

Colour Vision Revisited

Delhi J Ophthalmol 2014; 24 (4): 223-228

DOI: <http://dx.doi.org/10.7869/djo.48>

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Congenital red-green colour deficiency is an X-linked inherited disorder more frequently encountered in males compared to females. Screening of colour deficiency is crucial in various occupations, at times to safeguard colour deficient individual as well as vulnerable population. Acquired colour deficiency is seen in multiple inherited retinal photopigment disorders, vascular retinopathies and optic nerve disorders. Multiple tests have been devised to detect, classify and grade colour deficiency, each having a specific purpose. Tinted spectacles and contact lenses have been tried to rehabilitate colour deficient individuals with limited success.

Keywords : congenital colour deficiency • acquired colour vision deficiency • colour vision tests

Colour vision testing often forms integral part of ophthalmology examinations for job requirements and at times its anomalies are crucial to the diagnosis of various retinal and optic nerve diseases in ophthalmology. Hence, it is imperative to have a sound knowledge about the physiology of colour vision and its anomalies.

Theories of Colour Vision

Trichromatic theory

Young-Helmholtz (1802) proposed colour vision depends on the three different sets of retinal fibres responsible for perception of red, green, and violet.¹

Opponent theory

Hering (1878) proposed that yellow-blue and red-green represent opponent signals producing four colour primaries red, green, yellow, and blue and not just three.

Zone theory

Donder (1881) proposed that the Trichromatic theory operates at the receptor level and the Opponent theory applies to the subsequent neural level of colour vision processing. This is the basis of modern colour vision theory. At the receptor level each photopigment absorbs particular wavelengths of light in the short (blue, 440-nm), middle (green, 545-nm), or long (red, 560-nm) wavelength region of the visible spectrum. If one or more of their pigments is missing, colour deficiency results.^{2,3}

Congenital Colour Vision Anomalies

Genetics

L, M and S photopigments on cones are responsible for determination of colour pattern. The genes encoding the L and M photopigments are arranged in head-to-tail arrays on the X-chromosome, beginning with the L and followed by one or more M pigment genes. S pigment gene located on chromosome 7. Differences at a few amino acid positions that influence the spectra of the L and M cone pigments account for most of the variation in colour vision (Protanomaly, protanopia, deuteranomaly, deuteranopia) Blue cone monochromacy (rare) results from mutations that abolish function of both the L and M pigment genes. Tritanopia (rare) autosomal dominant colour vision defect caused by mutations in the S pigment gene. Total colour blindness (achromatopsia or rod monochromacy) is another rare autosomal recessive trait caused by mutations in genes encoding the proteins of the photoreceptor cation channel or cone transducin that are essential for function of all classes of cone.⁴ The types of colour deficiency is depicted in (Table 1).

As ophthalmologists, we are bestowed with the responsibility to screen professionals for colour deficit as well as grade them. If encountered with a colour deficient subject, they can be graded as given below.

Table 1: Types of colour vision defect

Type of Colour Deficiency	Red Cone	Blue Cone	Green Cone
Trichromatism (Normal sight)	Can differentiate all colours		
Anomalous Trichromatism	Can differentiate all colours but one colour has reduced or displaced sensitivity		
Protanomaly	Displaced sensitivity		
Deuteranomaly	Displaced sensitivity Most common colour vision defect		
Tritanomaly	Displaced sensitivity		
Dichromatism			
Tritanopia	Receptor normal	Receptor missing	Receptor normal
Deuteranopia	Receptor normal	Receptor normal	Receptor missing
Protanopia	Receptor missing	Receptor normal	Receptor normal
Monochromatism(Achromatopsia)	Totally unable to differentiate colours of equal brightness		

Grading Level of Colour Perception

Colour perception (CP) can be graded as follows:

CP-1: Correct answers to the series of colours with smallest aperture of Martin Lantern test at 6 meters.

CP-2: Must pass the Ishihara book with no errors.

CP-3: Should be able to recognize white, red and green colour signal correctly at a distance of 1.5 meters using largest aperture OR able to read requisite plates of Ishihara.

CP-4: When mistakes are made with white, red or green colours in the tests described under CP-3

Accepted level of colour perception is CP-III for Army and CP-1 for Indian Air force and Navy.

