers of the LGN and many cells in all areas of visual cortex behave in this way[27–30]. However, many of these cells deviate slightly from V_λ and, in a strict sense, would give a differential response to colour. For example, a cell that sums L- and M-cones with a ratio of 2.1:1 would respond slightly better to red than to green of the same luminance. According to that definition, most cells in

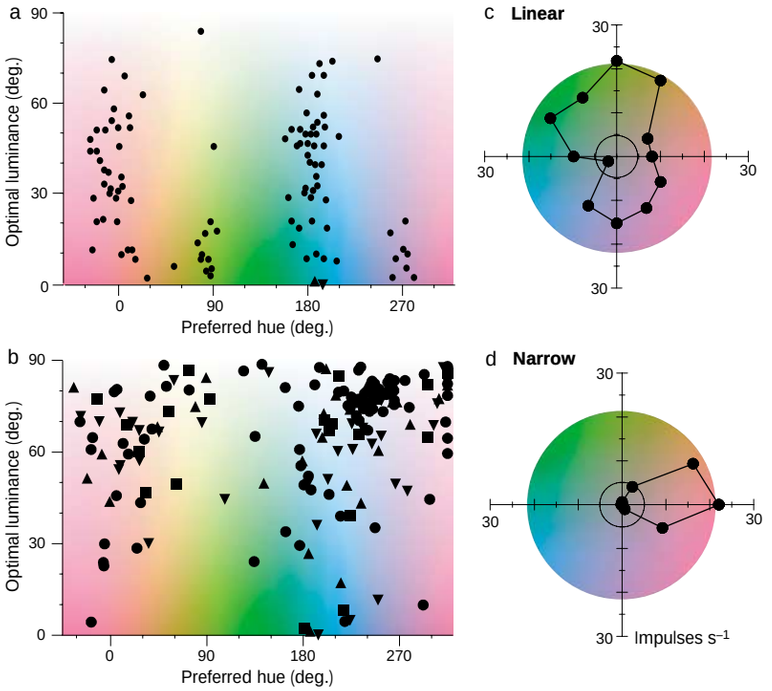

Figure 4 | **Colour tuning in lateral genciulate nucleus (LGN) and cortex. a** | Distribution of optimal colours for LGN cells[8]. **b** | Distribution of optimal colours for a sample of V2 cells from different cytochrome oxidase compartments (upward pointing triangle, thin stripe; downward pointing triangle, thick stripe; square, interstripe) and V3 cells (circles)[36]. The *x*-axis gives the cell's preferred hue and the *y*-axis specifies how much luminance is required for the optimal stimulus. A cell responding to luminance only has an optimal luminance of 90°, and a cell responding to colour only has an optimal luminance of 0°. Whereas LGN cells cluster around the two cardinal directions of colour space, cortical cells differ widely in their chromatic preferences. There is a continuum between cells that respond to luminance only and cells that respond to colour only. **c** | Chromatic tuning of linear cells, such as those found in the LGN and in all areas of visual cortex[36]. **d** | Narrow tuning of a cell from area V2. The magnitude of the response (in impulses per second) to different colours is indicated by the distance from the origin[36].

visual cortex would probably be classified as colour cells. The definition of colour cell that is most frequently adopted in the current literature lies in between these two. Cells that add L- and M-cone inputs are called luminance cells, and cells that subtract L-, M- or S-cone inputs are called colour cells. With this definition, many luminance cells would also give differential responses to colour modulations, and many colour cells would respond to variations in luminance.

Using this definition, the proportion of colour-selective cells is estimated to be about 50% in the early visual areas of macaque monkeys, with little difference between V1, V2, V3 and V4 (REFS 31–38). These results agree with studies using functional magnetic resonance imaging (fMRI) that showed a strong colour-opponent response in the visual cortex of human subjects[39,40]. Past investigators assumed that there were two distinct sub-populations of cells, one responding solely to luminance, the other only to colour. The picture that emerges from many recent studies is that there is a continuum of cells, varying from cells that respond only to luminance, to a few cells that do not respond to luminance at all[34–36,41–43]. The exact proportion of colour cells found in these studies depends to some degree on the relative scaling of luminance and chromatic inputs (see earlier discussion). But the results clearly indicate that — in the cortex — the analysis of luminance and colour is not separated.

The chromatic response properties of cortical cells show both differences from and similarities to those at earlier stages of visual processing. In visual cortex, unlike the LGN, the distribution of a cell's preferred colours does not obviously cluster around particular directions in colour space[36,41,42,44]. FIGURE 4a shows that nearly all colour-selective LGN cells prefer stimuli that are modulated along the cardinal red–green or blue–yellow directions. By contrast, cells in the cortex can have preferences for many other hues (FIG. 4b). As many natural objects are uniformly coloured, cells that respond to a single, specific colour would aid enormously in segmenting these objects. Some neurons in area V2 (REFS 36,45), and to a lesser degree in V1 (REFS 32,33,42,46), have been found to be tuned to certain colour directions with a narrow bandwidth. FIGURE 4c shows the typical, relatively wide tuning of a V1 cell. By contrast, FIG. 4d shows a narrowly tuned cell from area V2. About one-third of all cells in V2 possess such narrow tuning. There is some evidence that these specialized mechanisms might be important for the detection of coloured stimuli in visually variegated environments and for the segmentation of scenes into different objects[47,48].

Colour constancy

What other computations related to colour need to be performed on visual scenes? Once objects are segmented from the background, we need to identify them. Colour can be useful for identification, because — unlike other visual features, such as shape — colours do not change under different viewpoints. We can rotate objects and move them around; their colour does not change, because the light being reflected from the object into the eye remains about the same. However, the light entering the eye does change when we view an object under different illumination conditions (FIG. 3). Only the proportion of light reflected by the object at each wavelength is an inherent property of each object. To be most useful for object recognition and visual search, the visual system has several mechanisms — both retinal and cortical — to discount changes in illumination[49]. We can assign a fixed colour to an object even when the wavelength composition of the light entering the eye changes under different daylight or artificial lighting conditions. There has been quite a debate over the last three decades about where and how colour constancy is achieved in the primate visual system. It is known from behavioural experiments that light adaptation in single cones[50] and local contrasts between adjacent cones[51] are important for colour constancy. Another powerful cue for colour constancy seems to be the average colour over a large region of the scene. The long-range interactions that are necessary to compute this average are beyond the size of receptive fields in the LGN or V1.

