

## Diagnosing protan heterozygosity using the Medmont C-100 colour vision test

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**Background:** A surprisingly high 15 per cent of women in Caucasian societies are carriers of the genes for abnormal colour vision but there is no clinical method to identify them. It has long been known that heterozygotes for the protan colour vision deficiencies can demonstrate a reduced luminous sensitivity to red light. This is known as Schmidt's sign, which is thought to arise from mosaicism (Lyonisation). The Medmont C-100 colour vision test measures relative spectral sensitivity using flicker photometry to differentiate protans and deutans. It should be able to diagnose Schmidt's sign.

**Method:** We tested six known protan heterozygotes (four whose sons have a protan colour vision deficiency and two whose fathers are protan) with the Medmont C-100 test.

**Results:** All six heterozygotes made average settings of  $-1.75$  or more negative at the Medmont C-100 test, settings which are at or beyond the boundary of the distribution of settings made by observers with normal colour vision. There have been two previous cases reported in the literature of protan heterozygotes, who made protan settings on the Medmont C-100 or its predecessor test, the OSCAR. We also tested six daughters of the known heterozygotes, 50 per cent of whom are likely to be heterozygotes. Four of the six (66 per cent) made protan settings on the Medmont C-100. The other two made normal 0.0 settings.

**Conclusion:** We conclude that the Medmont C-100 can be used clinically to diagnose carriers of protan colour vision deficiency.

Key words: abnormal colour vision, Medmont C-100 test, protan heterozygosity, Schmidt's sign

The mode of inheritance of the most common forms of abnormal colour vision is sex-linked recessive, which makes the inheritance of abnormal colour vision a chancy business. There is a 50 per cent chance that female carriers of a gene for abnormal colour vision will pass on their abnormal gene to their sons (Figure 1a). There is a much greater chance of women being carriers of abnormal colour vision: 100 per cent of the daughters of a father with abnormal colour vision will be carriers,

(Figure 1b) and there is a 50 per cent chance that the daughters of carriers will themselves be carriers (Figure 1a).

Carriers of abnormal genes for colour vision usually have normal colour vision because the normal colour vision gene on one of their X chromosomes is dominant over an abnormal gene inherited from their father or mother on the other X chromosome. They have abnormal colour vision only when, by chance, they inherit two abnormal colour vision genes on the

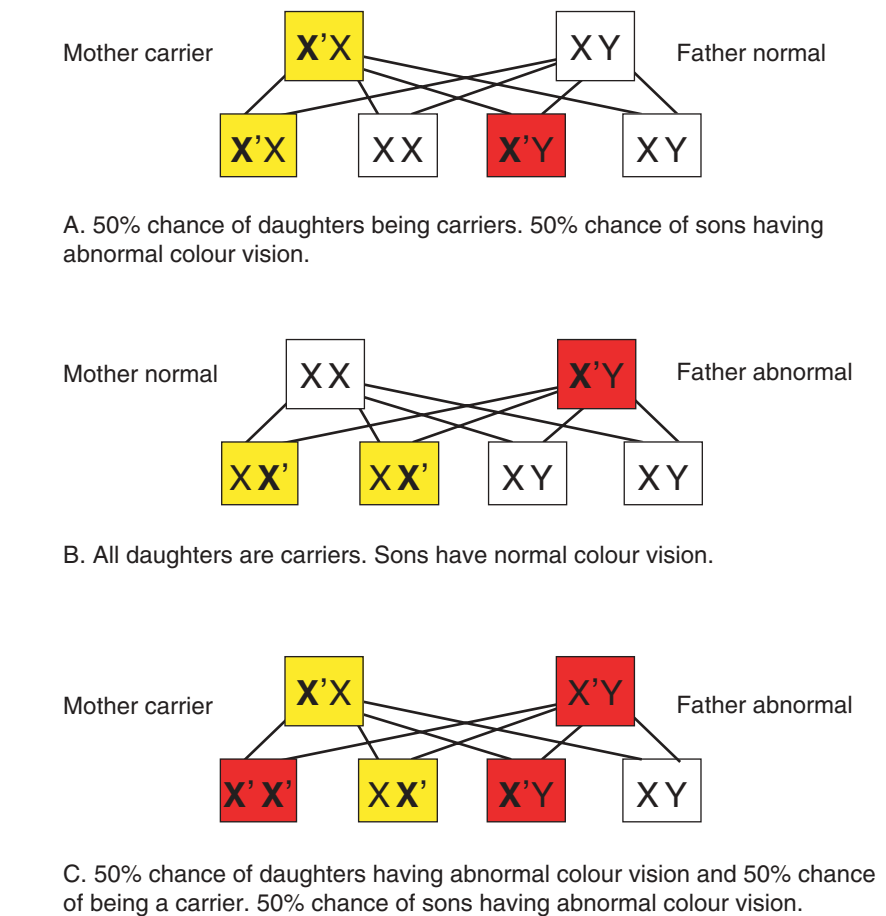
one allele,<sup>a</sup> one from their mother on one X-chromosome and one from their father on the other X-chromosome (Figure 1c).

