ORIGINAL ARTICLE

Evidence for Non-Opponent Coding of Colour Information in Human Visual Cortex: Selective Loss of “Green” Sensitivity in a Subject with Damaged Ventral Occipito-Temporal Cortex

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ABSTRACT

Damage to ventral occipito-temporal extrastriate visual cortex leads to the syndrome of prosopagnosia often with coexisting cerebral achromatopsia. A patient with this syndrome resulting in a left upper homonymous quadrantanopia, prosopagnosia, and incomplete achromatopsia is described. Chromatic sensitivity was assessed at a number of locations in the intact visual field using a dynamic luminance contrast masking technique that isolates the use of colour signals. In normal subjects chromatic detection thresholds form an elliptical contour when plotted in the Commission Internationale d’Eclairage, (x-y), chromaticity diagram. Because the extraction of colour signals in early visual processing involves opponent mechanisms, subjects with Daltonism (congenital red/green loss of sensitivity) show symmetric increase in thresholds towards the long wavelength (“red”) and middle wavelength (“green”) regions of the spectrum locus. This is also the case with acquired loss of chromatic sensitivity as a result of retinal or optic nerve disease. Our patient’s results were an exception to this rule. Whilst his chromatic sensitivity in the central region of the visual field was reduced symmetrically for both “red/green” and “yellow/blue” directions in colour space, the subject’s lower left quadrant showed a marked asymmetry in “red/green” thresholds with the greatest loss of sensitivity towards the “green” region of the spectrum locus. This spatially localized asymmetric loss of “green” but not “red” sensitivity has not been reported previously in human vision. Such loss is consistent with selective damage of neural substrates in the visual cortex that process colour information, but are spectrally non-opponent.

KEYWORDS: cerebral achromatopsia; chromatic sensitivity; colour opponency; colour vision; prosopagnosia

INTRODUCTION

Early stage mechanisms for colour vision rely on comparing the outputs of long (L) and medium (M)

wavelength sensitive cones ($L - M$) and on comparing the sum of those outputs with the output of short wavelength sensitive cones ($S - (L + M)$). These mechanisms are the basis of opponent processing: one channel signals the difference between L and M cone output (the “red/green” channel) and the other the difference between S cone output and the sum of the other two (the “blue/yellow” channel). Because the cone outputs are combined in this way, it is not possible at this stage in the visual system for those cone outputs to be affected independently

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in disease—damage to the (L – M) pathway will for example impair the processing of information from the two cone classes equally. Despite the fact that the processing of opponent colours relies initially on responses from distinct ganglion cells with ON centre sensitivity for the corresponding wavelengths, the evidence so far suggests that the absence or abnormal functioning of cone photoreceptors as a result of congenital deficiencies or damage to the retina or optic nerve caused by disease result in symmetric loss of either (L – M) or (S – (L + M)) chromatic sensitivity or both.

Colour sensitivity is often assessed by measuring chromatic thresholds for detection of targets that differ in spectral composition to that of the surrounding background.¹ When plotted in the Commission Internationale d'Eclairage (CIE) (x,y) chromaticity chart, thresholds measured towards different regions of the spectrum locus form an elliptical contour with well defined major and minor axes. Analysis of the cone contrasts generated for each threshold direction reveals the isolation of S-cone responses along the major axis (that yields yellow/blue discrimination) and M and L cone responses, along the minor axis (that yields red/green discrimination).² The increment in S-cone signal needed for threshold detection in the bluish direction is usually matched in amplitude by a decrement in S-cone contrast needed for threshold in the yellowish direction. This is also the case when only L and M cone contrast signals are involved along the (L + M) (greenish) and (L – M) (reddish) directions. Colour detection at threshold is mediated largely independently by either the (L – M) or the (S – (L + M)) or both chromatic mechanisms and this results in equal and opposing cone contrasts along any line that passes through background chromaticity and intersects the threshold detection contour.² This symmetry in cone contrast thresholds is consistent with opponent processes and has been observed in normal trichromats, in subjects with Daltonism (congenital colour deficiency) and in subjects with acquired colour deficiency as a result of retinal or of optic nerve disease.^{3–5} Loss of chromatic sensitivity following damage to extrastriate visual cortex has been described in numerous studies, which have been reviewed.^{6–9} However, as the majority of studies employed only conventional colour screening tests (such as the Farnsworth-Munsell 100 Hue test), it has not been established whether such losses are always consistent with opponent processing of colour signals.

