Insights into the different exploits of colour in the visual cortex

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SUMMARY

A new method that allows controlled masking of luminance contrast has been developed to study the use of chromatic signals in human vision. The method also makes it possible to examine the different uses of chromatic signals (e.g. the generation of perceived colour, or the construction and representation of object structure and form). By using this technique, we studied the threshold detection of chromatic signals in normal trichromats. The results show that chromatic signals are virtually unaffected by ongoing, randomly varying, luminance contrast changes. These findings suggest that chromatic signals are either processed independently or can be separated completely from any confounding luminance contrast components in the stimulus. Thresholds for detection of colour changes only, and for extraction of stimulus structure from chromatic signals in normal trichromats, in subjects with single cone receptor deficiency (i.e. dichromats) and in three subjects with abnormal colour vision caused by bilateral damage to ventromedial, extra-striate visual cortex (i.e. subjects with cerebral achromatopsia) have also been measured. No significant difference in thresholds for the two conditions was observed either in normal trichromats or in dichromats. Subjects with cerebral achromatopsia, however, reveal markedly different thresholds. The results suggest that chromatic signals are processed independently to generate perceived object colour or to construct spatially structured objects, and that these functions involve different neural substrates. The results help to explain, at least in part, why cerebral achromatopsia is a heterogeneous disorder, and why there can be significant differences in the effective use of chromatic signals in subjects described as cerebral achromatopsics.

1. INTRODUCTION

The image of an object can be generated in a uniform background field by producing appropriate, spatially localized changes in luminance. When the spectral composition of the light in the image of the object also changes significantly, the eye sees a coloured object, the colour being determined largely by the signals generated in the three classes of cone receptor which respond maximally to either long (L), medium (M) or short (S) wavelengths. A large amount of processing of receptor signals takes place in the retina, and this results functionally in the formation of two principal classes of ganglion cells which form the magnocellular and the parvocellular pathways. We know that in primates ca. 80% of ganglion cells have spatial, temporal and chromatic properties that enable them to respond to both intensity and/or wavelength changes in the retinal image (De Monasterio & Gouras 1975; Perry et al. 1984; Shapley & Perry 1986). These cells connect to the primary visual cortex (area VI) via the four uppermost layers of the dorsal lateral geniculate nucleus, and may account for both colour vision and discrimination of fine spatial detail (Merigan 1989; Schiller et al. 1990).

In general, most parvocellular retinal ganglion cells have a centre-surround receptive field organization that is chromatically and spatially opponent. Consequently, such neurons respond to both achromatic and chromatic contrast components in the image (Wiesel & Hubel 1966; De Valois & Pease 1971; De Valois & De Valois 1975). The composite signal generated can therefore reflect both wavelength and intensity changes in the image. In an achromatic world, this signal would be ideal to code the presence of borders and edges in the image of an object because it is based on both intensity and spectral changes, with no need for further processing. If, however, the colour of the object is also of interest, then the chromatic and the luminance contrast components that are carried in the firing rate of parvocellular neurons must somehow be separated. Cells that exhibit only spectral opponency with no trace of centre-surround organization have also been described (Wiesel & Hubel 1966; Drcher et al. 1976). The properties of such cells are ideal for coding pure chromatic signals, but they represent only a small percentage of parvocellular neurons (Wiesel & Hubel 1966). The separation and the degree of independence of the chromatic and the luminance contrast contributions to the firing rate of parvocellular neurons has therefore been of great interest (Ingling & Martinez-Uriegas 1983; Lennie & D’Zmura 1988). Schemes for extraction and separation of the chromatic and achromatic contrast components from the composite signals of most parvocellular cells have been proposed and can, at least in principle, result in the formation of...
separate chromatic and achromatic channels (De Valois & De Valois 1993). A second achromatic channel involves the magnocellular pathways and combines signals from L and M cones (Lee et al. 1988). Because these achromatic channels have different spectral and spatio-temporal properties (Livingstone & Hubel 1984; Merigan & Maunsell 1993), silencing both channels when saturated colours are involved is difficult to achieve experimentally.

