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## **Sex-related differences in chromatic sensitivity**

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**Running head: Chromatic sensitivity**

**Key words: colour vision, heterozygote, X-chromosome inactivation, L- cone polymorphism, CAD test**

## Abstract

Generally women are believed to be more discriminating than men in the use of colour names and this is often taken to imply superior colour vision. However, if both X-chromosome linked colour deficient males (~8%) and females (<1%) as well as heterozygote female carriers (~15%) are excluded from comparisons, then differences between men and women in red-green colour discrimination have been reported as not being significant (e.g., Pickford, 1944; Hood et al., 2006). We re-examined this question by assessing the performance of 150 males and 150 females on the Colour Assessment and Diagnosis (CAD) test (Rodriguez-Carmona, 2005). This is a sensitive test that yields small colour detection thresholds. The test employs direction-specific, moving, chromatic stimuli embedded in a background of random, dynamic, luminance contrast noise. A four-alternative, forced-choice procedure is employed to measure the subject's thresholds for detection of colour signals in 16 directions in colour space, while ensuring that the subject cannot make use of any residual luminance contrast signals. In addition, we measured the Rayleigh anomaloscope matches in a subgroup of 111 males and 114 females. All the age-matched males ( $30.8 \pm 9.7$ ) and females ( $26.7 \pm 8.8$ ) had normal colour vision as diagnosed by a battery of conventional colour vision tests. Females with known colour deficient relatives were excluded from the study. Comparisons between the male and female groups revealed no significant differences in anomaloscope midpoints ( $p=0.709$ ), but a significant difference in matching ranges ( $p=0.040$ ); females on average tended to have a larger mean range (4.11) than males (3.75). Females also had significantly higher CAD thresholds than males along the red-green ( $p=0.0004$ ), but not along the yellow-blue discrimination axis. The differences between males and females in red-green discrimination may be related to the heterozygosity in X-linked cone photopigment expression common among females.

## **Introduction**

Investigations designed to directly compare colour discrimination performance in males and females have been only rarely carried out and have yielded ambiguous or inconsistent results. On the one hand, gender differences are typically presupposed to be non-differentiating factors in colour vision. On the other hand, it is often assumed that, owing largely to social or cultural influences, women may be more critical in the use of subtle colour names and be more interested in distinguishing colours than men. Indeed, such assumptions, commonly appear in introductory textbooks, even though they have not been well documented or validated experimentally (for an insightful review, see Reynolds, 1966).

Within the context of cognitive and social phenomena, several studies have compared male and female use of colour vocabulary and have indicated that women access a larger repertoire of words to describe sets of colour stimuli (Rich, 1977; Swaringen et al., 1978; Thomas et al., 1978; Nowaczyk, 1982; Simpson & Tarrant, 1991). For instance, Furbee et al. (1997) found that females require an increased scaling dimension relative to males in order to represent the red-yellow-green colour axis. And, Bimler et al. (2004) have shown that males tend to attend more to a lightness axis and less to a red-green axis than females; as well as being less precise in their judgments (Bimler & Kirkland, 2002; Bimler et al., 2004). Other studies have shown that women perform better than men at matching memory colours (Perez-Carpinell et al., 1998). Differences in colour preferences between males and females have also been demonstrated (Saito, 1994; Saito, 1996; Saito, 1999; Hurlbert & Ling, 2007); as have differences in unique-hue judgments for yellow and red (Kuehni, 2001).

However, despite such indications that males and females may differ perceptually or cognitively in their colour experience, it is uncertain as to whether these differences reflect actual differences in their underlying chromatic sensitivity capabilities. It may simply be that, as a result of divergent patterns of socialization for males and females, a greater verbal skill and interest in colours is instilled in women (Anyan & Quillian, 1971). Intriguingly, however, an underlying physiological explanation is possible for why there should be sex-linked differences in colour vision: sexual dimorphisms in the genes that encode the X-linked cone photopigments that manifest as different frequencies of expression in men and women (see, for instance, Pardo et al., 2007).