Many colour-selective cells in the cortex respond best to uniform surfaces of their preferred colour. They compute a weighted mean of the cone excitations within

their receptive field. As such, their responses will change when the distribution of light in their receptive field or even in the whole scene changes. In other words, these cells will not be able to distinguish between local changes in surface reflectance and global changes in illumination. To achieve this, one has to take the difference between intensities at different image locations. For example, cells in the retina use LATERAL INHIBITION to compute local luminance differences, independently of the overall luminance. To achieve the same thing for colour, we would need a cell with a receptive field that is colour opponent (for example, + L – M) in both its centre and its surround. Then, the difference will be a measure of the chromatic contrast between centre and surround. Such a cell would measure the centre's colour relative to that of its surround. This type of cell is called 'double-opponent'. Double-opponent cells with concentric receptive fields and perfectly balanced chromatic mechanisms were first found in the goldfish retina[52] and later in the macaque primary visual cortex[26,53–56]. These cells would respond preferentially to small (the size of the receptive field centre) coloured objects on a background of different colour but equal luminance. The status of this type of cell was unclear until recently, because other studies failed to find many cells of the concentric (unoriented) double-opponent type[34,41]. Instead, double-opponent cells with oriented receptive fields seem to be more common in V1 (REFS 34,35). Furthermore, the chromatic mechanisms in these cells were often not perfectly balanced. Because of the imbalance, these cells would respond best to edges defined by chromatic and luminance differences. As most edges in the real world do combine luminance and chromatic differences, such cells would be extremely useful for the analysis of natural scenes. Because these cells respond to luminance, Johnson *et al.*[35] made sure that in control experiments these cells take the difference, not the sum, of L- and M-cone inputs. They are therefore *bona fide* colour cells. These double-opponent cells represent a big step towards the neural correlate of colour constancy. Contrary to the earliest reports[57,58], V1 cells should be able to achieve some degree of colour constancy, and there are some hints that this might be the case[42].

However, local computations by double-opponent neurons are probably only part of colour constancy. Neurons with larger receptive fields would be well suited to compute an average colour over a larger region, which could then be used to normalize the colours within that region to an average grey[24,49]. Zeki[57,58] observed some cells that had colour constancy in area V4 of macaque monkeys. In addition to cells being selective to certain cone ratios, which he called wavelength selective, he found cells whose response correlated closely with the colour perceived by a human observer. Schein and Desimone[59] determined the receptive field properties of V4 cells and found a large proportion of cells with large suppressive regions outside their 'classical' receptive field. The experiments that have not been performed until now, and which would give conclusive evidence about the role of V4 in colour constancy, are single-unit recordings in awake behaving monkeys performing a colour categorization task. An individual neuron's capability for colour constancy could be compared to that of the whole monkey, as has been done in the motion domain[60]. Then, we would hope to be able to influence the monkey's judgements by actively stimulating groups of neurons with a certain colour preference. A prerequisite for this type of microstimulation experiment is an ordered map of colour. Such a chromatotopic organization has been shown in optical imaging experiments to exist in monkey visual areas V1 and V2 (REFS 61–64).

LATERAL INHIBITION
Neurons in the retina receive inhibitory input from neighbouring neurons. This reduces the response to slowly changing image intensities and increases the response to sharp edges.

Segregation and integration

One of the main questions about the cortical processing of colour is whether colour is computed separately from other visual attributes, such as form, motion or depth, or whether these computations are carried out simultaneously in the same pool of neurons. This question needs to be answered separately for each level of processing. We will look first at the segregation of colour in early visual processing, in areas V1, V2 and V3. We will then consider a possible segregation at a later stage of processing, perhaps when assigning colours to objects in extrastriate cortical area V4 or in parts of inferotemporal cortex (IT). In the retinogeniculate pathways, there is a good degree of separation between the magno-, parvo- and koniocellular streams. In extrastriate cortex, Ungerleider and Mishkin[65] found evidence for two mainly separate processing streams, ventral and dorsal. The ventral pathway, including parts of V2, V4 and inferotemporal cortex, is thought to be involved with processing what objects are — their shape, size and colour. The processing of object colour in area V4 was considered to be an essential part of the ventral pathway. The dorsal pathway, including areas V3, MT (middle temporal area) and MST (medial superior temporal area), was thought to be involved primarily in analysing where objects are in the environment.

According to the 'segregation' hypothesis[37,66–70], the independent processing of visual attributes is maintained between the retinogeniculate pathway and extrastriate cortex. Proponents of this hypothesis claim that different visual attributes are analysed independently throughout most of the visual cortex. Support for this notion came from anatomical studies. Staining cortical tissue with the mitochondrial enzyme cytochrome oxidase (CO) revealed a parcellation of areas V1 and V2 into CO rich- and CO-poor regions[67–69,71,72]. Cells in CO-rich regions of layers 2 and 3 in V1, the CO-blobs, were found in some studies to have a preponderance of unoriented, colour-selective cells[63,67,73,74]. In area V2, unoriented, colour-selective cells were reported in the thin CO-bands[37,68,70]. So, the anatomical organization revealed by CO staining would have a functional counterpart, in that colour signals would be processed, in V1 and V2, by a population of unoriented neurons, located primarily in the CO-rich blobs of V1 and the thin bands of V2. There have been many studies investigating segregation. Here, I would like to point out that — despite the hot debate — the results of the different studies agree remarkably well. It is mostly the conclusions that differ.

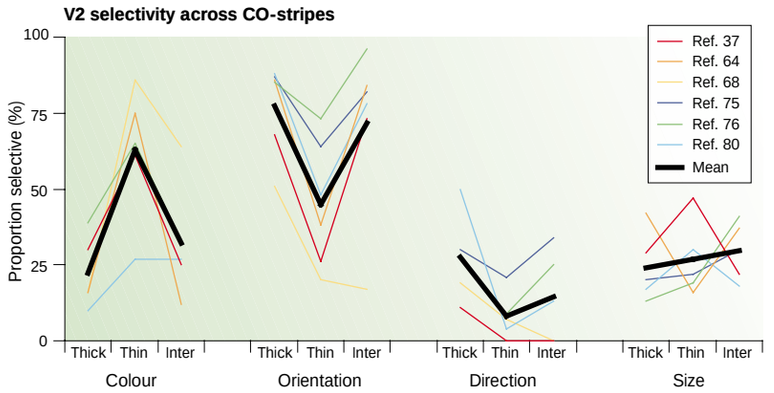

Figure 5 | **Segregation and integration in V2.** The graph shows the proportions of cells selective for colour, orientation, direction of motion and size in different cytochrome oxidate (CO) compartments (thick stripes, thin stripes and interstripes) of macaque monkey area V2. The data are from six studies[37,64,68,75,76,80]. The heavy black lines represent the means across all six studies. Despite the different methods used in these studies, the results show remarkable agreement.