To investigate how chromatic signals contribute to visual perception, coloured objects of low chromatic saturation that carry little or no luminance signals when viewed against a uniform background have been used in many studies. Under such conditions the coloured objects are often labelled isoluminant, the assumption being that no achromatic signals contribute to the perception of the stimulus. This implies the existence of a single, null, equiluminant point for the system as a whole, an assumption that has often been questioned in view of the different luminance ratios required to show null activity in different magnocellular neurons (Lee et al. 1988).

2. METHODS

Unlike normal trichromats, dichromats cannot discriminate certain colours when presented against an achromatic background field, as a result of single cone receptor deficiency (Wright 1946; Dartnall et al. 1983), but their ability to see spatially structured objects is only rarely impaired. This happens either when the object carries no achromatic luminance information for that class of dichromat or when such signals have been made ineffective by a suitable luminance modulation (Livingstone & Hubel 1984; Merigan & Maunsell 1993), silencing both channels when saturated colours are involved is difficult to achieve experimentally.

To investigate the effectiveness of the RLM technique in masking the detection of achromatic contrast changes, we measured thresholds for detection of achromatic bars embedded in the same RLM noise. In this experiment, the checks which form the coloured test pattern (those illustrated in figure 1b) undergo a systematic change in luminance contrast, instead of the usual chromatic modulation. The subject’s task is to detect vertical bars (that are defined only by an achromatic contrast change). The results of such experiments show that the RLM technique can be used to provide a controlled amount of achromatic contrast masking. The RLM amplitudes investigated in this way extended from 0 to ±35% modulation. Over this range all subjects showed the same linear increase in achromatic contrast thresholds. Results measured for both increments and decrements in luminance in a normal trichromat and in one of the subjects with cerebral achromatopsia are presented in figure 2a. The results show that the RLM technique makes it possible to control the amount of luminance contrast masking generated. In addition to isolating the use of pure chromatic signals, this technique also makes it possible to examine the independent processing of chromatic signals in the presence of ongoing achromatic contrast changes.

3. SUBJECTS WITH CEREBRAL ACHROMATOPSIA

In addition to studying normal trichromats and several dichromats, we also investigated three subjects with cerebral achromatopsia. These subjects have bilateral ischaemic lesions that are restricted to the ventral, occipito-temporal cortex in areas typical of those found in other cases of cerebral achromatopsia (Zeki 1990a, b; Plant 1991). This region of the brain, particularly the fusiform gyrus, is also activated strongly in normal subjects when presented with coloured stimuli (Zeki et al. 1991). Computed tomography (CT) and magnetic resonance imaging (MRI) scans and conventional tests of visual function have been done in each of the three subjects with cerebral achromatopsia studied.

Subject 1 (male) is a 53 year-old translator who suddenly developed blurred vision and topographical disorientation. Colours and chromatic contours appeared washed out and difficult to identify. His achromatic visual acuity was 6/6 bilaterally, and measurements of visual field sensitivity revealed an upper homonymous visual field defect which was
Figure 1. Visual stimuli designed to measure threshold chromaticity changes needed to detect vertical bars (i.e. structure thresholds) generated from chromatic signals only (a, b), or a change in the colour of a square target (i.e. colour thresholds), the shape of the square being defined by luminance contrast (c, d). The template for the stimulus consists of a two-dimensional array of checks. Each check varies randomly in luminance every 0.05 s within a range specified as a percentage of background luminance. A restriction is imposed to ensure that the average luminance over the pattern remains equal to that of the background field. The average luminance of the test pattern and that of the surrounding background field (x, y chromaticity coordinates: 0.305, 0.323) was 34 cd m$^{-2}$. Each stimulus presentation lasts for 0.8 s and consists of random, spatio-temporal luminance modulation of the checks, as shown in (a and c). The change in chromaticity is added to the ongoing random luminance modulation (see (b) and (d)) 0.15 s after the onset of the stimulus and lasts for 0.5 s. The maximum possible difference in luminance between the checks and the surrounding background field is expressed as a percentage of background luminance and determines the level of luminance contrast masking achieved. The patterns shown were generated by using a random luminance modulation (RLM) amplitude of ±25%. The display monitor (HP Model D1187A) was calibrated for luminance against applied gun voltage for each phosphor over the region corresponding to the test pattern and in the presence of the uniform background field used in the experiment. The three display phosphors were also calibrated for chromaticity coordinates by using a Gamma Scientific telespectroradiometer (Model RD2). Standard colorimetric transformations (Wyszecki & Stiles 1982) were then used to compute the phosphor luminances required to generate a specified luminance-chromaticity triplet.

