6. Unique hues in heterozygotes for protan and deutan deficiencies

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Abstract

We measured the wavelengths of unique blue, green, and yellow in a population of normal women and carriers of different forms of red–green deficiencies. In the case of unique blue and green none of the groups of heterozygotes differed significantly from normals. In the case of unique yellow, at a retinal illuminance of 800 td, carriers of protanomaly made settings at significantly shorter wavelengths than any other group. Carriers of deuteranomaly, deuteranopia and protanopia did not differ from normal individuals. At 20 td none of the groups of heterozygotes differed from normal. Our results offer no support for the hypothesis of Cicerone (1987) that the wavelength seen as unique yellow is determined primarily by the relative numbers of L to M cones. Nor do we have any evidence for the report of Donders (1884) that the settings of unique yellow vary with Rayleigh matches.

Introduction

In the circle of hues there are four colours, the *Urfarben* of Hering (1878), that appear phenomenologically unmixed: unique blue, green, yellow, and red. For example, a unique yellow contains neither greenness nor redness. Hering further noted that the four unique hues are organized into two opponent pairs and that we never experience mixtures of the two components of one pair. Thus we never see a reddish-green or a bluish-yellow.

What are the physiological or ecological determinants of the unique hues? Why do observers differ in the wavelengths they choose for unique yellow, green and blue? There is little agreement on the answers to these questions or indeed on the domain in which the answers are to be sought. Historically, the unique hues have been taken to correspond to the null-points of Hering's putative opponent mechanisms: unique red and green are seen when the yellow–blue process is in equilibrium, and the red–green mechanism is polarized; unique blue and yellow are seen when the red–green (R–G) mechanism is in equilibrium and the blue–yellow (B–Y) system is polarized (Hering, 1878; Hurvich and Jameson, 1957).

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In the case of unique yellow, two current hypotheses relate individual judgments of unique yellow to properties of the cones. The first is the *cone ratio hypothesis*. Cicerone (1987) suggested that the variability in unique yellow derives from individual variability in the relative numbers of M and L cones. Within the tradition of opponent colour theory, unique yellow is seen when

$$[N_L \cdot k_L \cdot L(\lambda)] - [N_M \cdot k_M \cdot M(\lambda)] = 0 \qquad (1)$$

where N_L and N_M are the relative numbers of L and M cones, k_L and k_M are neural weighting factors, and $L(\lambda)$ and $M(\lambda)$ are the spectral sensitivities of the L and M cones at wavelength λ. Cicerone proposed that the numerosity of each class of cone is the main factor that determines the overall weighting of those cones at the input to the opponent channel. The greater the proportion of L cones, the shorter the wavelength of unique yellow.

The second hypothesis is the *cone sensitivity hypothesis*. Donders (1884) reported a strong correlation between unique yellow and Rayleigh matches: the more red a subject required in the match the shorter the wavelength of unique yellow. Westphal (1910) termed this relationship 'Donders' rule'. If this law holds, it suggests that the wavelength of unique yellow depends on the spectral positions of the M and L photopigments, since the latter variations are now recognized to be major determinants of Rayleigh matches (Winderickx *et al.*, 1992; Neitz and Neitz, 1994). In an analysis of anomalous trichromacy, Pokorny and Smith (1977) postulated that unique yellow corresponds to the wavelength that produces the same quantum catch in the long- and middle-wave cones as does white light. Their hypothesis could today be extended to relate the variations in unique yellow in normals to the known polymorphisms of visual pigments. An interesting question arises as to what white is the reference white. One possibility is that it is the average illuminant of the subject's recent environment (Mollon, 1982).

In the case of the other unique hues, theories of the relationship between receptor variations and phenomenological equilibria are less well developed. This is perhaps because the short-wave cones, as well as the long- and middle-wave cones, are thought to determine unique red, green and blue.