FIGURE 5 shows results from six studies investigating selectivity for orientation, colour, size and direction of motion in the three CO-compartments of V2 (REFS 37,45,64,68,75,76). For each attribute, the proportion of neurons that is selective for that attribute is shown, with the mean values over all six studies. If there was complete anatomical segregation, we would expect each attribute to be processed in only a single CO-compartment: colour in the thin stripes, direction of motion in the thick stripes, and orientation and size in the interstripe regions. If there was no segregation at all, the four black lines should be flat, with equal amounts of selectivity across all stripe compartments. It is obvious that both of these extreme views are wrong. How, then, can different groups of investigators arrive at such extreme and different conclusions? Although there is some variability between the studies, the main trends are similar, despite the different methods that were used in different laboratories.

For colour, there is remarkable agreement, with about 60% colour selectivity for cells in the thin stripes and around half that in the thick stripes and interstripes. The studies by Gegenfurtner *et al.*[76] and Shipp and Zeki[37] obtained nearly identical results close to the average of all studies. As the results are quite ambiguous, each investigator chose their preferred conclusion. Similarly, most studies agree on the relative prevalence of cells selective for the direction of motion. For orientation processing, the main difference lies in the proportion of oriented cells in the thin stripes. Here, there seems to be a systematic difference between studies that used quantitative methods[45,75,76] and those that used qualitative methods to estimate orientation selectivity[37,64,68]. However, whether there is a 5% or a 25% difference between orientation selectivity in the thin stripes and the other CO-compartments, it is without question that there is a difference. This does not seem to be the case for size tuning, which is at the same level — about 25% — in all three CO-compartments. Stimuli that are larger than the receptive field's preferred size lead to a decrease in firing rate. This result emphasizes the importance of complex receptive field surrounds and goes along with strong contextual effects in visual perception (for reviews, see REFS 77,78).

Can we conclude from these results that there is segregation of colour and form processing in early visual cortical areas? We cannot, because the results show only that the average cell, for example in the thin stripes, has a higher chance of being selective for colour and not selective for orientation. From these data, it cannot be concluded that it is the colour-selective cells in the thin stripes that are not orientation selective. Any conclusion is further complicated by the fact that orientation selectivity and colour selectivity are not binary measures. Cells vary continuously in their degree of tuning. A better approach to studying segregation of colour and orientation, for example, is to determine each cell's selectivity, and then to compute the correlation between these selectivites. Where this was done[38] it showed a continuous distribution of orientation and colour selectivity, and the correlation between colour and orientation selectivity was not significantly different from zero (FIG. 6). This lack of correlation also holds for each CO-compartment of V2 (REF. 76). The same is the case in V1, V2 and V3, in both awake behaving and anaesthetized monkeys. Therefore, at the functional level, there is no evidence for segregation. Even though there are more colour-selective cells in the V2 thin stripes, their chance of being orientation selective is just as high as for a luminance-selective cell from a thin stripe.

Several possible reasons for this lack of functional segregation have been discussed. Given that there is more or less complete segregation of colour responses in the LGN — no colour selectivity has been found in magnocellular layers — and that early studies of V1 reported more or less complete segregation of the blob and interblob systems, it was assumed that V2 would be the first stage to integrate the different systems. A rich system of intrinsic connections between neurons in the different CO-compartments would foster such interactions between functional streams[79–81]. However, more recent physiological experiments (FIG. 6) have made clear that there is just as little segregation in V1 as there is in V2 (REFS 34,35,38,82). This argues against the hypothesis recently raised by Shipp and Zeki[37] that there might be different degrees of segregation in different layers of V2. They argue that segregation is more complete in the middle layers of V2, which reflect the input signal from V1, and less so in the outer layers, which reflect feedback signals from other cortical areas. It would take data from many neurons to put this hypothesis to a strong test, but the existence of a large number of oriented colour cells in area V1 makes it unlikely that there is strong functional segregation in any layer of V2. Furthermore, recent anatomical evidence also shows much less segregation at the anatomical level than was previously assumed[83].

Along different lines, Conway and colleagues[26,56] suggest that only a small subset of unoriented colour-selective cells in V1 might be used to compute the colour of objects *per se*. Other colour-selective cells would be

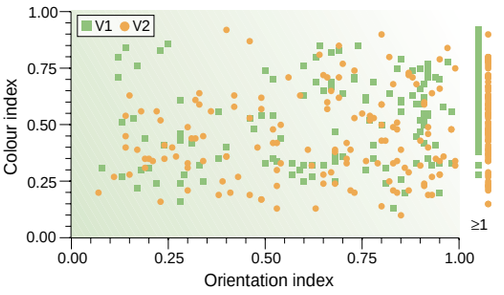

Figure 6 | **Joint neuronal selectivity for colour and orientation.** Squares and circles represent a large number of cells from areas V1 and V2, respectively, of awake behaving macaque monkeys[38]. The *x*-axis shows an index of orientation selectivity. Unoriented cells would respond equally well at all orientations and have an index of 0. Oriented cells would not respond at the orientation orthogonal to the preferred one and have an index of 1. Colour selectivity is specified as the relative volume in colour space to which a cell does not respond. A selective cell responding to only a single colour would have an index of 0.93, and a nonselective cell responding equally well to all colours would have an index of 0.

used for image segmentation and similar tasks, without colour itself being analysed. Although this view is essentially impossible to test empirically, it also implies that the visual system would compute a colour signal for detecting edges, only to ignore it later on when determining the colour of the edge. The contributions of colour cells to the analysis of edges and surfaces were explicitly tested by Friedman *et al.*[38]. They found no difference in edge enhancement between colour- and luminance-selective cells. These results contradict the idea that colour information is treated solely by a dedicated, specialized population of neurons in the early visual areas. They agree with psychophysical studies showing that the processing of form defined by colour is limited mainly by retinal factors — the contrast in the cones — but not by subsequent processing[84].

Is there a 'colour centre' in the cortex?