particularly dense in the upper left quadrant. This subject has great difficulty in reading any of the Ishihara pseudoisochromatic plates, and his error score on the Farnsworth-Munsell 100 hue test was 588. He has persistent prosopagnosia in addition to achromatopsia. MRI revealed bilateral ventral occipito-temporal infarction, with the right sided infarct being larger than the left.

Subject C (male) is a 74 year-old retired engineer who developed a left occipital infarct at the age of 65. A right homonymous quadrantopia was noted, together with dysphasia and dysgraphia. Three months later C recovered from all symptoms except the field defect. Three years later he suddenly developed blurred vision and he was unable to recognize faces including that of his daughter. Chromatic borders appeared washed out and saturated colours were often described as pastel shades. Subsequent imaging studies revealed a second right occipital infarct. At the time C was examined for the present study he had bilateral, upper, homonymous quadrantic field defects and his visual acuity was 6/9 with corrected near vision of N8. C was unable to read any of the Ishihara pseudoisochromatic plates with either eye, and his error score on the Farnsworth-Munsell 100 hue test was greater than 800. Magnetic resonance imaging confirmed results from previous cr scans and revealed a bilateral ventral, occipito-temporal infarction involving cortex
and underlying white matter. C's achromatopsia is accompanied by severe prosopagnosia.

Subject W (male) is a 54 year-old retired professional man who suddenly developed a right homonymous hemianopia at the age of 44. A left occipital infarct was demonstrated on CT scanning. Four years later, W suddenly became confused and topographically disoriented, his vision became unclear, and he was unable to recognize faces or to identify colours. When first examined for the present study, W's visual acuity was 6/5 bilaterally, and measurements of visual field sensitivity revealed right homonymous hemianopia with some loss also affecting his left upper quadrant. W can read all of the Ishihara plates, despite having great difficulties in identifying the colours, but makes some errors on the Farnsworth-Munsell 100 hue test. His severe prosopagnosia has persisted. MRI shows a large left occipital infarct and a smaller ventral infarct on the right.

4. RESULTS

Given that most parvocellular neurons respond to both chromatic and achromatic contrast components in the retinal image, we examined the extent to which ongoing achromatic contrast changes can affect the detection of chromatic signals by measuring chromatic thresholds for detection of vertical bars as a function of \( RLM \) amplitude. Results obtained in normal trichromats show that the size of the measured chromatic discrimination ellipses is independent of \( RLM \) amplitude (figure 2b), even for values as large as \( \pm 35\% \).