a. Alleles are different forms of the same gene. The gene for the L photopigment can have different forms (alleles) that either express the normal L photopigment or an anomalous or no L photopigment, which leads to protan colour vision deficiencies. Likewise, the gene for the M photopigment can have different forms leading to either the normal M photopigment or an anomalous or no M photopigment that leads to the deutan colour vision deficiencies.

A surprisingly high proportion of women are carriers of genes for red-green defective colour vision. The probability of a female having a defective gene on one X chromosome and the normal gene on the other X chromosome is twice the product of the odds of each event separately, or  $2a(1-a)$  where  $a$  is the prevalence of abnormal colour vision among males. Platt<sup>1</sup> calculates the prevalence of females who are heterozygous for defective colour vision as 14.72 per cent based on an overall eight per cent prevalence of abnormal colour vision among males<sup>b</sup> but his calculation is incorrect because the protan and deutan colour vision deficiencies are inherited separately. The prevalence of protan carriers is  $2 \times 0.02(1 - 0.02) = 3.92$  per cent if the prevalences of protanopia and protanomaly are each one per cent. The prevalence of deutan carriers is  $2 \times 0.06(1 - 0.06) = 11.28$  per cent, if the prevalence of deuteranomaly is five per cent and deuteranopia one per cent. Therefore, the prevalence of heterozygosity for protan and deutan colour vision deficiency is 15.20 per cent. In Table 1, we approach the calculation in a different way, which shows the chance of carrier females from the various unions of normal and colour vision defective males with normal, carrier and colour vision defective females. This approach shows the proportions of the different genotype combinations for carriers of the red-green colour vision deficiencies and how they come about.

A small number of female carriers will carry the abnormal genes for both protan and deutan colour vision deficiency, having inherited one abnormal gene on the deutan locus from their mother and one on the protan locus from their father or vice versa. They still have normal colour vision because the defective gene on one of their X-chromosomes is 'covered' by the normal gene on the other. The prevalence of these mixed heterozygotes can be calculated in a manner similar to that in

b. Platt<sup>1</sup> actually adds a further 0.24 per cent being mixed heterozygote carriers who have two abnormal genes for different kinds of abnormal colour vision to bring his calculation of the prevalence of carriers to 14.96 per cent.



**Figure 1. Inheritance of the sex linked red-green colour vision deficiencies. The genes for the L and M cone photopigments are carried on the X chromosome. X' designates the X chromosome carrying the abnormal gene for colour vision. The yellow boxes represent carriers and the red boxes those with abnormal colour vision.**

Table 1 and is 0.24 per cent. This means that the actual proportion of women who are carriers is 14.96 per cent, slightly less than the 15.2 per cent prevalence of abnormal protan and deutan genes among colour vision normal women ( $15.20 - 0.24 = 14.96$  per cent). Because of genotype variations<sup>2</sup> and regional and ethnic differences in the prevalence of abnormal colour vision,<sup>3</sup> this theoretical calculation should not be taken too literally. For example the prevalence of abnormal colour vision among Australian males is probably 6.3 per cent,<sup>4</sup> not the usually accepted eight per cent and if this is so, only 11.8 per cent of Australian women will be carriers.