There are many reports of patients who fail to see the world in colour. The most frequent condition is rod monochromacy, an inherited disease where the cone photoreceptors fail to develop[85]. Because rods have poor spatial and temporal resolution, are absent from the fovea, and are fully bleached at normal outdoor light levels, the vision of rod monochromats is highly impaired. Another type of achromatopsia occurs after damage to visual cortex. This condition is called cerebral or acquired achromatopsia. According to many clinical reports[86,87], some of these patients have a specific deficit of colour vision, with hardly any impairment of form vision. If this is the case, then we might expect to find an area of the brain where neurons respond predominantly to colour — a 'colour centre'. Colour-selective neurons are concentrated in the parvocellular layers of the LGN. Lesions of the parvocellular layers of the LGN in macaque monkeys lead to a severe deficit in colour vision, while most other aspects of vision are intact[88]. Interestingly, lesions to the magnocellular system also leave most of vision intact, even functions that are usually associated with the magnocellular system, such as the initiation of smooth-pursuit eye movements[89]. It seems that the retinogeniculate magnocellular and parvocellular systems are mostly redundant systems, with slightly different specializations[90]. So is achromatopsia due to damage to the parvocellular layers of the LGN?

Most human patients with cerebral achromatopsia have lesions in a part of extrastriate cortex that is presumably homologous to monkey area V4 (for reviews, see REFS 86,87). It is roughly the part of the human brain that also responds strongly to chromatic stimulation in functional neuroimaging experiments[40,91–95]. At first sight, the picture seems clear. Human V4 (hV4) is the colour centre of the human brain and damage to hV4 leads to cerebral achromatopsia. However, the situation is much more complicated. Despite the large number of patients studied, there is no well documented case of cerebral achromatopsia without any other visual defects. Neuroimaging experiments show strong responses to colour not only in hV4, but also in early visual areas V1 and V2 (REF. 40). Finally, it is established that area V4, both in monkeys and in humans, is not only responsible for colour vision. In fact, V4 is a main centre for all aspects of spatial vision and is an important link between vision, attention and cognition[96].

The most widely known case of human achromatopsia is that of the colour-blind painter Jonathan I., who started to see the world in black and white after a car accident. The case was described in detail by Sacks in a literary journal[97]. Unfortunately, even though many scientists performed tests on Jonathan I., there is not a single piece of scientific documentation of the case. Problems arise when a phenomenological description of the patient claiming improved night vision ("I can read license plates at night from four blocks away") is taken as equivalent to an objective assessment of his visual function — in this case, rod visual acuity. No documented case of achromatopsia has produced an improvement of any aspect of visual perception. The case is even more complicated, because no lesion could be found in computed tomography or magnetic resonance scans. What is clear is that Jonathan I. is by no means a typical case of achromatopsia.

Nearly all achromatopsic patients have scotomas, regions that are completely blind, mostly bilaterally and in the upper parts of the visual field[86,87,98]. The scotomas are probably due to the proximity of hV4 to striate cortex and the optic radiation. As hV4 is just below V1, damage to hV4 often also includes the lower parts of V1, which represent the upper visual fields. Most reported cases have bilateral lesions. Unilateral lesions can lead to achromatopsia in one hemifield, but this is often not noticed because foveal colour vision stays intact. Most importantly, other visual defects occur in conjunction with the lack of colour vision, such as deficits in contrast discrimination of black and white tones, and often (12 out of 14 cases reviewed in REF. 86; 15 out of 31 cases reviewed in REF. 98) prosopagnosia,

the inability to recognize familiar faces. However, if the damage occurs at a high level of visual processing, this implies that all the earlier mechanisms of colour processing should be intact, as has been shown in several newer studies[99–105]. These patients have normal or nearly normal chromatic sensitivity to chromatic gratings or adjacent fields, but fail to discriminate the colours of separated objects[102]. What seems to be dysfunctional in achromats is the assignment of colours to objects.

In monkeys, lesions of extrastriate area V4 lead to mild deficits in colour vision[106–108], but also to other perceptual, attentional and cognitive deficits[96]. Lesions to the next processing stage, IT cortex, seem to mimic the human condition of cerebral achromatopsia, producing a specific loss of colour vision. However, the colour deficit depends on the removal of all of IT cortex, and this has other effects on higher-level vision[109]. There is no perfect animal model of achromatopsia, which could be because there is no simple homology between monkey V4 and hV4 (REF. 110), or because some of the colour vision deficits in human achromatopsia are only accessible at the phenomenal level and monkeys cannot tell us what they see[111].

There is, however, good agreement between the work on human patients and human brain imaging studies. Invariably, neuroimaging experiments reveal an extrastriate area that is highly active in response to chromatic versus luminance stimulation. The coordinates for that region, which has been called the colour centre of the brain, are close to the region that is usually implicated in lesions leading to cerebral achromatopsia. The debate centres on whether this colour centre is the fourth visual area of the human brain. Hadjikhani *et al.*[94] argued that the colour centre is distinct from V4, and gave it a new name, V8. However, a careful analysis of the topographic representation of the visual field along the human ventral pathway[95] did not find an additional hemifield representation adjacent to hV4, as claimed by Hadjikani *et al.*[94]. It is most likely that V8 is what other authors called V4, and corresponds to what has been called the colour centre on the basis of lesion studies of achromatopsic patients[112–115].

Should we conclude that hV4 is the colour centre of the human brain? We can say with certainty that it is important for colour vision, but it seems unlikely that the whole human equivalent of monkey V4 — which takes up about 10% of monkey visual cortex — is devoted solely to the analysis of colour. Electrophysiological recordings in humans show that only a small part of hV4 is concerned with colour, and that nearby parts deal with other aspects of object recognition[116]. There are also other regions with a high sensitivity to colour, most notably V1 and V2 (REFS 40,93,94). As a further argument against the existence of a single colour centre in the human brain, other authors have reported that colour discrimination, colour constancy and colour memory can be dissociated in patients with lesions to extrastriate cortex[117–119]. So, as is the case for most other visual attributes, our experience of colour probably depends on the activity of neurons in several cortical areas.

Conclusions

Until single-cell recordings of neurons in primate visual cortex became widely available in the 1970s and 1980s, nearly all progress on colour vision related to retinal mechanisms. Over a period of more than 100 years, the cone absorption spectra have been determined more and more precisely. Starting with the work of DeValois *et al.*[7], the second-stage colour-opponent mechanisms have been characterized fully at the electrophysiological[8,27,120], psychophysical[9] and computational[10] levels. Many of the early recording studies of primate visual cortex were still hampered by unclear notions about the earlier stages of processing, and by a multitude of different notations for specifying colour coordinates. Looking at more recent studies, we can say that this stage has been left behind. Current research can concentrate on cortical processing and has already led to exciting new results[26,35,46,93].