The results shown for a deuteranope illustrate the effectiveness of the \( RLM \) method in masking the detection of achromatic signals (figure 2c). With zero \( RLM \) amplitude, the stimulus consists of coloured vertical bars which are isoluminant with respect to the uniform background, when seen by a normal trichromat. Because such a stimulus generates some achromatic luminance contrast, when seen by a deuteranope, some directions of chromatic displacement yield thresholds which are significantly smaller than those measured in normal trichromats. In the absence of chromatic signals, when the direction of modulation is along the deuteranope's colour-confusion line (Wright 1946) the subject fails to detect vertical bars even when the chromatic modulation reaches the limits imposed by the phosphors of the display (see figure 2e for \( RLM \) amplitudes of \( \pm 25\% \) and \( \pm 35\% \)). This is also observed for protanopes and tritanopes, the corresponding isochromatic band being oriented along the colour-confusion axis for each class of dichromat (Barbur et al. 1992). The results suggest that normal trichromats and dichromats can make use of chromatic signals to detect vertical bars, when such signals exist, and that the processing of these chromatic signals is independent of the ongoing achromatic contrast changes.

The patients with cerebral achromatopsia behave quite differently by comparison with normal subjects or dichromats in that, in the limit when chromatic displacements are large, each of the three cerebral achromatopsic subjects tested makes use of chromatic signals to detect vertical bars. This can be demonstrated for directions of chromatic modulation towards any region of the spectrum locus, but all three subjects have significantly higher thresholds. Data for subject I are shown in figure 2d with the limits of chromatic modulation imposed by the phosphors of the display. When no random luminance masking is used, subject I can detect bars, but they have no definite colour when viewed against the neutral background; a normal trichromat reports detection of coloured bars. I's chromatic displacement thresholds are also significantly larger than those measured in normal trichromats (figure 2d). This suggests that, when no achromatic contrast masking is used, subject I makes use of achromatic luminance contrast signals to detect vertical bars (figure 2d). A possible explanation for these results is that, as the \( RLM \) amplitude is increased, I's chromatic discrimination thresholds also increase, since they are based initially on detection of achromatic signals. This increase is similar to that observed in dichromats when the chromatic modulation is restricted to their corresponding colour-confusion lines (figure 2c), or in normal trichromats when only achromatic signals are involved (figure 2a).

In contrast to normal trichromats, who always report the detection of coloured vertical bars at threshold when only chromatic signals are involved, the three subjects with cerebral achromatopsia differ amongst themselves, in their ability to detect coloured bars. Subject I can detect bars at threshold and can name their colour correctly. This case corresponds to the largest two chromatic discrimination contours in figure 2d (i.e. squares and triangles), when the threshold chromatic displacements needed to detect vertical bars become independent of \( RLM \) amplitude. Subject C can also detect vertical bars at threshold, but they are not seen coloured. Much larger chromatic displacements, well above the threshold for detection of vertical bars, are needed for this subject to see coloured bars. Subject W has almost normal thresholds for detection of vertical bars, but his ability to discriminate colour changes is again impaired significantly. W often reports the detection of achromatic bars, but his distinction between coloured and achromatic bars at
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Figure 2. The effect of RLM amplitude on thresholds for detection of vertical bars using the stimulus pattern shown in figure 1 (a, b). (a) Threshold contrasts for detection of achromatic vertical bars (defined as log \( \frac{L_t}{L_h} \), where \( L_t \) and \( L_h \) represent the mean luminances of the test target and the remaining foreground checks, respectively) plotted as a function of RLM amplitude. The results show that both increments in luminance (open symbols) and decrements (filled symbols) increase almost linearly with RLM modulation. Data are shown for a normal trichromat (diamonds) and for one of the subjects with cerebral achromatopsia (circles). (b) Chromatic threshold discrimination ellipses measured in a normal trichromat for progressively larger RLM amplitudes. The solid cross at the centre of the ellipse marks the \((x, y)\) chromaticity point of the achromatic background field (0.305, 0.323). The continuous line represents the best-fit ellipse to the data measured for RLM = ±35%. The minor and the major semi-axes and the orientation of the best-fit ellipse are as follows: \(a = 0.0039, b = 0.01\) and \(e = 65.5^\circ\). The results show that, in a normal trichromat, chromatic thresholds for detection of vertical bars are independent of RLM amplitude. (c) Effect of RLM amplitude on chromatic thresholds for detection of vertical bars in a deuteranope. The phosphor limits for ±25% and ±35% modulation are shown as broken lines. A similar increase in the size of the colour-confusion zone with RLM amplitude is also observed in protanopes and tritanopes. For RLM amplitudes > ±25%, the deuteranopes reach the limit of chromatic displacement set by the phosphors of the display without the subjects being able to detect vertical bars. (d) The effect of RLM amplitude on the detection of the test pattern of figure 1c, in a subject with cerebral achromatopsia. Although much larger chromatic discrimination contours are involved, in the limit the subject behaves like a normal trichromat in that the size of the chromatic discrimination contour is no longer affected by an increase in RLM amplitude. This finding suggests that the subject makes use of chromatic signals to detect the test pattern.