Nevertheless it is startling to realise that as many as one in seven women are carriers of abnormal colour vision.

As the genes for abnormal colour vision are so common among women, there may be occasions when it would be helpful to be able to diagnose them, so that advice can be given on the likelihood of their sons having abnormal colour vision. However, there is no accepted clinical test to enable that diagnosis.

It has been reported that the colour vision of heterozygotic carriers of the abnormal gene is not entirely normal. Some known heterozygotes make a 'red + green = yellow' match with the Nagel anomaloscope that is at the extremes of

**Table 1.** The iterative calculation begins by inserting an estimate of the prevalences of deutan and protan carriers at (A) and the spreadsheet calculates the chance of a carrier offspring for each type of union by multiplying the prevalence of each type of female genotype by that for each type of male genotype. The sum of these chances is the prevalence of carriers at (B). The estimate A is adjusted until it and the calculated prevalence B are equal. Protan and deutan prevalences are treated separately because the genes for the L (red) and the M (green) photopigments are on separate allelic loci. X<sub>nd</sub> and X<sub>np</sub> mean normal genes on the deutan and protan loci, respectively, X<sub>pa</sub> and X<sub>da</sub> the genes for protananomaly and deuteranomaly, respectively, and X<sub>p</sub> and X<sub>d</sub> for protanopia and deuteranopia. The calculation is based on the assumption that the prevalence of abnormal colour vision is eight per cent among males, two per cent being protan and six per cent deutan. The prevalences of DCV among females have been calculated from the known prevalences of DCV among males and the calculated prevalences of the corresponding carriers.

<b>CARRIERS OF DEUTAN COLOUR VISION DEFICIENCY</b>						
		Prevalence in females	X <sub>nd</sub> Y	X <sub>da</sub> Y	X <sub>d</sub> Y	Calculated carrier prevalence
Prevalence in males			94.00%	5.00%	1.00%	
<b>(A) Total Deutan carriers (estimated)</b>		<b>11.27%</b>				
<b>CV normal females (deutan locus)</b> X <sub>n</sub> X <sub>n</sub> 88.37%						
Frequency of union			83.07%	4.42%	0.88%	
Chance of female carrier from this union			0.00%	100.00%	100.00%	
Percentage of carriers from this type of union			0.00%	4.42%	0.88%	<b>5.30%</b>
<b>Female carriers</b> X <sub>da</sub> X <sub>n</sub> <b>9.39%</b>						
Frequency of union			8.83%	0.47%	0.09%	
Chance of female carrier from this union			50.00%	50.00%	50.00%	
Percentage of carriers from this type of union			4.41%	0.23%	0.05%	<b>4.70%</b>
X <sub>d</sub> X <sub>n</sub> <b>1.88%</b>						
Frequency of union			1.77%	0.09%	0.02%	
Chance of female carrier from this union			50.00%	50.00%	50.00%	
Percentage of carriers from this type of union			0.88%	0.05%	0.01%	<b>0.94%</b>
<b>CVD females</b>						
<b>Homozygotic deuteranomaly</b> X <sub>da</sub> X <sub>da</sub> <b>0.25%</b>						
Frequency of union			0.23%	0.01%	0.00%	
Chance of female carrier from this union			100.00%	0.00%	0.00%	
Percentage of carriers from this type of union			0.23%	0.00%	0.00%	<b>0.23%</b>
<b>Homozygotic deuteranopia</b> X <sub>d</sub> X <sub>d</sub> <b>0.01%</b>						
Frequency of union			0.01%	0.00%	0.00%	
Chance of female carrier from this union			100.00%	0.00%	0.00%	
Percentage of carriers from this type of union			0.01%	0.00%	0.00%	<b>0.01%</b>
<b>Mixed heterozygotic deuteranomaly</b> X <sub>da</sub> X <sub>d</sub> <b>0.10%</b>						
Frequency of union			0.09%	0.01%	0.00%	
Chance of female carrier from this union			100.00%	0.00%	0.00%	
Percentage of carriers from this type of union			0.09%	0.00%	0.00%	<b>0.09%</b>
<b>Total female deutan CVD</b>		<b>0.36%</b>	<b>Total deutan carriers calculated B</b>			<b>11.27%</b>
Total females		100.00%				