Heterozygotes for red–green deficiencies offer an interesting approach to receptoral theories of the variation in unique hues. A random process of X-chromosome inactivation determines which of the two X-chromosomes is expressed in any individual cell of a woman's body (Lyon, 1972). Thus the retinal mosaic of a heterozygote is thought to contain a subset of cones that express the abnormal chromosome that her colour-deficient son inherits. This assumption is consistent with the finding that heterozygotes exhibit altered luminous–efficiency functions (Schmidt, 1934; Jordan and Mollon, this volume) and with the finding that very small (1 min) probes reveal colour-deficient patches in the parafoveas of heterozygotes (Born *et al.*, 1976).

A test of the cone-ratio hypothesis is offered by carriers for dichromacy. Carriers of protanopia are expected to have a reduced number of long-wave

cones and carriers of deuteranopia a reduced number of middle-wave cones. According to the cone ratio hypothesis these variations in cone numerosities should alter the weightings of L- and M-cone inputs to opponent channels and thus lead to clear differences in unique yellow and perhaps in the other unique hues.

According to the pigment-shift model of anomalous trichromacy (Pokorny and Smith, 1977), the anomalous retina contains cones that differ in sensitivity from the normal L and M cones. If these anomalous pigments do exist, then carriers of anomalous trichromacy offer a test of what we have above called the cone sensitivity hypothesis; a subset of their cones should express the shifted photopigment that their anomalous sons inherit, and their unique hues should be altered.

Method

The subjects were 14 normal controls and 37 carriers (22 deutan and 15 protan) whose genotypes were inferred from their sons' phenotypes. Sons and mothers were tested on a battery of colour vision tests including the Nagel anomaloscope and the Farnsworth-Munsell 100-hue test.

We measured unique green, yellow and blue for each subject. Since unique-red is extra-spectral (Dimmick and Hubbard, 1939) we did not attempt its determination with our present apparatus. The stimulus was a 1° circular field that was flashed on for 1 sec on a dark background. The interstimulus interval depended upon the spectral separation of successive stimuli but had a minimum value of 4 sec when the stimuli were 1–2 nm apart. Before the subject started, the concept of each unique hue was explained to her, and the corresponding spectral range was shown in Maxwellian view to familiarize her with the stimuli. Our Maxwellian-view optical system incorporates a computer-controlled monochromator with an integral stepping motor. Thus it was possible to determine the unique hues by a staircase method. The subject indicated by push buttons the direction in which a given stimulus departed from a unique hue, e.g. 'too red' or 'too green' in the case of unique yellow. First a two-staircase procedure with starting values 50 nm apart was used to familiarize the subjects with the task and to obtain an initial estimate of the unique hue. Subsequently, four staircases, centred on the subject's initial mean setting, were randomly interleaved. The last six reversals of each independent staircase were used to estimate the wavelength of the unique hue and the values plotted in the figures are the means of the four estimates.

In our first experiment, the luminance of the stimuli varied with the output of the monochromator at different wavelengths. For unique blue the value was approximately 20 td, for unique green approximately 100 td, and for unique yellow approximately 800 td. In a second experiment, we repeated the measurements for unique yellow, now holding the troland value at 20 td: under computer control, adjustments of a neutral density wedge were yoked to