Previous quantitative studies of cerebral achromatopsia were also restricted to foveal vision and the loss of chromatic sensitivity as a result of damage to extrastriate visual cortex can in principle affect

selectively any area of the visual field. Unilateral lesions can give rise to hemiachromatopsia in the contralateral field.^{10,11} It is therefore of interest to examine any differential loss of chromatic sensitivity within colour opponent channels in the more peripheral regions of the visual field and not only in central vision.

In this paper we report the full findings of a colour vision assessment carried out at three distinct locations in the peripheral visual field in a patient (subject "I") with incomplete cerebral achromatopsia. The experimental work was carried out in 1992 and the findings were reported in an abstract published in 1996.¹² Much progress has been made in understanding the processing of chromatic signals along the visual pathways since this study was carried out—as a result of which there is increased interest in the pathophysiological basis of asymmetric losses of chromatic sensitivity. As far as the authors are aware the experimental findings in this patient represent the first report of loss of chromatic sensitivity caused by damage to the cortex that lacks chromatic opponency and is specific to a localised region of the visual field. For this reason we have taken this opportunity to report the findings in full as a single case report in advance of a systematic study of a large cohort which is in progress.

CASE REPORT

At the time these experiments were carried out, subject I was a 58-year-old white Caucasian male with persistent prosopagnosia and incomplete cerebral achromatopsia due to bilateral occipito-temporal cerebral infarction. Magnetic resonance imaging

FIGURE 1 (A) T1-weighted coronal and (B) T2-weighted axial magnetic resonance imaging showing bilateral ventral occipital infarction more extensive in the right hemisphere (on the left of the images). The right-sided lesion has given rise to a left upper quadrantic field defect. That on the left has caused only a small homonymous scotoma along the horizontal meridian (see Figure 2).

(MRI) revealed bilateral ventral occipito-temporal infarction, with the right sided infarct being more extensive than the left (Figure 1). His best corrected visual acuity was 6/6 bilaterally, and measurements of visual field sensitivity revealed left upper homonymous quadrantanopia and a small homonymous scotoma in the right visual field. A schematic representation of subject I's visual field in relation to the testing locations can be seen in Figure 2.

This subject had difficulty in reading the Ishihara pseudoisochromatic plates and his error score on the Farnsworth-Munsell 100 hue test was 588 (well outside normal limits¹³). I has been the subject of a previous report demonstrating foveal chromatic sensitivity loss.¹⁴

Experiment 1: Assessment of Achromatic Contrast Sensitivity and Scattered Light

Achromatic contrast sensitivity and the amount of scattered light in the eye were measured using bespoke tests available on the P_SCAN system¹⁵. Subject I's data compare favourably with similar results obtained for subjects in the normal age matched range (Figure 3). The amount and angular distribution of scattered light in subject I's eyes were also well within the normal range for his age group (results not shown).

FIGURE 2 Schematic representation of subject I's bilateral patchy field loss. The sketch is derived from a binocular test (hence there is no physiological blindspot), there is a left upper homonymous quadrantanopia and in the right hemifield a homonymous scotoma. Also shown are the locations of test stimuli employed for achromatic contrast sensitivity and colour assessment.