Continued next page

<b>CARRIERS OF PROTAN COLOUR VISION DEFICIENCY</b>						
	Prevalence in males	Prevalence in females	X <sub>n</sub> Y	X <sub>pa</sub> Y	X <sub>p</sub> Y	Calculated carrier prevalence
<b>(A) Total protan carriers (estimated)</b>		<b>3.92%</b>	98.00%	1.00%	1.00%	
<b>CV normal females (protan locus)</b>	X <sub>n</sub> X <sub>n</sub>	96.04%				
Frequency of union			94.12%	0.96%	0.96%	
Chance of female carrier from this union			0.00%	100.00%	100.00%	
Percentage of carriers from this type of union			0.00%	0.96%	0.96%	<b>1.92%</b>
<b>Female carriers</b>	X <sub>pa</sub> X <sub>n</sub>	<b>1.96%</b>				
Frequency of union			1.92%	0.02%	0.02%	
Chance of female carrier from this union			50.00%	50.00%	50.00%	
Percentage of carriers from this type of union			0.96%	0.01%	0.01%	<b>0.98%</b>
	X <sub>p</sub> X <sub>n</sub>	<b>1.96%</b>				
Frequency of union			1.92%	0.02%	0.02%	
Chance of female carrier from this union			50.00%	50.00%	50.00%	
Percentage of carriers from this type of union			0.96%	0.01%	0.01%	<b>0.98%</b>
<b>CVD females</b>						
<b>Homozygotic protanomaly</b>	X <sub>pa</sub> X <sub>pa</sub>	<b>0.01%</b>				
Frequency of union			0.01%	0.00%	0.00%	
Chance of female carrier from this union			100.00%	0.00%	0.00%	
Percentage of carriers from this type of union			0.01%	0.00%	0.00%	<b>0.01%</b>
<b>Homozygotic protanopia</b>	X <sub>p</sub> X <sub>p</sub>	<b>0.01%</b>				
Frequency of union			0.01%	0.00%	0.00%	
Chance of female carrier from this union			100.00%	0.00%	0.00%	
Percentage of carriers from this type of union			0.01%	0.00%	0.00%	<b>0.01%</b>
<b>Mixed heterozygotic protanomaly</b>	X <sub>pa</sub> X <sub>p</sub>	<b>0.02%</b>				
Frequency of union			0.02%	0.00%	0.00%	
Chance of female carrier from this union			100.00%	0.00%	0.00%	
Percentage of carriers from this type of union			0.02%	0.00%	0.00%	<b>0.02%</b>
<b>Total female protan CVD</b>		<b>0.04%</b>	<b>Total protan carriers calculated B</b>			<b>3.92%</b>
Total females		100.00%				
<b>TOTAL PROTAN + DEUTAN CARRIERS</b>						<b>15.19%</b>



**Figure 2.** The Medmont C-100 test. Left. The subject views a yellowish flickering light generated by alternating red and green LEDs and adjusts the control knob until the flicker disappears or is a minimum. Right. The settings chosen to achieve no or minimum flicker are read on an arbitrary scale from -5 to +5 where +2.0 to -2.0 is the extreme range of normal settings but typically settings are within  $\pm 1.0$ . The scale is colour-coded red for protan settings, green for deutan and yellow for normal. The colour-coded scale lights correspond to integers (1, 2, 3 ...) but can be interpolated to 0.5, when two adjoining lights are illuminated.

the normal distribution of matches<sup>5,6</sup> or have a slightly larger than normal matching range, suggesting they have slightly reduced colour discrimination.<sup>7</sup> Some may have a higher than normal error score with the Farnsworth Munsell 100 hue test, which also suggests some loss of colour discrimination<sup>5</sup> and there is one report of reduced wavelength discrimination in carriers.<sup>8</sup> However, Jordan and Mollon<sup>7</sup> did not find higher Farnsworth Munsell 100 Hue scores and did not find displaced anomaloscope matches among their 34 carriers. There have been reports that some carriers make more errors than usual with pseudoisochromatic plates<sup>5,9</sup> even though the number of errors they make is within the clinical limits of normality and they pass the test.