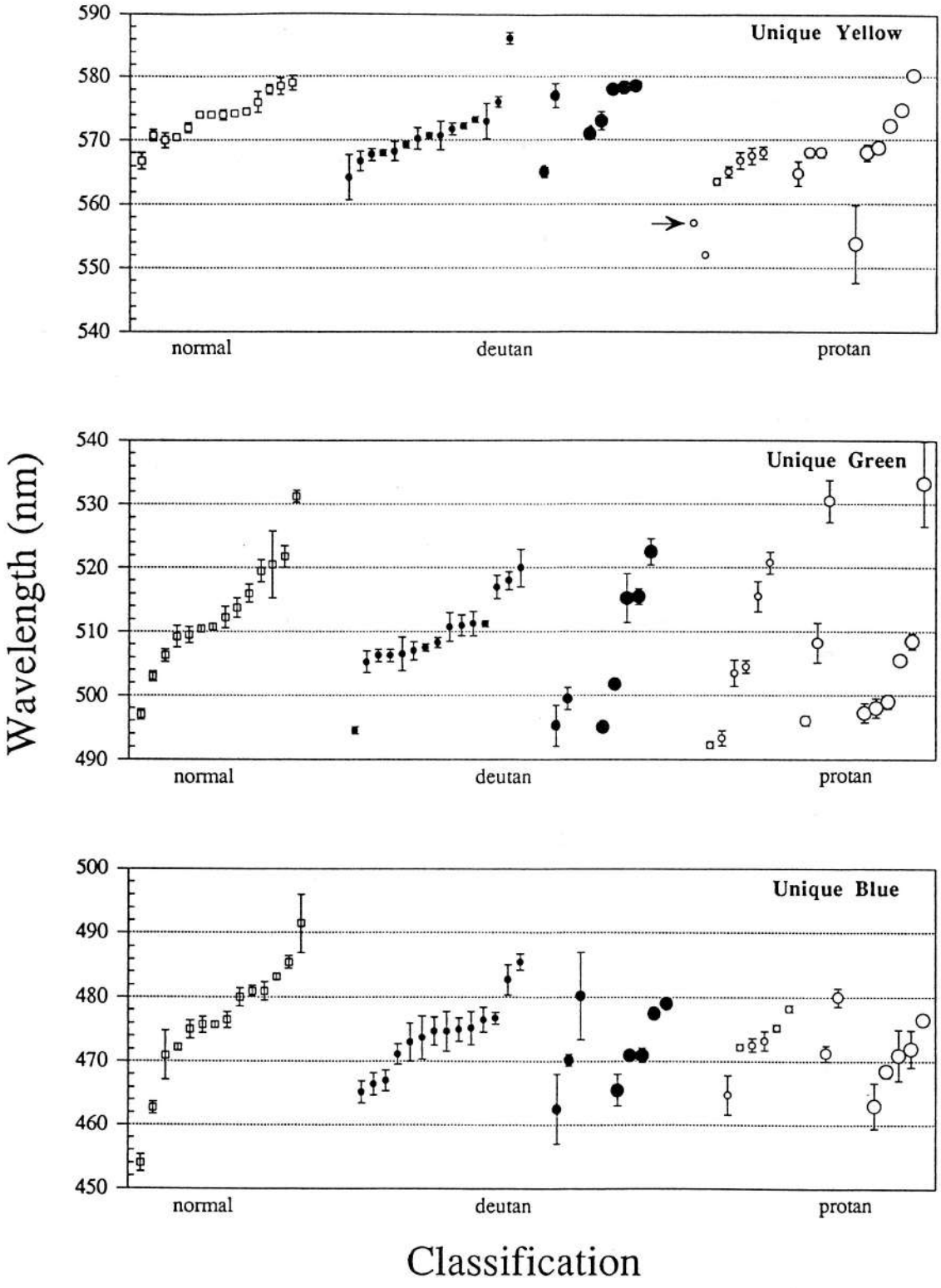

Fig. 1. The wavelengths seen as unique yellow (top panel), green (middle), and blue (bottom panel) for normals (open squares), deutan carriers (filled circles), and protan carriers (open circles). Gradation in symbol size represents the degree of deficiency of the abnormal gene: small symbols represent carriers for anomalous trichromacy, large symbols carriers for dichromacy, and intermediate symbols carriers for extreme anomalies. The error bars are standard deviations. The carriers for protanomaly set the wavelength of unique yellow significantly shorter than any other group.

variations in wavelength, so as to hold constant the monochromator output.

In addition, Rayleigh matches were measured with the Nagel anomaloscope (Schmidt and Haensch) and relative sensitivity to long and middle wavelengths was measured with the OSCAR test (see Jordan and Mollon, this volume).