### *Genetic and other factors underlying differences in colour vision discrimination*

Variability in chromatic sensitivity in normal trichromats can be attributed to a number of factors. The best known sources are the inherited X-chromosome-linked or red-green colour

vision deficiencies that arise from small genetic mutations in the long-wavelength sensitive (L-) and / or middle-wavelength sensitive (M-) cone photopigment coding genes. The prevalence of red-green colour vision defects amongst men is about 8%, whilst in women it is less than 1% (for a recent review, see Sharpe et al., 1999). The nature of the defect ranges from mild to extreme; but all generally manifest as poorer red-green colour discrimination. Additionally, about 15% of women, known as heterozygote carriers, inherit an X-chromosome carrying an abnormal L/M array from one parent and an X-chromosome carrying a normal L/M array from the other. Owing to random X-chromosome inactivation, one of their two X-chromosomes is transcriptionally silenced in each L/M cone cell. Thus such carriers may partly share the colour-defect (Pickford, 1944; 1947), resulting from a loss of a normal photopigment genes on one of their X-chromosomes, or in the case of an inherited red-green (L-M) hybrid or abnormal photopigment gene on one of their X-chromosomes, may even benefit from the presence of a fourth expressed cone pigment (De Vries, 1948; Jordan & Mollon, 1993). In the latter case, this could lead to tetrachromatic colour vision and enhanced colour discrimination along the red-green colour axes. If such heterozygotes are not screened for and eliminated from the female population, they can distort any gender comparisons (Pickford, 1947).

There is also a more subtle source of common heterozygosity among females in photopigment expression. Several studies have demonstrated that the pigment genes of colour normals are polymorphic with regard to the amino acid encoded at codon/residue 180 (Winderickx et al., 1992; Neitz et al., 1993; Sanocki et al., 1994; Sharpe et al., 1998). Current estimates suggest that this polymorphic variation is not equally distributed; among human L-cone pigment genes approximately 56.3% have serine and 43.7% have alanine at codon 180, whereas among M-cone pigment genes, approximately 6% have serine and 94% have alanine (Sharpe et al., 1999). The serine/alanine polymorphism on the L-cone pigment gene, which is by far the most commonly observed, results in the serine-containing L-cone pigment being red-shifted by more than ~2.7 nm with respect to the alanine-containing one (Sharpe et al., 1999). Therefore, the spectral separation with respect to the mean peak sensitivity of the M-cone pigment will vary depending whether the L(ser<sup>180</sup>) or L(ala<sup>180</sup>) variant is expressed. This in turn may result in variations in chromatic discrimination (Neitz et al., 1991; Nathans et al., 1992; Asenjo et al., 1994). Indeed, Winderickx et al. (1992) have shown that the spectral separation between the alanine and serine L-cone variants alone can account for measured differences in Rayleigh (red-green) equation anomaloscope matches, consistent with the bimodality observed in male populations (Neitz & Jacobs, 1986). And, Pardo et al. (2007) have argued that the frequency of expression of the polymorphic variants of the L-cone photopigment in males and females can be used to explain significant gender

differences in colour vision discrimination, although they themselves did not control for heterozygote carriers of colour vision deficiencies in their study.

Other factors underlying differences in chromatic sensitivity that may or may not be genetically related, include slight differences in the optical density of the cone photopigments (Neitz et al., 1999) and / or variations in post-receptoral amplification of cone signals (Barbur, 2003). These factors can account for much of the observed variability both within 'normal' trichromats and amongst colour deficient observers with the same genotype (Neitz & Jacobs, 1986). However, such variability is generally assumed to be non-gender dependent.

#### *Previous gender comparisons of colour discrimination performance*

The evidence from the largest population sample (Pickford, 1947) suggests that there is no difference between males and females in colour discrimination when X-linked heterozygote female carriers as well as male and female colour deficient observers are excluded from comparisons. Using a rotating disk experiment, Pickford (1947) measured red-green and yellow-blue sensitivities in 191 men and 185 women with normal colour vision. No significant gender differences were found in yellow-blue thresholds. However, a higher proportion of women than men had more than twice the modal red-green threshold range, although the modal threshold was the same for both sexes. The number of women with twice the modal red-green matching range was similar to the number of heterozygote women expected in the random population sample. About 13.57% of the 185 women in the sample were expected to be heterozygous for sex-linked defects based on the 7.31% prevalence of red-green defectives among men in the region of Glasgow and western Scotland (Vernon & Straker, 1943). Pickford, therefore, concluded that '*if you control for heterozygotes, women are just as good judges as men*'. However, he assumed that all red-green heterozygotes have poorer red-green thresholds than normal males because of slight red-green weaknesses due to incomplete recessive inheritance. This assumption, however, has not been supported by a recent study. Hood et al. (2006) argue that chromatic discrimination along the red-green axis is impaired in female carriers of deutan deficiencies (a condition that reflects abnormality or absence of the M-pigment), but not in carriers of protan deficiencies (which reflects abnormality or absence of the L-pigment). Thus, Pickford's study is inconclusive as to whether gender differences occur in red-green discrimination. As is another, more recent study (Birch et al., 1991), which also did not control completely for the presence of heterozygotes. Using the red-green Rayleigh equation on a Nagel Type 1 anomaloscope, they also found the red-green matching range to be slightly larger in women than in men; indicating slightly poorer performance, which might be explained by the presence of

undetected female carriers. Pardo et al. (2007) also effectively used a Rayleigh equation metameric match in a population of 31 men and 32 women (who were not screened for heterozygotic female carriers). On average, women required significantly more red light in the mixture than men, but the standard errors of their settings did not differ.