First, it is now clear that there are more cells in visual cortex that are concerned with colour than was first assumed. Comparisons between luminance stimuli and colour stimuli are often problematic and akin to comparing apples and oranges. Now that we know how the colour signal is sent from the eye to the brain, we are better able to scale stimuli and observe their effect on the cortical visual areas. This approach has led to consistent results from single-unit recordings and neuroimaging experiments[35,36,40,93]. Similarly, the detailed knowledge of early processing stages has enabled us to characterize the differences between colour mechanisms in the retina, LGN and visual cortex. In agreement with the processing of nearly all other visual attributes, cortical neurons are more specialized in their chromatic preferences than neurons at earlier stages. Neurons can have a chromatic preference for any hue[41], and often their tuning is narrow and specific[36,42,46]. Concerning the combination of colour information with the other visual attributes, the results from different investigators seem to converge, despite widely differing conclusions[37,76]. It can safely be stated that there is some degree of segregation in anatomically defined subregions of V1 and V2, but there is much less evidence for any kind of functional segregation in these early visual areas[38].

The only way to reach a definitive answer to the question of segregation is to find a better way of integrating single-unit recording data from monkeys with psychophysical and neuroimaging data from humans. This is a problem that concerns all aspects of perception and cognition. The most pressing question for cognitive neuroscience in the near future is to find out how similar or different monkey and human extrastriate cortical areas are. Additional experiments using modern imaging techniques in monkeys[121,122] will help to clarify the relationship between the visual responses in the monkey brain, where single neurons have been studied extensively, and the visual responses in the human brain, where mostly behavioural and imaging data are available. In the long run, more emphasis has to be given to the computations that are performed on the colour signals (and visual signals in general), rather than to the localization of regions that are important for the analysis of colour.