threshold is less clear. The observed differences between the three subjects are at least qualitatively consistent with observations from other studies which suggest that subjects with cerebral achromatopsia may sometimes detect stimulus borders or chromatic gratings without seeing colour (Mollon et al. 1980; Heywood et al. 1987, 1991; Victor et al. 1989). These observations suggest that chromatic signals are either independent or can be separated completely from confounding achromatic contrast changes. In addition, these signals carry information that can be used to generate colour and also, independently, to construct spatially structured patterns.

To distinguish between these two possible uses of chromatic signals in human vision, we have extended the RLM technique by including the stimulus shown in figure 1 (c, d). The square test object at the centre of the display is seen clearly at all times as a result of its
achromatic contrast with respect to the surrounding background field. The same RLM modulation is applied to the surrounding checks and to the elements which make up the square object. The only visual attribute that can change systematically when a chromatic displacement is added to the checks that make up the square object is the perceived colour of the object. If the subject cannot process chromatic signals, or if such signals are not available, as can sometimes be the case in dichromats, no change in the square target is detected even for very large chromatic displacements, as shown for the deuteranope (figure 3a). The new test (see figure 1c, d) is therefore different in that it limits the possible use of chromatic signals to the detection of colour changes only.

5. STRUCTURE VERSUS COLOUR THRESHOLDS

Structure thresholds (i.e. detection of vertical bars from chromatic signals as illustrated when going from figure 1a to 1b) and colour thresholds (i.e. detection of colour changes only when going from figure 1c to 1d) were measured in normal trichromats, in dichromats and in the three subjects with cerebral achromatopsia. The two stimulus conditions yield no significant difference in thresholds, both in subjects with normal colour vision and in dichromats (figure 3a). Data for the same tests measured in the three subjects with cerebral achromatopsia are shown in figure 3b, c, d. An RLM amplitude of ± 25%, was selected for all these tests to ensure that only chromatic signals are effective.

The results were unexpected in that the three subjects with cerebral achromatopsia show significant differences between colour and structure thresholds when only chromatic signals are involved. This contrasts with the measurements obtained in normal trichromats and dichromats who show little or no difference between structure and colour thresholds (figure 3a). Interestingly, the cerebral achromatopsia subjects do not show the same pattern of deficit. Subject C (figure 3b) requires considerably larger chromatic displacements when only changes in stimulus colour can be detected, i.e. C's colour thresholds are significantly larger than his structure thresholds when only chromatic signals are involved. Subject I, however, has smaller colour thresholds and much larger structure thresholds (figure 3c). Neither of the first two subjects can read the Ishihara plates, and their performance on the Farnsworth-Munsell 100-hue test shows significant impairment, the score for subject C being close to that expected on the basis of random ordering. Subject W can read all the Ishihara plates, but makes some errors on the 100-hue test. The two tests are different since the Ishihara plates presumably test the use of chromatic signals to extract the contours of the numbers involved, whereas the ordering of coloured discs in the 100-hue test can only be based on detection of colour or luminance differences. This subtle difference between the two tests may not matter in the case of normal trichromats or dichromats who have very similar structure and colour thresholds (figure 3a), but can be important when testing subjects with cerebral achromatopsia.