Other studies have addressed gender differences in discrimination, but have used different measurement procedures and/or much smaller sample sizes. For instance, Costa et al. (2006) measured both monocular and binocular thresholds along the protan, deutan and tritan colour confusion axes. In a small population, which was not screened for female heterozygote carriers, they found no significant differences between males and females. In another study, Jameson et al. (2001) required their subjects to divide a diffracted spectrum stimulus into bands of colour. They argued that on average a specific subgroup of women heterozygous for X-linked photopigment genes (i.e., female heterozygous carriers), who presumably may be tetrachromats (the molecular genetic analysis is unclear), perceived a greater number of distinct bands than either male or homozygous female controls.

Given the discrepant results, we think that the question of whether there are differences between men and women in colour discrimination merits closer examination. Because gender differences may be small and hence easily overlooked, we employed an efficient and sensitive method, the Colour Assessment and Diagnosis (CAD) test (Rodriguez-Carmona, 2006). The CAD test allows us to measure small differences in chromatic discrimination in various directions in colour space. Additionally, we compared the CAD results with those measured in the same observers on a conventional colour test, the Rayleigh red-green equation of the Nagel anomaloscope. We excluded from our sample colour deficient men and women, and those females that could be identified from the family history of colour deficiency as being heterozygote. Because the number of suspected heterozygotes (2) was smaller than the estimated number (22) for our sample size (152), we also applied a rigorous statistical procedure to eliminate the influence of undetected heterozygotes upon our gender comparisons.

## **Materials and Methods**

### *Observers*

A random population sample of 302 observers participated in this study; 150 males and 152 females. Although all observers performed the CAD test, only a smaller population of 225 observers, 111 males and 114 females, completed the red-green matches on the Nagel Type 1 anomaloscope. The mean age for females was  $26.7 \pm 8.8$  years, with a median of 24

years; the mean age for males was  $30.8 \pm 9.7$  years, with a median of 28 years. The observers were recruited largely from the university environment and all had normal colour vision according to standard clinical tests (Ishihara and AO-HRR pseudoisochromatic plates) and normal or corrected-to-normal visual acuity with no recorded pathology or ocular abnormalities. Subjects found to have a family history of colour vision deficiency, as revealed through clinical questioning, were excluded from the study. All experiments were conducted in accordance with the tenets of the Declaration of Helsinki (Code of Ethics of the World Medical Association), and the study had the approval of the research and ethical committee of City University.

#### *Assessment of chromatic discrimination*

Colour vision was assessed using the CAD test and the Nagel Type 1 anomaloscope (Schmidt and Haensch GmbH and Co., Berlin, Germany). The anomaloscope presents a circular bipartite field, in which the top half is illuminated by a mixture of spectral red and green wavelengths; 670 and 546 nm, and the lower half illuminated by spectral yellow, 589 nm. Two control knobs are used, one to alter the red/green colour mixture ratio of the top field, and the other to alter the intensity of the spectrally yellow lower field. The limits of the matching range were determined first by allowing the observer to make several initial matches. Then the examiner set a predetermined red/green mixture ratio and the observer was instructed to adjust only the intensity of the yellow half-field in order to achieve the best possible match. Using the same procedure, a number of red/green mixture ratios were investigated in order to establish the extreme ends of the range when the subject was no longer able to match the two hemifields. The number of scale units between the matching limits was recorded as the matching range. The midpoint was then recorded as the midpoint value of the red/green matching range.

The CAD test employs moving, chromatic stimuli embedded in a background of dynamic luminance contrast (LC) noise. This technique isolates the use of colour by masking any residual LC signals in the colour defined stimulus (Barbur et al., 1994; Barbur, 2004). This is particularly important in subjects with unusual L to M cone ratios and / or cone pigments that differ significantly in spectral responsivity to the standard normal templates. The observer's task is to report the direction of the colour-defined moving stimulus using a four-alternative forced-choice method. Chromatic displacement thresholds are measured along 16 randomly interleaved directions in CIE (Commission Internationale d'Eclairage) 1931 colour space. These directions were chosen to correspond broadly to the red-green (RG) and yellow-blue (YB) colour sensitivity axes (see Fig.1). The staircases followed a two-down,

one-up rule (Levine, 2000) and terminated after 12 reversals, taking approximately 12 minutes to complete. The statistical limits for the 'standard normal' (SN) observer on the CAD test is based on 240 normal trichromats (age range 14-60; mean =  $30 \pm 10$  years). The measured thresholds were used to calculate the 2.5% and 97.5% limits and the medians for both RG and YB discrimination (Rodriguez-Carmona et al., 2005). The data for the SN observer provide an efficient way of assessing the severity of colour vision loss, i.e. an observer with a RG threshold of 2 SN units requires twice the colour signal strength that is needed by the average standard CAD observer.