1. Young, T. The Bakerian lecture: on the theory of light and colours. *Phil. Trans. R. Soc. Lond.* **92**, 12–48 (1802).
2. Helmholtz, H. L. F. Über die Theorie der zusammengesetzten Farben. *Ann. Phys. Leipzig* **887**, 45–66 (1852).
3. Stockman, A. & Sharpe, L. T. The spectral sensitivities of the middle- and long-wavelength-sensitive cones derived from measurements in observers of known genotype. *Vision Res.* **40**, 1711–1737 (2000).
4. Nathans, J. The evolution and physiology of human color vision: insights from molecular genetic studies of visual pigments. *Neuron* **24**, 299–312 (1999).
5. Mollon, J. D. & Jordan, G. "Tho' she kneel'd in that place where they grew…" — the uses and origins of primate colour vision. *J. Exp. Biol.* **146**, 21–38 (1988).
6. Rushton, W. A. H. in *Handbook of Sensory Physiology Vol VII/1. Photochemistry of Vision* (ed. Dartnall, H. J. A.) 364–394 (Springer, New York, 1972).
7. DeValois, R. L., Abramov, I. & Jacobs, G. H. Analysis of response patterns of LGN cells. *J. Opt. Soc. Am. A* **56**, 966–977 (1966).
8. Derrington, A. M., Krauskopf, J. & Lennie, P. Chromatic mechanisms in the lateral geniculate nucleus of macaque. *J. Physiol. (Lond.)* **357**, 241–265 (1984).
9. Krauskopf, J., Williams, D. R. & Heeley, D. W. Cardinal directions of color space. *Vision Res.* **22**, 1123–1131 (1982).
10. Buchsbaum, G. & Gottschalk, A. Trichromacy, opponent colours coding and optimum colour information transmission in the retina. *Proc. R. Soc. Lond. B* **220**, 89–113 (1983).
11. Zeki, S. M. Functional organization of a visual area in the posterior bank of the superior temporal sulcus of the rhesus monkey. *J. Physiol. (Lond.)* **236**, 549–573 (1974).
12. Zeki, S. M. Colour coding in the superior temporal sulcus of rhesus monkey visual cortex. *Proc. R. Soc. Lond. B* **197**, 195–223 (1977).
13. Liebmann, S. Über das Verhalten farbiger Formen bei Helligkeitsgleichheit von Figur und Grund. *Psychol. Forschung* **9**, 300–353 (1927).
14. Ramachandran, V. S. & Gregory, R. L. Does colour provide an input to human motion perception? *Nature* **275**, 55–56 (1978).
15. Livingstone, M. S. & Hubel, D. H. Psychophysical evidence for separate channels for the perception of form, color, movement, and depth. *J. Neurosci.* **7**, 3416–3468 (1987).
16. Krauskopf, J. in *Color Vision: From Genes to Perception* (eds Gegenfurtner, K. R. & Sharpe, L. T.) 303–316 (Cambridge Univ. Press, New York, 1999).
17. Hawken, M. J. & Gegenfurtner, K. R. in *Color Vision: From Genes to Perception* (eds Gegenfurtner, K. R. & Sharpe, L. T.) 283–299 (Cambridge Univ. Press, New York, 1999).
18. Albrecht, D. G. & Hamilton, D. B. Striate cortex of monkey and cat: contrast response function. *J. Neurophysiol.* **48**, 217–237 (1982).
19. Chaparro, A., Stromeyer, C. F., Huang, E. P., Kronauer, R. E. & Eskew, R. T. Jr. Colour is what the eye sees best. *Nature* **361**, 348–350 (1993).
A psychophysical study showing the extremely high sensitivity of the red–green colour vision system.
20. Gegenfurtner, K. R. & Hawken, M. J. Temporal and chromatic properties of motion mechanisms. *Vision Res.* **35**, 1547–1563 (1995).
21. Gegenfurtner K. R. & Rieger, J. Sensory and cognitive contributions of color to the recognition of natural scenes. *Curr. Biol.* **10**, 805–808 (2000).
22. Wichmann, F. A., Sharpe, L. T. & Gegenfurtner, K. R. The contributions of color to recognition memory for natural scenes. *J. Exp. Psychol. Learn. Mem. Cogn.* **28**, 509–520 (2002).
23. Kirschmann, A. Ueber die quantitativen Verhaeltnisse des simultanen Helligkeits- und Farben-Contrastes. *Philos. Stud.* **6**, 417–491 (1891).
24. Hurlbert, A. in *Perceptual Constancy: Why Things Look A They Do* (eds Walsh, V. & Kulikowski, J.) 283–321 (Cambridge Univ. Press, Cambridge, 1998).
25. Hubel, D. H. & Wiesel, T. N. Receptive fields and functional architecture of monkey striate cortex. *J. Physiol. (Lond.)* **195**, 215–243 (1968).
26. Conway, B. R., Hubel, D. H. & Livingstone, M. S. Color contrast in macaque V1. *Cereb. Cortex* **12**, 915–925 (2002).
27. Lee, B. B., Martin, P. R. & Valberg, A. The physiological basis of heterochromatic flicker photometry demonstrated in the ganglion cells of the macaque retina. *J. Physiol. (Lond.)* **404**, 323–347 (1988).
28. Shapley, R. Visual sensitivity and parallel retinocortical channels. *Annu. Rev. Psychol.* **41**, 635–658 (1990).
29. Gegenfurtner, K. R. *et al.* Chromatic properties of neurons in macaque MT. *Vis. Neurosci.* **11**, 455–466 (1994).
30. Dobkins, K. R. & Albright, T. D. Behavioral and neural effects of chromatic isoluminance in the primate visual motion system. *Vis. Neurosci.* **12**, 321–332 (1995).
31. Dow, B. M. & Gouras, P. Color and spatial specificity of single units in Rhesus monkey foveal striate cortex. *J. Neurophysiol.* **36**, 79–100 (1973).
32. Gouras, P. Opponent-colour cells in different layers of foveal striate cortex. *J. Physiol. (Lond.)* **199**, 533–547 (1974).
33. Yates, J. T. Chromatic information processing in the foveal projection (area striata) of unanesthetized primate. *Vision Res.* **14**, 163–173 (1974).
34. Thorell, L. G., DeValois, R. L. & Albrecht, D. G. Spatial mapping of monkey V1 cells with pure color and luminance stimuli. *Vision Res.* **24**, 751–769 (1984).
The first study to show that most cells in area V1 of macaques respond to luminance and colour stimuli. The authors also observed a large number of cells that show spatial tuning to isoluminant stimuli.
35. Johnson, E. N., Hawken, M. J. & Shapley, R. The spatial transformation of color in the primary visual cortex of the macaque monkey. *Nature Neurosci.* **4**, 409–416 (2001).
The authors present single unit recordings from area V1 of macaque monkeys showing that most neurons respond to colour and luminance, many of them with an oriented double-opponent receptive field organization.
36. Kiper, D. C., Fenstemaker, S. B. & Gegenfurtner, K. R. Chromatic properties of neurons in macaque area V2. *Vis. Neurosci.* **14**, 1061–1072 (1997).
An investigation into the chromatic tuning properties of neurons in area V2 of macaque monkeys. About a third of the neurons in this study show a highly selective response to particular colours.
37. Shipp, S. & Zeki, S. The functional organization of area V2, I: specialization across stripes and layers. *Vis. Neurosci.* **19**, 187–210 (2002).
38. Friedmann, S., Zhou, H. & von der Heydt, R. The coding of uniform color figures in monkey visual cortex. *J. Physiol. (Lond.)* **548**, 593–613 (2003).
For the first time, the responses to colour and form are investigated in a large population of neurons in V1 and V2 of awake behaving monkeys. The authors find no correlation between colour and form responses.
39. Kleinschmidt, A., Lee, B. B., Requart, M. & Frahm, J. Functional mapping of color processing by magnetic resonance imaging of responses to selective p- and m-pathway stimulation. *Exp. Brain Res.* **110**, 279–288 (1996).
40. Engel, S. A., Zhang, X. & Wandell, B. A. Color tuning in human visual cortex measured using functional magnetic resonance imaging. *Nature* **388**, 68–71 (1997).