Experiment 2: Chromatic Sensitivity—Foveal Measurements

Chromatic sensitivity was measured using an early version of the colour assessment and diagnosis (CAD) test.^{3,14} This test makes use of dynamic luminance contrast noise to isolate the use of colour signals. The stimulus conditions ensure that the subject is unable to make use of any residual luminance contrast signals in a colour-defined stimulus that is isoluminant for the standard CIE observer. In the absence of colour signals, the subject is unable to see the coloured stimulus, even for the maximum chromatic saturations that can be generated on the visual display employed. The test makes it possible to examine both the sensitivity for detection of objects defined by colour as well as the detection of colour in objects defined by luminance contrast.¹⁴ The amount of dynamic, achromatic luminance contrast noise employed has no effect on the thresholds for detection of colour signals. When the colour thresholds no longer increase with increasing amounts of luminance contrast noise, the subject is able to use chromatic signals and using this technique we were able to show that subject I has reduced chromatic sensitivity at the fovea.¹⁴ Subject I's thresholds are much larger than those expected for normal trichromats and this demonstrates his significant, but uniform loss of chromatic sensitivity in foveal vision (Figure 4). In normal subjects chromatic sensitivity worsens with stimulus eccentricity. Both (L – M) and

FIGURE 3 Foveal achromatic contrast sensitivity for subject I: I's contrast sensitivity curve, as a measure of his achromatic vision is well within the normal age matched range.

FIGURE 4 Subject I's colour detection thresholds measured at the fovea for a number of different colour directions plotted in the CIE (1931) (x-y) chromaticity chart. The centre cross plots the chromaticity of daylight (D_{65}) reflected from a gray, spectrally neutral object. When one moves away from this gray point towards any region of the spectrum locus (i.e. the contour that plots the chromaticity of monochromatic wavelengths), the colour of the object becomes more saturated, but for any constant angular direction, the perceived hue remains largely unchanged. The insets illustrate the complementary colours one would normally perceive along the yellow/blue axis (when the L and M cone signals remain largely unchanged) and along the red/green axis (when the stimulus is not detected by S-cones). In general, any other directions of chromatic modulation result in both (L – M) and (S – (L + M)) signals and generate a range of perceived hues, some of which are labelled as unique hues. It is well established that the colours shown in the insets are not, in general, perceived as unique hues that should be described as red, green, yellow and blue. In spite of this limitation, red/green (RG) and yellow/blue (YB) labels have been used in this paper to describe colour modulations that correspond to L – M and S – (L + M) signals. A normal trichromat requires only a small displacement away from the gray background to detect the coloured object. The statistical limits that define the "standard" normal CAD test observer are shown by the three ellipses at the centre of the graph. The dotted, black ellipse is based on the mean red/green and yellow/blue thresholds measured in 330 normal trichromats. The grey shaded area shows the 97.5% and 2.5% limits of variability within these observers. The deuteranopic, protanopic, and tritanopic colour confusion bands (for dichromats) are also shown for comparison. When compared to normal trichromats, subject I's foveal colour thresholds are much larger indicating large, but almost symmetrical loss of red/green and yellow/blue chromatic sensitivity. In general, both congenital and acquired colour deficiencies result in symmetric loss of red/green and/or yellow/blue chromatic sensitivity.

(S – (L + M)) thresholds increase in the periphery and become more dependent on stimulus size. Subject I's chromatic sensitivity measured in the upper and lower right quadrants is similar to what is expected

in normal subjects when using the same stimulus conditions, but his foveal colour thresholds are well outside the normal range (Figure 4). Since peripheral chromatic sensitivity has rarely been measured in clinical studies, the possibility remains that many of the patients diagnosed with cerebral achromatopsia using foveal colour vision tests may have shown different findings in peripheral vision. As in subject I, patients may show abnormal foveal sensitivity and normal results in the periphery: the converse might be true in cases of hemi-achromatopsia: such findings challenge our understanding of the representation of the visual field in areas of visual cortex processing chromatic information. Of even greater significance is the observation discussed below where sensitivity is reduced or lost selectively for certain colour directions and not others at the same location in the field: such findings provide support for the notion that opponent colour mechanisms no longer apply in the processing of colour information in the damaged areas of visual cortex.