This partial expression of characteristics of abnormal colour vision by carriers of an abnormal gene for colour vision is usually explained by random X-chromosome inactivation early in embryonic development of one or other of the X-chromosomes in a given cell.<sup>10</sup> For women who are homozygous and carry the normal

genes on both X-chromosomes, this is of no consequence as the X-chromosome that is not inactivated carries the normal genes for the L and M cone photopigments. Carriers of abnormal colour vision are heterozygotic: the genes on their two X-chromosomes are not the same: one X-chromosome either lacks the gene for the L or M photopigment or carries a gene for an anomalous L or M photopigment. For heterozygotic women, inactivation of the X-chromosome carrying the normal genes means that the normal photopigment will not develop in those cells. The result is that the retina is a mosaic of normal and abnormal cells, a phenomenon sometimes called mosaicism.

None of the methods that detect slightly reduced colour discrimination or disturbed colour matching in carriers of abnormal colour vision genes offers much promise as a useful clinical test for colour vision heterozygosity because of overlap of the results with persons having normal colour vision.

However, it has long been known that carriers of the protan gene have reduced lumi-

nous sensitivity to long wavelength light. This was reported by Schmidt<sup>11</sup> some 70 years ago. She measured relative spectral luminous sensitivity by the flicker method for nine deutan carriers (two deuteranomals, four extreme deuteranomals and three deuteranopes), all of whom were found to have normal spectral luminous sensitivity. She also tested nine carriers for protan colour vision deficiency (one protanomal, four extreme protanomals and four protanopes) and found the peak of the luminous sensitivity curve was displaced from the normal 580 to 590 nm to 570 to 575 nm, with no relationship between the extent of the displacement and the severity of the colour vision abnormality carried.

The reduced red-light luminous sensitivity of protan carriers is called Schmidt's sign and has been confirmed by a number of other investigators<sup>12-16</sup> and there is supporting evidence from ERG studies.<sup>17,18</sup> This offers some promise for clinical diagnosis, although no practitioner would wish to measure the  $V_{\lambda}$  function or order an ERG to diagnose heterozygosity for a protan colour vision deficiency.

Many optometrists have a Medmont C-100 test,<sup>c</sup> which they use to differentiate protan and deutan colour vision deficiencies among those who fail the Ishihara test. The Medmont C-100 (Figure 2) measures relative spectral sensitivity for red and green light by flicker photometry. It presents red and green light emitted by two alternating LEDs and the patient adjusts their relative intensities to achieve cessation of flicker or minimum flicker.

The principle of the test was developed by Estvez and his colleagues<sup>19</sup> and it was marketed originally as the OSCAR test (OSCAR stands for 'objective screening of colour anomalies and reductions') by Medilog. The OSCAR is no longer made but its currently available successor is the Medmont C-100.

The OSCAR reliably differentiates protans from deutans among those with defective colour vision, but it does not always differentiate normal colour vision from abnormal colour vision because the distribution of settings by colour normal observers overlaps the protan and deutan distributions.<sup>20,21</sup> The Medmont C-100 also validly categorises protan and deutan colour vision deficiencies<sup>22,23</sup> and gives repeatable results.<sup>24</sup> Because it identifies protans so well, it deserves a place in every optometric practice to enable better advice to be given to patients with abnormal colour vision. The C-100 is inexpensive and the test takes only two or three minutes to administer, making it a practical test for clinical use.

There is some evidence that the OSCAR and the Medmont C-100 test can detect Schmidt's sign in protan heterozygotes. Verriest and Uvijls<sup>20</sup> report one known protan carrier with a clear protan setting of -3.0 on the OSCAR, although they report a second protan carrier with a normal setting of 0.0. Metha and Vingrys<sup>23</sup> report one known protan carrier who makes an average setting with the Medmont C-100 of -2.0 that is outside the range of settings

made by their subjects with normal colour vision (their normal observers had average settings calculated over five to seven readings that fell between +1.8 and -1.8) and in the domain of settings made by protans. The daughter of this carrier also made similar protan settings on the Medmont C-100 and could be presumed to be a carrier like her mother.