## Results

Figures 2A and 2B display, respectively, the distribution of midpoint values and the size of the matching range, for the red-green match on the Nagel anomaloscope. A statistical comparison between the anomaloscope results for males and females revealed no significant difference in their average midpoint values ( $p=0.709$ ,  $df=203$ ; two-sample *T*-test).

However, there is a significant difference between the sizes of their matching range; with males having slightly smaller ranges ( $p=0.040$ ,  $df=220$ ; two-sample *T*-test), implying superior red-green colour discrimination. These results are largely consistent with those of previous studies (e.g. Birch et al., 1991), although they do not reveal subgroups within the male population that has been previously reported as being associated with the alanine/serine polymorphic variation in the L-cone photopigment (Neitz & Jacobs, 1986; Winderickx et al., 1992).

Figure 3 shows the distribution in YB and RG colour discrimination thresholds in SN CAD units for the whole population and then separately for males and females. The YB and RG thresholds do not strictly follow normal distributions (as revealed by normality tests; Anderson-Darling, Ryan-Joiner, Kolmogorov-Smirnov). Thus, non-parametric statistics were applied (Mann-Whitney Test) revealing no significant difference in YB chromatic discrimination between males and females ( $p=0.4115$ ). The median (inter quartile range) YB thresholds units for males and females are 0.991 (0.294) and 1.026 (0.316), respectively. However, a comparison between the male and female RG chromatic discrimination thresholds did reveal a significant difference ( $p=0.0004$ ); with women on average having larger RG thresholds than men. The median (inter quartile range) RG thresholds units for males and females are 0.968 (0.271) and 1.054 (0.254), respectively.

It is possible that the poorer group performance in RG colour discrimination of females when compared with males can be attributed to the presence of heterozygote carriers of

either deutan or protan defects who remained undetected by our screening procedures. Although all females were specifically asked about their family history of colour vision deficiency, only two had known relatives with red-green defects (and were subsequently excluded from the analysis). This small number of heterozygote females is very likely an underestimate. Unfortunately, molecular genetic analysis was not available to determine the remaining females in the sample that were probably heterozygotes. However, it is possible to estimate the expected numbers of women carriers of deutan and protan deficiency in our random population sample of 150 women, using the known prevalence of ~6% and ~2%, for deutan and protan defects in hemizygous male (Sharpe et al., 1999). The expected numbers of women carriers of deutan and protan defects would be 16 and 6, respectively<sup>1</sup>; that is, a total of 22 heterozygote carriers of colour vision deficiency (~15%).

Having estimated the total numbers of heterozygote carriers, we then decided to employ a statistical procedure to remove their possible deleterious influence upon the average female RG colour discrimination thresholds. We assumed that the females with the poorest RG thresholds were more likely to be heterozygotes (i.e. carriers for red-green colour defects). However, this general assumption is not supported by findings from Hood et al.'s (2006) study that showed some heterozygote females (i.e., carriers for deutan defects), but not others (i.e., carriers for protan defects) have poorer RG colour discrimination when compared with homozygotes. We, therefore, applied two different analyses: one which controlled for the possible influence of carriers of deutan defects and a second which controlled for the possible influence of carriers of either defect. Figure 4 provides a comparison between the RG thresholds of all the males and all females (left panel). The results of a Mann-Whitney test indicate a significant difference between males and females as described above ( $p=0.0004$ ). The middle panel then provides a comparison between the RG thresholds of the males and females, after excluding the ~11% females with the highest RG colour thresholds (i.e. the assumed 16 carriers for deutan defects who may have gone undetected in our screening procedures). The gender difference remains significant ( $p=0.0178$ ). Finally, the right panel shows a comparison of RG thresholds for males and females, after excluding the ~15% females with the highest RG colour thresholds (i.e. the assumed 22 carriers for either deutan or protan defects who may have gone undetected). Once again, the gender difference is significant ( $p=0.0484$ ).

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<sup>1</sup> The percentage of heterozygotes can be calculated using the equation  $2p(1-p)(1-d^2) + 2d(1-d)(1-p^2)$ , where  $p$  and  $d$  is the prevalence of protan defects (protanopia or protanomaly) and deutan (deutanopia or deutanomaly) defects, respectively, taken from (Sharpe et al., 1999).