These authors use functional neuroimaging to show that visual cortical areas V1 and V2 give a strong colour-opponent response.
41. Lennie, P., Krauskopf, J. & Sclar, G. Chromatic mechanisms in striate cortex of macaque. *J. Neurosci.* **10**, 649–669 (1990).
This was the first study to show that the chromatic tuning of neurons in area V1 of macaque monkeys is not restricted to the three colour-opponent mechanisms of the LGN.
42. Wachtler, T., Sejnowski, T. J. & Albright, T. D. Representation of color stimuli in awake macaque primary visual cortex. *Neuron* **37**, 681–691 (2003).
43. Shapley, R. M. & Hawken, M. J. Neural mechanisms for color perception in the primary visual cortex. *Curr. Opin. Neurobiol.* (in the press).
44. Yoshioka, T., Dow, B. M. & Vautin, R. G. Neuronal mechanisms of color categorization in areas V1, V2 and V4 of macaque monkey visual cortex. *Behav. Brain Res.* **76**, 51–70 (1996).
45. Levitt, J. B., Kiper, D. C. & Movshon, J. A. Receptive fields and functional architecture of macaque V2. *J. Neurophysiol.* **71**, 2517–2542 (1994).
46. Cottaris, N. P. & DeValois, R. L. Temporal dynamics of chromatic tuning in macaque primary visual cortex. *Nature* **395**, 896–900 (1998).
47. Gegenfurtner, K. R. & Kiper, D. C. Contrast detection in luminance and chromatic noise. *J. Opt. Soc. Am. A* **9**, 1880–1888 (1992).
48. Healey, G. Using color for geometry-insensitive segmentation. *J. Opt. Soc. Am. A* **6**, 920–937 (1989).
49. Kraft, J. M. & Brainard, D. H. Mechanisms of color constancy under nearly natural viewing. *Proc. Natl Acad. Sci. USA* **96**, 307–312 (1999).
50. von Kries, J. *Chromatic Adaptation*. Selection translated and reprinted in *Sources of Color Science* (ed. MacAdam, D. L.) 109–119 (MIT Press, Cambridge, Massachusetts, 1970).
51. Foster, G. H. & Nascimento, S. M. Relational colour constancy from invariant cone-excitation ratios. *Proc. R. Soc. Lond. B* **257**, 115–121 (1994).
52. Daw, N. W. Goldfish retina: organization for simultaneous color contrast. *Science* **158**, 942–944 (1967).
53. Michael, C. R. Color vision mechanisms in monkey striate cortex: dual-opponent cells with concentric receptive fields. *J. Neurophysiol.* **41**, 572–588 (1978).
54. Michael, C. R. Color vision mechanisms in monkey striate cortex: simple cells with dual opponent-color concentric receptive fields. *J. Neurophysiol.* **41**, 1233–1249 (1978).
55. Michael, C. R. Color-sensitive complex cells in monkey striate cortex. *J. Neurophysiol.* **41**, 1250–1266 (1978).
56. Conway, B. R. Spatial structure of cone inputs to color cells in alert macaque primary visual cortex (V-1). *J. Neurosci.* **21**, 2768–2783 (2001).
57. Zeki, S. Color coding in the cerebral cortex: the reaction of cells in monkey visual cortex to wavelengths and colors. *Neuroscience* **9**, 741–765 (1983).
58. Zeki, S. Color coding in the cerebral cortex: the responses of wavelength-selective and color-coded cells in monkey visual cortex to changes in wavelength composition. *Neuroscience* **9**, 767–781 (1983).
59. Schein, S. J. & Desimone, R. Spectral properties of V4 neurons in the macaque. *J. Neurosci.* **10**, 3369–3389 (1990).
60. Salzman, C. D., Britten, K. H. & Newsome, W. T. Cortical microstimulation influences perceptual judgements of motion direction. *Nature* **346**, 174–177 (1990).
61. Xiao, Y., Wang, Y. & Felleman, D. J. A spatially organized representation of colour in macaque cortical area V2. *Nature* **421**, 535–539 (2003).
62. Landisman, C. E. & Ts'o, D. Y. Color processing in macaque striate cortex: relationships to ocular dominance, cytochrome oxidase, and orientation. *J. Neurophysiol.* **87**, 3126–3317 (2002).
63. Landisman, C. E. & Ts'o, D. Y. Color processing in macaque striate cortex: electrophysiological properties. *J. Neurophysiol.* **87**, 3138–3151 (2002).
64. Roe, A. W. & Ts'o, D. Y. Visual topography in primate V2: multiple representation across functional stripes. *J. Neurosci.* **15**, 3689–3715 (1995).
65. Ungerleider, L. G. & Mishkin, M. in *Analysis of Visual Behavior* (eds Ingle, D. J., Goodale, M. A. & Mansfeld, R. J. W.) 549–586 (MIT Press, Cambridge, Massachusetts,1982).
66. Zeki, S. Functional specialisation in the visual cortex of the rhesus monkey. *Nature* **274**, 423–428 (1978).
67. Livingstone, M. S. & Hubel, D. H. Anatomy and physiology of a color system in the primate visual cortex. *J. Neurosci.* **4**, 309–356 (1984).
68. DeYoe, A. & Van Essen, D. C. Segregation of efferent connections and receptive field properties in visual area V2 of the macaque. *Nature* **317**, 58–61 (1985).
69. Hubel, D. H. & Livingstone, M. S. Segregation of form, color, and stereopsis in primate area 18. *J. Neurosci.* **4**, 309–356 (1987).
70. Livingstone, M. S. & Hubel, D. H. Segregation of form, color, movement, and depth: anatomy, physiology, and perception. *Science* **240**, 740–749 (1988).
71. Shipp, S. & Zeki, S. Segregation of pathways leading from area V2 to areas V4 and V5 of macaque monkey visual cortex. *Nature* **315**, 322–325 (1985).
72. Livingstone, M. S. & Hubel, D. H. Connections between layer 4B of area 17 and the thick cytochrome oxidase stripes of area 18 in the squirrel monkey. *J. Neurosci.* **7**, 3371–3377 (1987).
73. Ts'o, D. Y. & Gilbert, C. D. The organization of chromatic and spatial interactions in the primate striate cortex. *J. Neurosci.* **8**, 1712–1727 (1988).
74. Roe, A. W. & Ts'o, D. Y. Specificity of color connectivity between primate V1 and V2. *J. Neurophysiol.* **82**, 2719–2730 (1999).
75. Peterhans, E. & von der Heydt, R. Functional organization of area V2 in the alert macaque. *Eur. J. Neurosci.* **5**, 509–524 (1993).
76. Gegenfurtner, K. R., Kiper, D. C. & Fenstemaker, S. B. Processing of color, form, and motion in macaque area V2. *Visual Neurosci.* **13**, 161–172 (1996).
77. Albright, T. D. & Stoner, G. R. Contextual influences on visual processing. *Annu. Rev. Neurosci.* **25**, 339–379 (2002).
78. Spillmann, L. & Werner, S. Long-range interactions in visual perception. *Trends Neurosci.* **19**, 428–434 (1996).
79. Rockland, K. S. A reticular pattern of intrinsic connections in primate area V2 (area 18). *J. Comp. Neurol.* **235**, 467–478 (1985).
80. Levitt, J. B., Yoshioka, T. & Lund, J. S. Intrinsic cortical connections in macaque visual area V2: evidence for interaction between different functional streams. *J. Comp. Neurol.* **342**, 551–570 (1994).
81. Levitt, J. B., Lund, J. S. & Yoshioka, T. Anatomical substrates for early stages in cortical processing of visual information in the macaque monkey. *Behav Brain Res.* **76**, 5–19 (1996).
82. Leventhal, A. G., Thompson, K. G., Liu, D., Zhou, Y. & Ault, S. J. Concomitant sensitivity to orientation, direction, and color of cells in layers 2, 3, and 4 of monkey striate cortex. *J. Neurosci.* **15**, 1808–1818 (1995).
This study shows that colour and form processing are not correlated in area V1. The authors observe numerous cells that are selective for colour and orientation, even in the CO-blobs of V1.