In this paper, we report the results of the Medmont C-100 for four protan carriers whose sons have a diagnosed protan colour vision deficiency and two protan carriers whose fathers are protans.

## METHODS

The colour vision of six known protan heterozygotes was tested using the Ishihara test and the Medmont C-100 test. Three were known to be protan heterozygotes because their sons had protanopia (two cases) or protanomaly (one case). These are Carriers 1, 2 and 3 in Table 2. Two further known heterozygotes were identified because they were daughters of one of the protanopes and the protanomalous. The sixth known protan heterozygote was a daughter of Carrier 1 and has a young son who failed the Ishihara test and made protan settings with the Medmont C-100.

Three of the protan heterozygotes were recruited from a concurrent research project, which involved the study of the colour vision of a family, and the other three were from the families of two colleagues.

The colour vision deficiency of the three colour vision deficient sons was diagnosed using the Ishihara test, the Medmont C-100, the Farnsworth D15 test and the Nagel anomaloscope. The fourth son was too young to be tested with the anomaloscope or the Farnsworth D15.

The Medmont C-100 test was given at least three times to those known to be protan heterozygotes. It was given binocularly, under indoor domestic lighting, at arm's length distance of about 40 cm

The Ishihara test and the Medmont C-100 test were also given to the daughters of the carrier mothers and the daughters of the protan sons who were available for testing.

## RESULTS

The results are summarised in Table 2. All four mothers known to be protan heterozygotes because their sons were protans made average settings on the Medmont C-100 of -1.7 or more negative. Metha and Vingrys<sup>23</sup> found that the most minus average setting with the Medmont C-100 made by a sample of 23 colour vision normal subjects was -1.8.

The two daughters of two protan sons, who are also obligatory protan heterozygotes, had average Medmont C-100 readings of -2.1 and -2.7.

Four of the six daughters (67 per cent) of two of the known protan carriers had average Medmont C-100 settings of -1.75 or more negative. It is expected that 50 per cent of daughters of carriers will be carriers.

One of the daughters (Daughter 4 in family 1) has a son aged four years. She gives a protan Medmont C-100 reading and accordingly her son could be a protan. Despite his young age and his inability to recognise all numbers, he did fail the Ishihara. In particular, he failed to see the protan numbers in the diagnostic plates and he failed to see or trace several other numbers (numbers that we established he could recognise and verbalise) on other plates. He also made confident protan settings on the Medmont C-100 and as his uncle is a protanope we think it is reasonable to diagnose him as a protanope. Therefore, his mother is an obligatory carrier of protanopia.

## DISCUSSION

This study reports six known protan heterozygotes, all of whom made protan settings with the Medmont C-100. Metha and Vingrys<sup>23</sup> and Verriest and Uvijls<sup>20</sup> have each reported one similar case, so the total number of reported protan heterozygotes giving protan settings on the Medmont C-100 (or OSCAR) is eight. The exception is one known protan heterozygote reported by Verriest and Uvijls, who made an OSCAR setting of zero.

It is of interest that the three very young subjects aged four, five and eight years all understood what was required with the

c. The Medmont C-100 colour vision tester is manufactured and distributed by Medmont Pty Ltd, Whitehorse Business Park, 170-180 Rooks Road, Vermont, Victoria 3133 Australia. URL <http://www.medmont.com/>