Results

Figure 1 shows a summary of all unique hue settings for the first experiment: unique yellow at the top; unique green in the middle; and unique blue at the bottom. The ordinate shows wavelength in nm. Open squares represent the settings of the normal controls, filled circles represent the deutan carriers and open circles represent the protan carriers. Within both groups of carriers the symbols are graded in size: the smallest symbols indicate the carriers of simple anomaly, the intermediate ones the carriers of extreme anomaly and the largest symbols the carriers of dichromacy.

There are no significant differences between carriers and normal controls in the settings of unique green or of unique blue. In the case of unique yellow, a one-way analysis of variance for normal, protan and deutan groups showed a significant effect of group ($F = 6.09$, $df = 2$, $p = 0.004$). This effect is due to the carriers of protanomaly, who set the wavelength of unique yellow very significantly shorter than the normal group ($p < 0.001$). Note that one carrier of protanomaly sets the wavelength of unique yellow at a remarkably short value of 552 nm. When retested 6 months later, with the output of the monochromator held constant at a similar troland value, she still made a setting of 557 nm. Her second setting is represented by the arrow in Figure 1. (This subject recalls the female observer of Akita *et al.* (1982), who made a Rayleigh match within the normal range but chose a wavelength of 536 nm for her unique yellow. That subject might also be a protanomalous carrier.) Carriers of protanomaly were the only divergent group: neither the dichromat carriers nor the carriers of extreme anomaly nor the carriers of simple deuteranomaly differed from the normal women.

Figure 2 shows the results for the second experiment on unique yellow, where the stimulus was held constant at the lower value of 20 td. There is now no significant difference in the wavelength judged to be unique yellow between any group of carrier and the normal controls. The carriers of protanomaly have shifted towards longer wavelengths. Despite the absence of differences in settings, the subjective comments of some subjects in this experiment may be worth mentioning. Some carriers of dichromacy reported an almost colourless sensation at wavelengths near their final setting. Two carriers of deuteranomaly (whose settings lie at the short-wave end of the distribution) said that the hue they ended up with was neither red nor green and yet not pure yellow: one did not know what to call the colour, the other said that, if forced, she would say a 'bluish-yellow' (a forbidden colour in the Hering scheme).