Experiment 3: Colour Assessment in the Periphery of Subject I's Visual Field

In spite of subject I's normal achromatic vision his use of colour signals in central vision is affected severely by the bilateral cortical lesions. Further tests were carried out to investigate subject I's chromatic sensitivity in the periphery of his visual field. The coloured stimulus was presented at each of the three peripheral locations shown in Figure 2. The stimuli were $6^\circ \times 6^\circ$ in diameter and were centred 6° horizontally and 11° vertically away from fixation in the three quadrants, as shown in Figure 2. Data for a normal trichromat were also obtained at the corresponding locations in the lower left and right visual field quadrants. The results shown in Figure 5A reveal almost normal chromatic sensitivity in both the upper right and lower right quadrants. The more rapid increase in (L – M) than (S – (L + M)) thresholds with eccentricity was also observed in other subjects. What is unexpected and surprising is the grossly asymmetric increase in thresholds towards the "green" region of the spectrum locus in the lower left quadrant (Figure 5B).

The results for subject I showed significantly higher colour discrimination thresholds at the fovea (see Figure 4) when compared to statistical limits based on normal trichromatic colour vision. In addition, in a peripheral location subject I exhibits selective loss for "green" sensitivity, as if colours within this category have been more severely affected. Although subject I's chromatic sensitivity in these peripheral locations

FIGURE 5 Colour detection thresholds measured in the periphery of the visual field at the locations shown in Figure 2. Data for a normal trichromat and for I's upper and lower right quadrants are shown in panel A. Panel B shows I's results in the lower left quadrant together with those measured in the lower right field (for comparison).

is grossly normal, the specific loss of "green" sensitivity is specific to a particular location in the visual field and was only present in the lower left quadrant. Such asymmetric loss of chromatic sensitivity has not been previously reported in any pathological disorder in humans. The results suggest that localised lesions in the ventral region of occipito-temporal cortex can cause loss of chromatic sensitivity that is both non-uniform over the visual field and may also be colour specific.

DISCUSSION

Chromatic sensitivity in human peripheral vision has not been extensively studied. Results in normal trichromats show a systematic increase in thresholds with stimulus eccentricity and decreasing ellipticity at larger eccentricities (i.e. the ratio of the major to the minor axis of the chromatic threshold ellipse approaches unity). Thresholds measured in opposite directions along any line that passes through background chromaticity, however, remain equal and this reflects the opponent nature of the mechanisms that generate chromatic signals.

The patient with cerebral achromatopsia examined in this study can make use of chromatic signals, but has much reduced chromatic sensitivity. The subject also exhibits markedly different thresholds when the task involves detection of colour changes, or the processing of structure from colour, as described in an earlier article.¹⁴ However, by probing localised regions of the visual field we have been able to show that not only is the loss of sensitivity not uniform over the visual field but, in addition, subject I shows

colour-specific loss that spares complementary colours in one of the three tested locations, with symmetric loss of colour sensitivity in the remaining quadrants.

Our findings suggest that discrete lesions in ventral occipito-temporal cortex can, in some cases, cause chromatic sensitivity loss in the contralateral hemifield that is "colour" specific. This pattern of response resembles the properties of some V2 cells that exhibit narrow colour tuning and respond specifically only to certain colours.^{16,17,18}

The exact location and extent of the lesion in I is difficult to establish from available scans but the lesion affects mostly the lateral portion of the fusiform gyrus that has been identified with the human V4.^{19,20}

It was not possible to relate more precisely the extent and position of subject I's corresponding lesion to the grossly abnormal processing of chromatic signals. However, the asymmetric findings for the lower left quadrant suggest independent higher-order processing of single colour categories that may be selectively damaged.

Further work is needed to examine the conditions, which will produce this entirely novel type of colour defect—particularly in terms of lesion location. The authors are not aware that any similar report has appeared since this case was studied approaching 20 years ago. Advances in imaging methodology and improved techniques for testing chromatic sensitivity in the peripheral visual field as well as better understanding of the underlying physiological mechanisms²⁰ have led us to consider it timely to report this case in detail in preparation for a more extensive study.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

Note: Figures 2 and 4 of this article are available in colour online at www.informahealthcare.com/oph

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