Subject	Age	Ishihara test	Medmont C-100 settings (Average)	D 15 test	Nagel anomaloscope	Diagnosis
<b>Carrier 1</b>	63	Pass	-1.0, -2.0, -2.0 (-1.7)			Obligatory protanope heterozygote
Son	36	Fail	-2.0, -4.0, -3.0 (-3.0)	Fail. Multiple diametric crossings	Range 0-72. Y decreased with increasing R	Protanope
Daughter of son	8	Pass	-2.0, -3.0, -3.0 (-2.7)			Obligatory protanope heterozygote
Daughter 1	39	Pass	-2.0, -3.0 (-2.5)			Presumed protanope heterozygote
Daughter 2	35	Pass	0.0, 0.0 (0.0)			
Daughter 3	33	Pass	0.0, 0.0, 0.0 (0.0)			
Daughter 4	31	Pass	-3.0, -3.0, -3.5 (-3.2)			Obligatory protanope heterozygote
Son of daughter 4	4	Fail	-3.0, -3.0 (-3.0)	Not done	Not done	Probable protanope
<b>Carrier 2</b>	62	Pass	-3.0, -2.0, -2.0 (-2.3)			Obligatory protanope heterozygote
Son	34	Fail	-3.5, -2.5, -2.5, -4.0, -4.0, -4.0 (-3.4)	Pass. One diametric crossing	MMP 62 Range ± 5	Protanomal
Daughter of son	5	Pass	-3.0, -1.5, -2.5, -1.5 (-2.1)			Obligatory protanope heterozygote
Daughter 1	32	Pass	-2.0, -1.0, -2.0, -2.0 (-1.75)			Presumed protanope heterozygote
Daughter 2	28	Pass	-2.0, -1.5, -1.5, -2.0 (-1.75)			Presumed protanope heterozygote
<b>Carrier 3</b>	~ 52	Pass	-2.0, -2.0, -2.0 (-2.0)			Obligatory protanope heterozygote
Son	28	Fail	-5.0, -5.0, -5.0 (-5.0)	Fail. Multiple diametric crossings	Range 0-72. Y decreased with increasing R	Protanope

NOTE: Presumed protan heterozygosity is based solely on the minus Medmont C-100 readings. The obligatory heterozygotes are diagnosed by having a protan son or a protan father.

**Table 2. Results of colour vision tests**

Medmont C-100 and made repeatable settings. It seems the test can be used on young children.

It is possible that the Medmont C-100 may also diagnose deutan heterozygotes. Metha and Vingrys<sup>23</sup> and Verriest and Uvijls<sup>20</sup> report six cases of deutan heterozygotes who make settings in excess of +1.5, which is at the extreme of the distribution of settings made by colour vision normal observers. This is an unexpected result because Schmidt<sup>11</sup> did not find her

deutan heterozygotes to have abnormal spectral luminosity.

We conclude that the Medmont C-100 can be used to diagnose protan carriers. An average reading of -1.7 or more for a female who passes the Ishihara test can be taken as indicative of protan heterozygosity, as the least negative reading among our six protan heterozygotes was -1.7, and the limits of the average setting with the Medmont C-100 made by those with normal colour vision is ± 1.8.<sup>23</sup>

The test could be used for this purpose for women who think their father or a grandfather may have abnormal colour vision and wish to know if they are carriers. It can also be used to identify and exclude carriers, when recruiting subjects for experiments in normal colour vision or cone function.

All patients attending an optometric practice for the first time should have their colour vision screened with the Ishihara test, so there is a record of the normality

or otherwise of their colour vision. This should include female patients as four in 1,000 women have abnormal colour vision. The Medmont C-100 given to women who pass the Ishihara test will establish, by an average reading of -1.8 or more negative, which of them are carriers of a protan gene and might even identify deutan carriers with a reading of +2.0 or more.

The second author has regularly suggested to those of his male students who had defective colour vision that they should test the colour vision of any prospective spouse to avoid the certainty of all their offspring having abnormal colour vision if the spouse should have abnormal colour vision of like kind. To his knowledge none has taken his advice. As the prevalence of abnormal colour vision among females is so low (0.4 per cent) the risk would have seemed very small for young risk-taking males or perhaps, it is other attributes that determine romantic attachment. The risk of union by a male with abnormal colour vision with a carrier of an allelic abnormal gene is much greater, as 15 per cent of women are carriers, with the prospect of 50 per cent of their sons and daughters having abnormal colour vision. The Medmont C-100 could be used to identify such carriers.

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#### FINANCIAL DISCLOSURE

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