Figure 3 shows the relationship between settings on the OSCAR test and

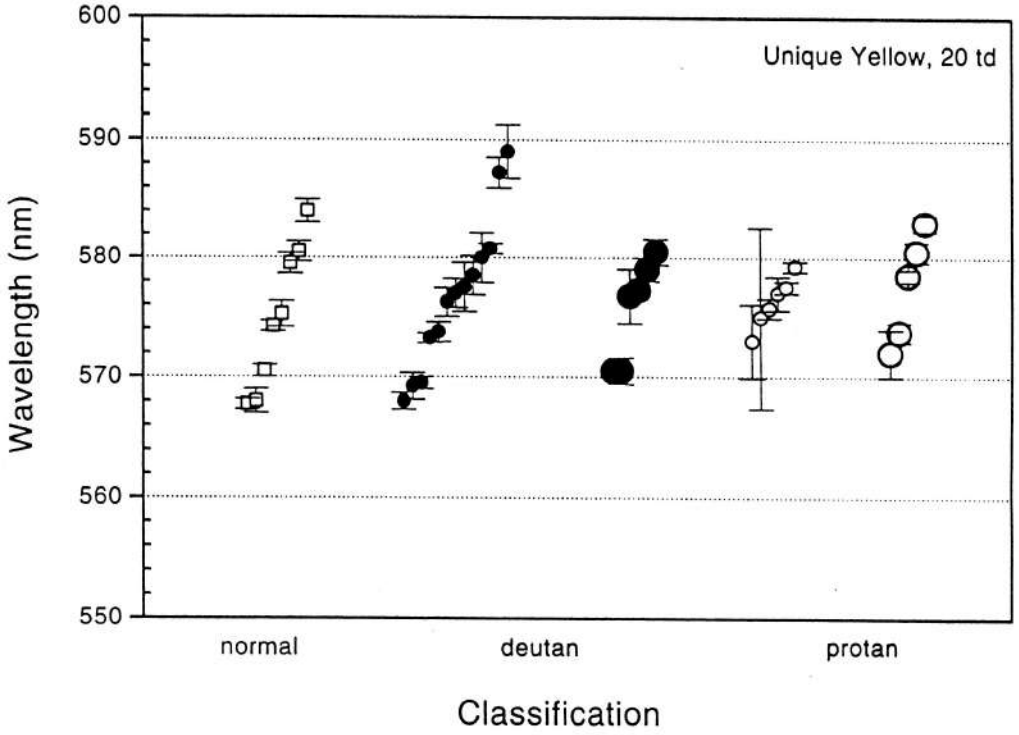

Fig. 2. The unique yellow settings at a retinal illuminance of 20 td for normals and carriers. There is no significant difference in the settings between any group. Symbols as in Figure 1

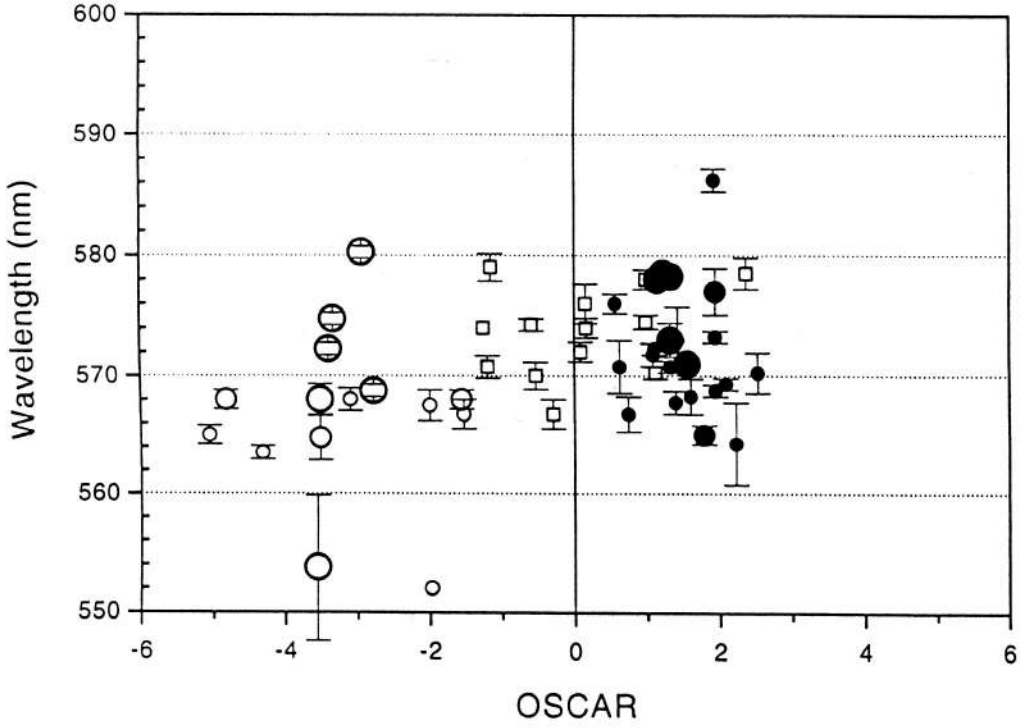

Fig. 3. Relationship between the wavelengths seen as unique yellow and the subjects' OSCAR settings. Symbols are used as before. Zero on the abscissa represents the mean OSCAR setting of normals. As expected carriers for protan deficiencies make settings on the left-hand side of zero, indicating a lowered sensitivity for long wavelengths (Schmidt's sign). Carriers for deutan deficiency make settings on the right-hand side of zero, indicating a lowered sensitivity for middle wavelengths; but there is considerable overlap with the normals. Symbols as in Figure 1