## Discussion

Comparisons between our male and female groups reveal no significant difference in anomaloscope midpoints (this contradicts Pardo et al., 2007), but a significant difference in matching ranges. Females on average tend to have a larger mean range than males (see Fig. 2), which is consistent with an average poorer RG colour discrimination. Additionally, females have significantly higher thresholds than males along the red-green colour discrimination axis but not along the yellow-blue (see Fig. 3), as revealed by the CAD test.

We find that these differences in the CAD thresholds remain, even after making statistical corrections to exclude the influences of heterozygote female carriers from our comparisons; that is by removing 15% of the females (i.e., 22) in our sample of 150 with the highest red-green (RG CAD) thresholds. However, we must be cautious about drawing rigorous conclusions from such comparisons. Such corrections are difficult to evaluate, because we actually know neither how many heterozygote carriers we may have in the sample nor the nature of their heterozygosity (i.e., whether they are deutan or protan carriers). Statistically, 15% is only an estimate; the actual number of carriers could be smaller or greater. In fact, when we exclude one more female (i.e., the subject with the highest remaining RG CAD colour threshold; corresponding to 23 rather than 22 removals), the differences between males and females become statistically insignificant ( $p=0.0576$ ). This is within the limits of uncertainty as regards the number of possible heterozygote carriers within our female population.

On the other hand, we may be overcorrecting for the influence of heterozygote carriers. Hood et al. (2006) argue that only the carriers of deutan defects and not the carriers of protan defects have impaired red-green colour discrimination. Thus, it may not be necessary to remove the estimated number of protan carriers from our comparisons. To definitively decide the issue, however, we will need to combine our psychophysical sensitivity measurements with the relevant molecular genetic analysis of the same subjects. Thus, we cannot conclude yet on the basis of our study that females on average are poorer than males in red-green colour discrimination.

Nevertheless, it still prompts a speculative question. Although it is commonly assumed that women may, on average, have superior colour discrimination to men, they may in fact have poorer, when all heterozygotic female carriers are excluded from comparisons. Why should this be the case?

Hood et al. (2006) have offered one explanation for why deutan carriers may have slightly impaired red-green colour discrimination compared with males. They speculate these differences can be explained by differences in the relative numbers of L- and M-cones in heterozygous retinæ. Assuming an average cone ratio of L to M cones close to 2:1 (Carroll et al., 2000), there will be a biasing of cone numbers rendering unusual or extreme L:M cone ratios especially in deutan carriers (assuming equal X-chromosome inactivation). An imbalance of the two cone types may therefore impair chromatic discrimination (Hood et al., 2006).

Another possible explanation for observed differences in red-green chromatic discrimination sensitivity between males and females may be a sexual dimorphism in the expression of the “normal” X-linked cone pigment genes. As we discussed in the Introduction, several studies have demonstrated that the pigment genes of colour normals are polymorphic with regard to the amino acid encoded at residue 180 (Winderickx et al., 1992; Neitz et al., 1993; Sanocki et al., 1994; Sharpe et al., 1998). And, this polymorphism may lead to subtle perceptual differences in red-green colour discrimination (Neitz et al., 1991; Nathans et al., 1992; Winderickx et al., 1992; Asenjo et al., 1994; Sharpe et al., 1999). If among males, approximately 56.3% have serine and 43.7% have alanine at codon 180 of the L-cone pigment gene (Sharpe et al., 1999), then, owing to the presence of duplicate X-chromosomes in females, 49% ( $2 \times 0.563 \times 0.437$ ) of women will have about equal numbers of L(ser<sup>180</sup>) and L(ala<sup>180</sup>), about 32% of women will have L(ser<sup>180</sup>) / L(ser<sup>180</sup>) and about 19% of women will have L(ala<sup>180</sup>) / L(ala<sup>180</sup>). This X-linked polymorphism is expected to be subject to X-inactivation in females. That is, heterozygous females (i.e., 49% of the female population) will, therefore, exhibit retinal patches with either serine or alanine at residue 180 of the L-cone visual pigment.

To pursue this line of reasoning, the majority of males (56.3%) will have L(ser<sup>180</sup>) which results in a greater spectral separation with respect to the peak sensitivity of the M-cone pigment by approximately 2.7 nm. In contrast, the largest number of females will be heterozygous having L(ser<sup>180</sup>) and L(ala<sup>180</sup>) on each of their X-chromosomes resulting in a peak sensitivity that lies between the L(ser<sup>180</sup>) and L(ala<sup>180</sup>) peak sensitivities. Thus, there will be more women (81%) – that is, including both those having L(ser<sup>180</sup>) / L(ser<sup>180</sup>) and those having L(ser<sup>180</sup>) / L(ala<sup>180</sup>) expression – with an average greater spectral separation than men.