83. Sincich, L. H. & Horton, J. C. Divided by cytochrome oxidase: a map of the projections from V1 to V2 in macaques. *Science* **295**, 1734–1737 (2002).
84. Webster, M. A., DeValois, K. K. & Switkes, E. Orientation and spatial-frequency discrimination for luminance and chromatic gratings. *J. Opt. Soc. Am. A* **7**, 1034–1049 (1990).
85. Hess, R. F., Sharpe, L. T. & Nordby, K. (eds) *Night Vision* (Cambridge Univ. Press, Cambridge, 1990).
86. Meadows, J. C. Disturbed perception of colours associated with localized cerebral lesions. *Brain* **97**, 615–632 (1974).
87. Zeki, S. A century of cerebral achromatopsia. *Brain* **113**, 1721–1777 (1990).
A thorough and entertaining description of the history of the investigation of colour blindness.
88. Schiller, P. H., Logothetis, N. K. & Charles, E. R. Role of the color-opponent and broad-band channels in vision. *Vis. Neurosci.* **5**, 321–346 (1990).
89. Page, W. K., King, W. M., Merigan, W. & Maunsell, J. Magnocellular or parvocellular lesions in the lateral geniculate nucleus of monkeys cause minor deficits of smooth pursuit eye movements. *Vision Res.* **34**, 223–239 (1994).
90. Merigan, W. H. & Maunsell, J. H. How parallel are the primate visual pathways? *Annu. Rev. Neurosci.* **16**, 369–402 (1993).
91. Lueck, C. J. *et al.* The colour centre in the cerebral cortex of man. *Nature* **340**, 386–389 (1989).
92. McKeefry, D. & Zeki, S. The position and topography of the human colour centre as revealed by functional magnetic resonance imaging. *Brain* **120**, 2229–2242 (1997).
A detailed neuroimaging study of chromatic responses in the visual cortex of 12 human subjects.
93. Engel, S. A. & Furmanski, C. S. Selective adaptation to color contrast in human primary visual cortex. *J. Neurosci.* **21**, 3949–3954 (2001).
This study demonstrates selective adaptation of different populations of neurons in V1, in line with behavioural measurements.
94. Hadjikhani, N., Liu, A. K., Dale, A. M., Cavanagh, P. & Tootell, R. B. Retinotopy and color sensitivity in human visual cortical area V8. *Nature Neurosci.* **1**, 235–241 (1998).
In this highly controversial article, the authors present evidence that a visual area other than hV4 gives strong and selective responses to colour.
95. Wade, A. R., Brewer, A. A., Rieger, J. W. & Wandell, B. A. Functional measurements of human ventral occipital cortex: retinotopy and color. *Proc. R. Soc. Lond. B* **357**, 963–973 (2002).
An extensive neuroimaging investigation into the representation of visual stimuli in different cortical areas.
96. Chelazzi, L., Miller, E. K., Duncan, J. & Desimone, R. Responses of neurons in macaque area V4 during memory-guided visual search. *Cereb. Cortex* **11**, 761–772 (2001).
97. Sacks, O. & Wasserman, R. The case of the colorblind painter. *NY Rev. Books* **34**, November 19 (1987).
98. Zihl, J. & von Cramon, D. *Zerebrale Sehstörungen* (Kohlhammer, Stuttgart, 1986).
99. Victor, J. D., Maiese, K., Shapley, R., Sidtis, J. & Gazzaniga, M. S. Acquired central dyschromatopsia: analysis of a case with preservation of color discrimination. *Clin. Vision Sci.* **4**, 183–196 (1989).
100. Heywood, C. A., Kentridge, R. W. & Cowey, A. Form and motion from colour in cerebral achromatopsia. *Exp. Brain Res.* **123**, 145–153 (1998).
101. Cowey, A. & Heywood, C. A. Cerebral achromatopsia: colour blindness despite wavelength processing. *Trends Cogn. Sci.* **1**, 133–139 (1997).
A review article that presents evidence for intact processing of colour stimuli at the early stages of the visual system in cerebral achromatopsia.
102. Heywood, C. A., Cowey, A. & Newcombe, F. Chromatic discrimination in a cortically colour blind observer. *Eur. J. Neurosci.* **3**, 802–812 (1991).
103. Heywood, C. A., Nicholas, J. J. & Cowey, A. Behavioural and electrophysiological chromatic and achromatic contrast sensitivity in an achromatopsic patient. *J. Neurol. Neurosurg. Psychiatry* **60**, 638–643 (1996).
104. Troscianko, T. *et al.* Human colour discrimination based on a non-parvocellular pathway. *Curr. Biol.* **6**, 200–210 (1996).
105. Cavanagh, P. *et al.* Complete sparing of high-contrast color input to motion perception in cortical color blindness. *Nature Neurosci.* **1**, 242–247 (1998).
106. Heywood, C. A. & Cowey, A. On the role of cortical area V4 in the discrimination of hue and pattern in macaque monkeys. *J. Neurosci.* **7**, 2601–2617 (1987).
107. Walsh, V., Kulikowski, J. J., Butler, S. R. & Carden, D. The effects of lesions of area V4 on the visual abilities of macaques: colour categorization. *Behav. Brain Res.* **52**, 81–89 (1992).
108. Schiller, P. H. The effects of V4 and middle temporal (MT) area lesions on visual performance in the rhesus monkey. *Vis. Neurosci.* **10**, 717–746 (1993).
109. Cowey, A., Heywood, C. A. & Irving-Bell, L. The regional cortical basis of achromatopsia: a study on macaque monkeys and an achromatopsic patient. *Eur. J. Neurosci.* **14**, 1555–1566 (2001).
110. Merigan, W. H. Human V4? *Curr. Biol.* **3**, 226–229 (1993).
111. Heywood, C. A., Kentridge, R. W. & Cowey, A. Cortical color blindness is not 'blindsight for color'. *Conscious. Cogn.* **7**, 410–423 (1998).
112. Heywood, C. A. & Cowey, A. With color in mind. *Nature Neurosci.* **1**, 171–173 (1998).
113. Zeki, S., McKeefry, D. J., Bartels, A. & Frackowiak, R. S. J. Has a new color area been discovered? *Nature Neurosci.* **1**, 335 (1998).
114. Tootell, R. B. & Hadjikhani, N. Where is 'dorsal V4' in human visual cortex? Retinotopic, topographic and functional evidence. *Cereb. Cortex* **11**, 298–311 (2001).
115. Zeki, S. Improbable areas in the visual brain. *Trends Neurosci.* **26**, 23–26 (2003).
116. Allison, T. *et al.* Electrophysiological studies of color processing in human visual cortex. *Electroencephalogr. Clin. Neurophysiol.* **88**, 343–355 (1993).
117. Clarke, S., Walsh, V., Schoppig, A., Assal, G. & Cowey, A. Colour constancy impairments in patients with lesions of the prestriate cortex. *Exp. Brain Res.* **23**, 154–158 (1998).
118. Rüttiger, L. *et al.* Selective color constancy deficits after circumscribed unilateral brain lesions. *J. Neurosci.* **19**, 3094–3106 (1999).
119. Schoppig, A. *et al.* Short-term memory for colour following posterior hemispheric lesions in man. *Neuroreport* **10**, 1379–1384 (1999).
120. Reid, R. C. & Shapley, R. M. Spatial structure of cone inputs to receptive fields in primate lateral geniculate nucleus. *Nature* **356**, 716–718 (1992).
121. Brewer, A. A., Press, W. A., Logothetis, N. K. & Wandell, B. A. Visual areas in macaque cortex measured using functional magnetic resonance imaging. *J. Neurosci.* **22**, 10416–10426 (2002).
122. Logothetis, N. K., Pauls, J., Augath, M., Trinath, T. & Oeltermann, A. Neurophysiological investigation of the basis of the fMRI signal. *Nature* **412**, 150–157 (2001).

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Online links

FURTHER INFORMATION
Color matters: http://www.colormatters.com/
MIT Encyclopedia of Cognitive Science: http://cognet.mit.edu/MITECS/neurophysiology of color
Gegenfurtner's personal page: http://www.allpsych.uni-giessen.de/karl/
Hans Irtel's colour vision demonstrations: http://www.uni-mannheim.de/fakul/psycho/irtel/cvd.html
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