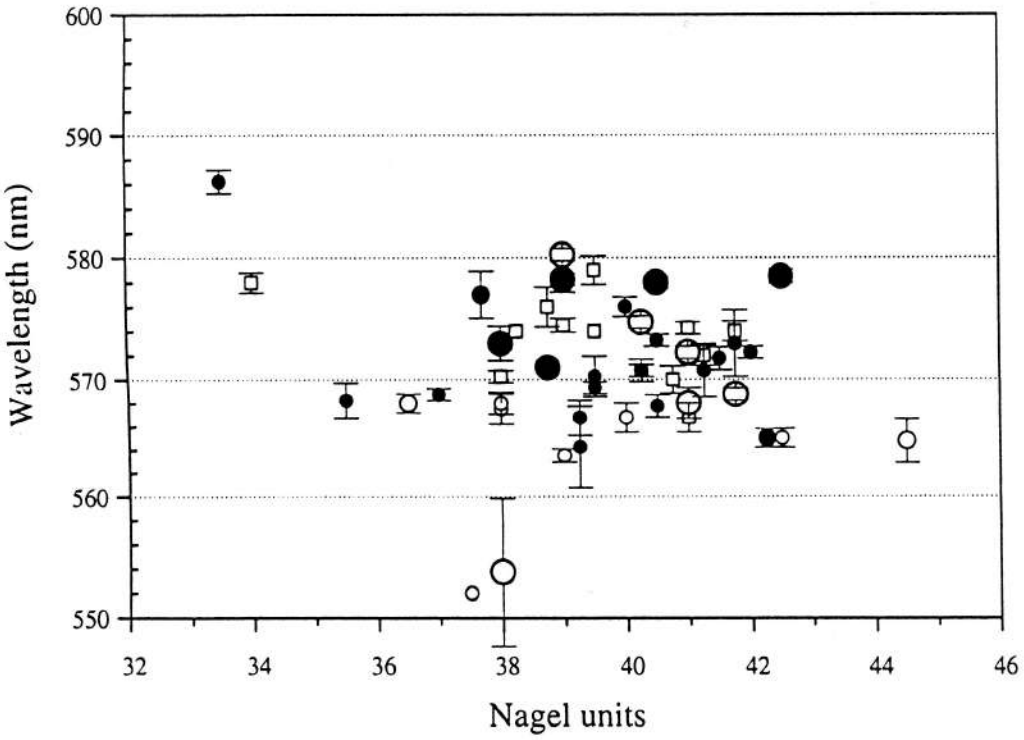

Fig. 4. The relationship between the wavelengths seen as unique yellow and the subjects' Rayleigh matches. Rayleigh matches are expressed in raw Nagel units. There is no significant correlation between the two variables.

estimates of unique yellow in the first experiment. As we show elsewhere (Jordan and Mollon, this volume), protan carriers tended to make settings on the left-hand side, indicating a reduced sensitivity to long wavelengths (Schmidt's sign), whereas deutan carriers tended to make settings on the right-hand side, indicating a reduced sensitivity to middle wavelengths. Normal individuals made settings in the middle range. However, there is only a modest, although significant, correlation between unique yellow and OSCAR settings ($R = 0.38$, $p = 0.005$). Similar analyses showed no significant relationship between the other unique hues and OSCAR settings.

Figure 4 shows the relationship between the subjects' Rayleigh matches and their settings of unique yellow in the first experiment. The abscissa is in Nagel units: subjects on the right need more red in the mixture to match the standard yellow; the ordinate shows the wavelength seen as unique yellow. There was no overall correlation between Rayleigh matches and the wavelength seen as unique yellow ($R = -0.13$, $p = 0.34$) and there are no obvious trends within any of the individual groups of carrier or the normal controls. Similar analyses showed no relationship between Nagel matches and the other two unique hues that we tested. Our result for unique yellow is concordant with that of Hailwood and Roaf (1937).