Among colour normal male observers, it has been demonstrated that the two L-cone photopigment subgroups can be distinguished in their Rayleigh (red-green equation) colour matches (Neitz & Jacobs, 1986; Winderickx et al., 1992); independently of variations in lens

density and in the optical density of the photopigments themselves (Winderickx et al., 1992). However, although the Rayleigh match midpoints of the two subgroups may differ – it is assumed that female heterozygotes for the alanine/serine polymorphism should have intermediate match midpoints (see also Neitz & Jacobs, 1986) – it does not necessarily follow that their red-green colour difference discrimination thresholds do. Pointedly, we did not observe any bimodality in the Rayleigh match range of our males; nor in the range of their RG CAD thresholds (see Fig. 2). The lack of such findings tends to undermine any arguments directly associating red-green colour discrimination with spectral separation between the L- and M-cone photopigments. Indeed, other recent studies (see Barbur et al. in this issue) show that ~10 nm separation (in deutan colour deficient subjects) suffices to produce RG discrimination thresholds that are only twice as large as the standard normal threshold (see also, Neitz et al., 1996). Thus, the ~2.7 nm separation between the two polymorphic variants of the L-cone photopigment may be an unlikely cause for producing measurable differences in red-green colour discrimination amongst normal trichromats. If anything, our results suggest the opposite effect: males appear to have improved red-green chromatic discrimination, even though on average they have a smaller spectral separation between the L- and M- cone pigments than women.

How could this unexpected result come about? It might be that it is not differences in spectral separation alone that account for any observed differences between the male and female groups. Rather, it could be owing to increased photoreceptor noise (see, for instance, Vorobyev, 2004) in the colour opponent channels. The 49% of females, excluding those who are heterozygotic carriers, having both variants of the L-cone photopigment would in fact have four rather than three pigments. They could benefit from this heterozygosity if separate retinal and cortical mechanisms were available for comparing signals of the additional X-linked photopigment with its different spectral sensitivity. On the other hand, if separate opponent channels are unavailable for analysing the signals of the fourth pigment in the heterozygotes this could lead to increased neural noise in the conventional opponent channels with potentially detrimental effects on colour discrimination.

## **Conclusion**

Chromatic sensitivity was measured in a large population of normal trichromat males and females, using the CAD colour vision test and the Nagel anomaloscope. On average, females were revealed to have significantly higher RG chromatic thresholds compared with men on the CAD test and larger red-green matching ranges on the Nagel anomaloscope. A correction was applied to exclude the possible effect of female heterozygotes that were undetected by our screening procedures upon our gender comparisons. On the basis of our results, we can

confidently conclude that women do not have superior red-green colour discrimination than men. However, the possibility arises that they may on average have poorer discrimination. Genetic and physiological explanations related to a sexual dimorphism in X-linked cone photopigment expression are offered as to why this may be the case. However, firm conclusions between perception and genetics will only be possible when comprehensive molecular genetic data is obtained from the same subjects who are participating in the sensitivity measurements.

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**Fig. 1.** Data indicating the statistical limits that define the standard normal (SN) CAD test observer (Rodriguez-Carmona et al., 2005). The results are plotted in the CIE  $-(x,y)$  1931 chromaticity chart. The black cross at the centre of the diagram shows the chromaticity of the white background i.e., 0.305, 0.323. The dotted black ellipse represents the median values computed from the distribution of red-green (RG) and yellow-blue (YB) thresholds in 240 normal trichromats. The grey shaded area shows the 97.5 and 2.5 % limits of variability within these observers. Thresholds that fall within the grey region are taken to reflect 'normal' chromatic discrimination sensitivity. The red, green and blue lines denote 'colour confusion bands' based on data measured in protanopes, deuteranopes and tritanopes, respectively. The large coloured dots show data measured for a typical normal trichromat. The insert shows the location of the RG and YB colour axes in CIE chromaticity space.

**Fig. 2.** (A) Distribution of midpoint matches for the Nagel anomaloscope red-green matching range for 225 subjects; 114 females (diamonds) and 111 males (circles). The mean midpoint match for females was  $40.04 \pm 1.50$  and for males  $39.95 \pm 2.02$ , both distributions have the same median value of 40.0. The difference between females and males is not significant ( $p=0.709$ ;  $df = 203$ , two-sample  $T$ -test). (B) Distribution of matching ranges for the Nagel anomaloscope match for the same subjects; females (squares) and males (diamonds). The mean range is  $4.11 \pm 1.28$  and  $3.75 \pm 1.38$ , for females and males, respectively. Both distributions have the same median value for the range of 4.0 scale units. The difference between females and males is significant ( $p=0.040$ ;  $df = 220$ , two-sample  $T$ -test).