The results of figure 3 can be used to predict and explain the performance of the three subjects studied in this investigation when using other colour vision tests. It also accounts, at least in part, for the diversity of results obtained from other investigations of colour vision in subjects with cerebral achromatopsia (Zeki 1990a, b; Plant 1991). For example, subject W reads correctly all the Ishihara plates since the chromatic differences which define the numbers involved are larger than his thresholds for pattern detection (figure 3d). Subjects with much higher chromatic thresholds for both colour and pattern detection cannot read the numbers on the Ishihara plates or order the colours in the 100-hue test. In principle, all three subjects investigated here should be able to read all the Ishihara plates provided the difference in chromaticity that defines the numbers involved is made greater than their corresponding structure thresholds. The degree of luminance masking which the Ishihara plates can generate is limited, however. When testing subjects with high thresholds for processing chromatic signals, it may not be possible to generate chromatic displacements that are large enough to be detected while ensuring that the subject does not make use of luminance contrast components to detect the numbers involved.

6. DISCUSSION

By achieving a controlled amount of luminance contrast masking, the results obtained by using the RLM technique in normal trichromats demonstrate that chromatic signals can be extracted and processed independently of any confounding achromatic contrast changes. The results obtained in subjects with cerebral achromatopsia suggest that chromatic signals can have at least two distinct functions, and that these functions can be affected differentially by the lesion. The results suggest that chromatic signals carry sufficient information to enable the generation and spatial representation of an object in terms of its form and structure, and they can be used to generate at least one more visual attribute, namely the perceived object colour. It follows that the neural substrates that are involved in these functions can be affected differently by the exact location and extent of the cortical lesion. Given the extensive damage to the occipito-temporal cortex in all three subjects studied, and the relatively coarse resolution of our existing MRI scans, it is not possible at the present time to relate more precisely the extent and position of their corresponding lesions to the grossly abnormal processing of chromatic signals and, in particular, the observed differences between structure and colour thresholds.

The results show that selective damage to areas of the occipito-temporal cortex can affect the use of chromatic signals differently depending on whether they are used to generate object structure and form or to code perceived object colour. We know from previous studies that chromatic signals can contribute to motion perception (Cavanagh & Anstis 1991). The
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Figure 3. Direct comparison of chromatic displacement thresholds for detection of vertical bars (i.e. structure thresholds, see figure 1b) or colour changes only (i.e. colour thresholds, see figure 1a). (a) Data for a normal trichromat (diamonds) and for a deuteranope (circles). (b), (c) and (d) Comparison of structure and colour thresholds in each of the three subjects with cerebral achromatopsia studied. Unlike thresholds measured in normal trichromats and in dichromats (a), the two tests yield markedly different threshold values in the patients with cerebral achromatopsia. Remarkable is the reversal of thresholds observed in subjects I and C when comparing structure and colour responses. While subject C needs a larger chromatic displacement to see that a square object defined by achromatic luminance contrast has changed colour without changing shape (c), subject I shows larger thresholds when the task is to detect vertical bars from chromatic signals only (c).

Present findings can therefore be extended by examining the use of chromatic signals in the visual cortex for the purpose of generating perceived movement. Measurement of chromatic thresholds for direction and speed discrimination under stimulus conditions that isolate chromatic signals in subjects with cerebral achromatopsia may yield results that are different to those obtained when the stimulus configuration makes possible only the discrimination of colour changes or the onset of a structured pattern. Further, detailed neuro-imaging studies in such subjects may help to determine more precisely the anatomical basis of these differences.

In summary, the results of this investigation suggest that chromatic signals are either processed independently (Rodieck 1991) or can be separated completely from any confounding luminance contrast components in the stimulus. The new findings also reveal more about the different exploits of colour in the cerebral cortex. The results help to explain, at least in part, why cerebral achromatopsia is a heterogeneous disorder, and why there can be significant differences in the effective use of chromatic signals in subjects described as cerebral achromatopsies.

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