Discussion

Cone ratio hypothesis

Our results offer no support for the hypothesis that the relative numbers of L and M cones determine the wavelengths of unique hues in general and unique yellow in particular. If the cone-ratio hypothesis of Cicerone were correct, then we should expect carriers of protanopia (who are thought to have reduced numbers of L cones) to set unique yellow at longer wavelengths than normal controls, whereas carriers of deuteranopia (who are thought to have fewer M cones) should make settings at shorter wavelengths than normals. In fact, the two kinds of carrier of dichromacy did not differ from normal individuals in our study, even though they make clearly different settings on the OSCAR test, a flicker-photometric measure of the type often taken to reflect cone ratios (de Vries, 1948).

If Alpern's account of anomalous trichromacy is correct – if, that is, the residual discrimination of anomals depends on two types of normal M cone or two types of normal L cone (Alpern and Moeller, 1977) – then our argument can be extended to include the carriers of anomalous trichromacy. For the retina of the carrier should contain only normal cones, but in abnormal proportions. Here again there is no support for Cicerone's proposal. In fact, at 800 td the carriers of protanomaly differed from normals in the direction opposite to that predicted from the cone-ratio hypothesis.

One counter-argument, put to us by J. Pokorny, would be that the fovea of the carrier contains relatively coarse patches of normal and defective retina and that settings of unique yellow (unlike flicker-photometric settings) are achieved only by the colour-normal patches. Against this argument may be cited the report of Grützner *et al.* (1976), who probed the retina with brief, tiny stimuli and found that dichromat carriers, though abnormal in the parafovea, were able to correctly identify the colours of 1 min targets for all eccentricities less than 2°: the implication is that cones expressing alternative X-chromosomes are well intermingled in the carrier's foveola and are not segregated in large patches. A second counter-argument, put to us by a referee, requires one to assume the replacement hypothesis for dichromacy, i.e. to assume that those cones that should have become L cones became M cones in the protanope and in the corresponding carrier. If these extra M cones contributed equally to the two expressions of equation 1, then unique yellow might be left unchanged in the carrier. This hypothesis requires the additional hypothesis that the extra M cones are neither labelled as M cones nor as L cones.

Cone-sensitivity hypothesis

In our population as a whole, we found no relationship between unique hues and the subjects' Rayleigh matches. In particular, we did not confirm the inverse relationship between unique yellow and Rayleigh match that was reported by

Donders (1884). (There is good reason to suppose that the result of Donders would be found for a sample of observers that included protanomalous and deuteranomalous trichromats as well as normal individuals, since anomalous individuals differ in the expected way in their settings of unique yellow (Westphal, 1910; Hurvich and Jameson, 1964; Pokorny and Smith, 1977).) Our result offers no support to hypotheses that relate individual differences in unique hues to variations in the spectral positions of the receptors. Our results for carriers of anomaly bear upon this issue in a more complicated way. In their actual settings of unique yellow, carriers of deuteranomaly did not differ from normals in either experiment: this result implies either that unique yellow is not affected by the presence of cones with abnormal spectral sensitivities, or that Alpern's hypothesis is correct and the retina of the deuteranomalous carrier contains only normal cones. In contrast, carriers of protanomaly, under the high-intensity conditions of the first experiment, differed systematically from normals, the shift in their settings being in the same direction as the shift seen in actual protanomalous observers (Hurvich and Jameson, 1964). This implies either that there are cones with abnormal spectral sensitivity in the retinae of heterozygotes for protanomaly or that their colour judgements are influenced both by a normal L/M channel and a channel that differences two types of M cones, the channel on which according to the Alpern hypothesis their sons rely. This conclusion must remain tentative, since the protanomalous carriers, like other carriers, did not differ from the normal individuals for the 20 td stimuli.

Acknowledgements

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