**Fig. 3.** (A) Distribution of YB (yellow-blue) and (B) RG (red-green) CAD thresholds in units of the standard normal (SN) CAD observer (see Fig. 1) for all subjects tested. The separate results for males and females are superimposed. The distributions of YB and RG thresholds are not normally distributed (revealed by normality tests; Anderson-Darling, Ryan-Joiner, Kolmogorov-Smirnov). Comparisons between females and males for YB chromatic thresholds are not significant ( $p=0.4115$ ; Mann-Whitney Test), whilst differences between females and males for RG chromatic thresholds are significant ( $p=0.0004$ ; Mann-Whitney Test). Females tend to have higher RG thresholds.

**Fig. 4.** Summarised RG CAD thresholds obtained for several male/female subgroups. Median, lower and upper quartiles, and outliers are shown for each group. Males differ significantly from females ( $p=0.0004$ ; Mann-Whitney Test), from females when all estimated deutan carriers (11%) are excluded ( $p=0.0178$ ; Mann-Whitney Test) and from females when all estimated protan and deutan carriers (15%) are excluded ( $p=0.0484$ ; Mann-Whitney Test).

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Figure 1

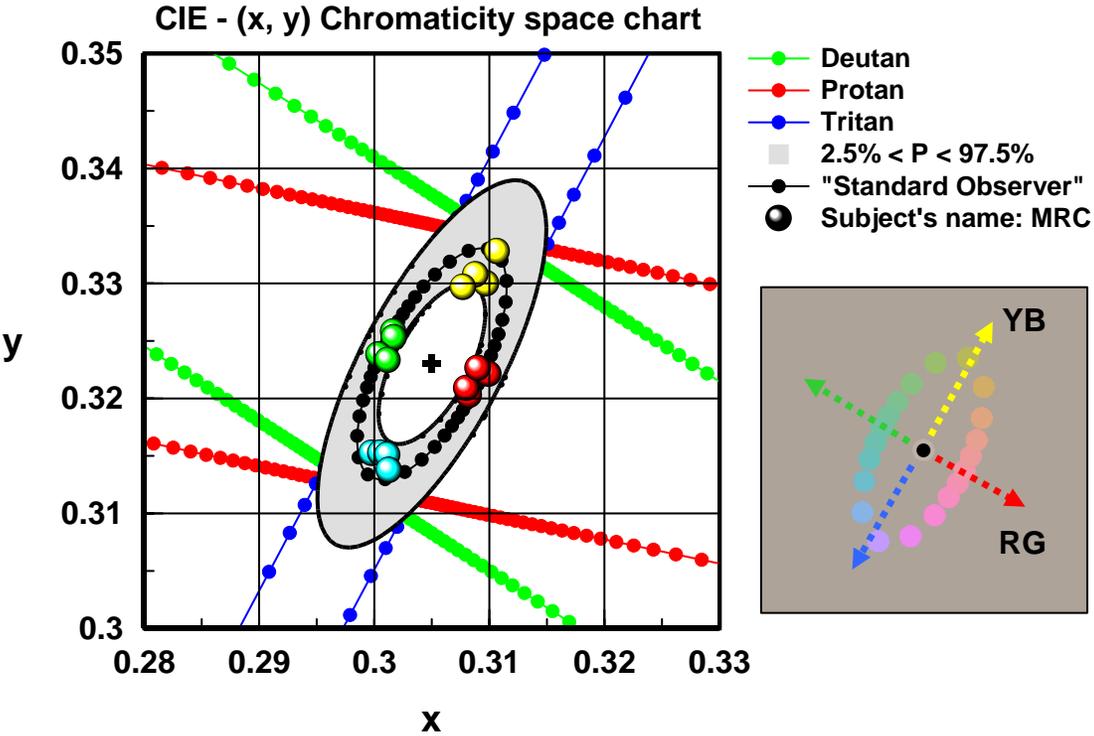


Figure 2

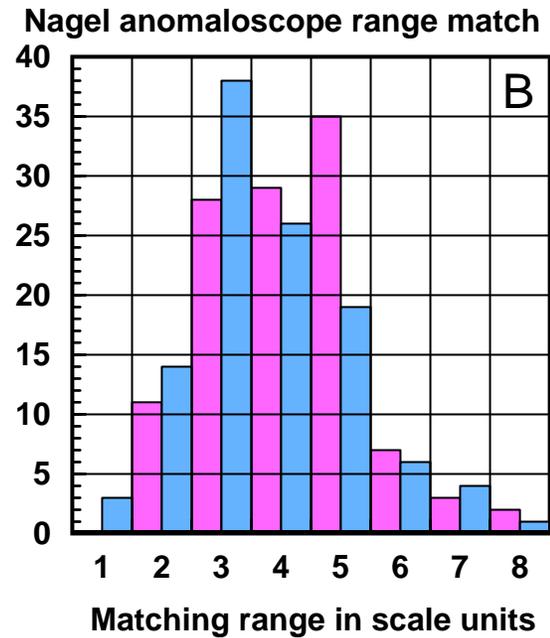
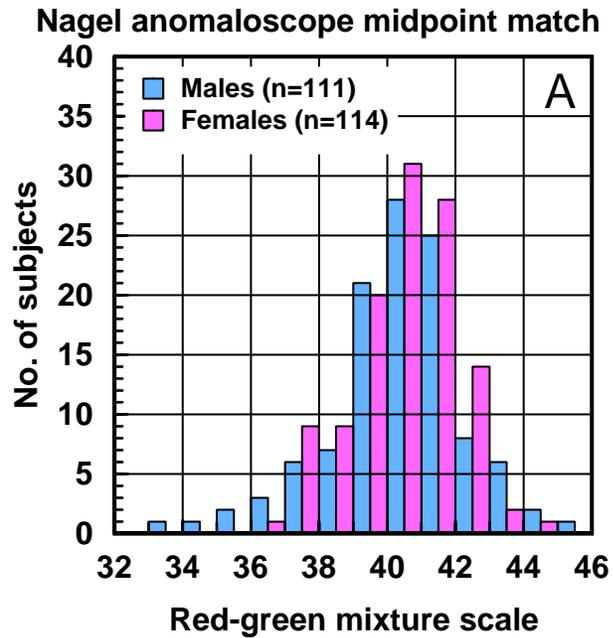


Figure 3

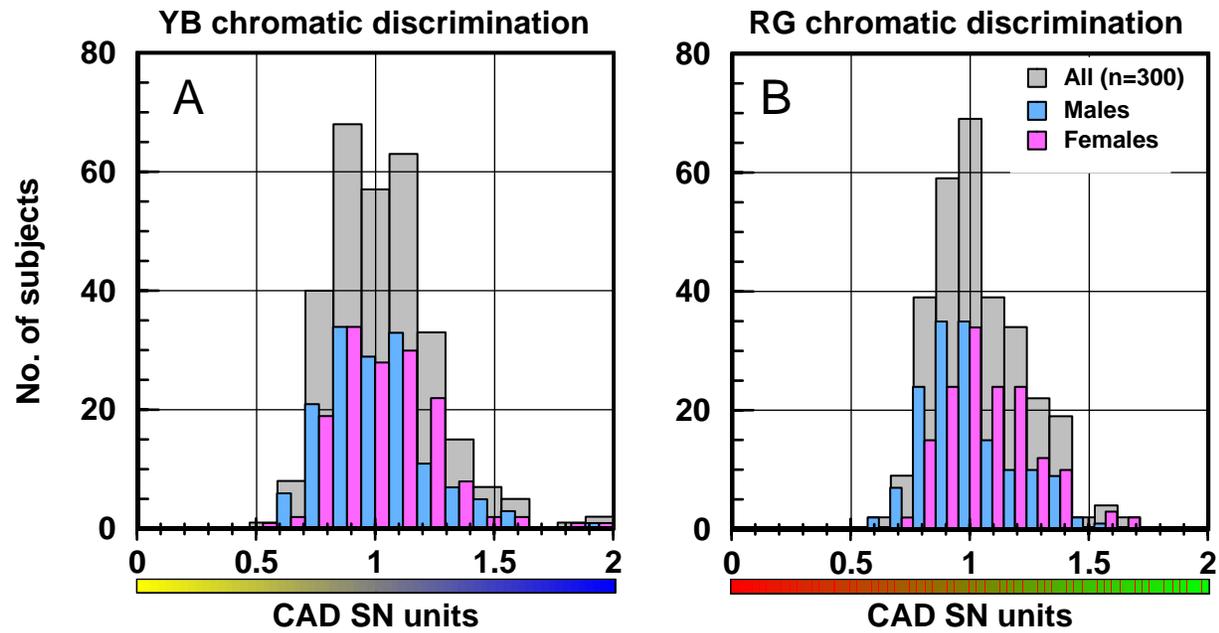


Figure 4