Congenital colour deficiency in medical profession

At multiple times ophthalmologists come across pre-occupational negative/positive screening of medical students based on severity of colour deficit. A number of descriptive terms like pallor, cyanosis, jaundice, altered blood, etc are used in medical profession. Colour vision forms an essential part of in blood and urine test strips, histological specimens, fundus examination, etc. It is advisable that medical students and doctors should be screened for the deficiency and advised about it and there should be more study of the effects of colour vision deficiency on decision-making in general practice and some specialties.⁵

Acquired Colour Vision Anomalies

Colour vision is a function of photoreceptors present on visual pigments. Any disease affecting the photoreceptors, optic nerve fibres can affect colour perception of an individual. Koellner's rule states that damage of the retina induces a tritan defect, and damage of the optic nerve induces a red-green-defect. Acquired colour vision defects unlike congenital colour vision defects can vary in type and severity during the course of disease. Each eye needs to be

checked separately as there can be monocular differences. Coexisting visual acuity and visual field anomalies often make the assessment of colour vision difficult. All the tests described below in (Table 2)² can be used for initial assessment of acquired colour vision defects, but FM 100 Hue test and anomaloscopes have an edge over other tests in classification and severity assessment.

Table 2: Acquired Colour Deficiency and their Clinical Association

Classification	Characteristics	Clinical association
Type 1 red-green	Similar to a protan defect Wavelengths of maximum luminous efficiency displaced to shorter wavelength	Progressive cone dystrophies (e.g. Stargardt's disease) Chloroquine toxicity
Type 2 red green	Similar to a deutan defect Reduction of relative luminous efficiency for short wavelengths	Optic neuropathy (e.g. retrobulbar neuritis associated with multiple sclerosis) Ethambutol toxicity
Type 3 blue (Most common)	Similar to a tritan defect (a) With reduction of relative luminous efficiency at both spectral limits (b) With displaced relative luminous efficiency to shorter wavelengths (pseudo-protanomaly)	Progressive rod dystrophies Retinal vascular lesions Peripheral retinal lesions (e.g. retinitis pigmentosa, diabetic retinopathy) Glaucoma Macular oedema (e.g. central serous retinopathy, diabetic maculopathy, age-related macular degeneration)

Optic neuritis is a clinical condition commonly encountered in clinical practice and the diagnosis of retrobulbar forms mainly depends upon examination of detailed visual function and electrophysiological investigative modalities. At the time of the acute attack of optic neuritis, the majority of selective color defects were blue/yellow defects, whereas at 6 months, more of the selective defects were red/green defects, though both types of defects (as well as nonselective defects) were seen acutely and at 6 months.⁶ The type of defect present at 6 months was not related to the severity of the initial visual loss.

Acquired colour vision defects are also an indicator of drug induced retinopathy and drug induced retrobulbar neuritis.⁷ In some cases, acquired colour vision disorders

may precede or reveal the onset of severe and sometimes irreversible eye damage. The drugs implicated mainly include: phosphodiesterase type 5 inhibitors such as sildenafil; digoxin; anti-infectives including interferon alfa; ethambutol; metronidazole; and some antimalarials.⁸ Patients on chronic therapy with above mentioned drugs must have periodic ophthalmic examinations including colour vision anomalies.

There have been multiple evidences as regards occupational exposure of certain organic solvents/ chemicals and acquired colour vision defects.⁹ Hence, it is advisable to screen subjects for the same for early detection of optic nerve and nervous system side effects.

Clinical evaluation and electrophysiological tests form an essential part in the evaluation of progressive cone and rod dystrophies. However, classification and grading of colour vision defect may help to reassure the diagnosis. Tan X et al. have reported color vision abnormality as an initial presentation of the complete type of congenital stationary night blindness.¹⁰ However, they may help as an allied test to judge the severity and progressive follow up of peripheral vascular lesions, retinitis pigmentosa and macular edema.

Colour Vision Tests

There is a large inventory of colour vision tests designed to perform various functions.

- **Screening tests:** Identifies subjects with normal and abnormal colour vision.
- **Grading tests:** Estimates severity of colour deficiency.
- **Classifying tests:** Diagnosing the type and severity of colour deficiency
- **Vocational tests:** Identifies colour matching ability, hue discrimination and colour recognition.

Some tests have more than one of the above listed functions (Table 3).

Function	Spectral Anomaloscope	Pseudoisochromatic plates	Hue discrimination	Lantern
Screening	excellent capability	excellent capability	-----	-----
Classifying/ Diagnosing	excellent capability	partially capable	partially capable	-----
Grading severity	excellent capability	few plates grade	partially capable	-----
Vocational suitability	-----	-----	excellent capability	Excellent capability

Pseudoisochromatic Plates

Pseudoisochromatic plates include the following:

- Ishihara Plates
- American Optical Hardy-Rand-Rittler Plates
- Standard Pseudoisochromatic plates
- City University test

Ishihara plates

This test is based on the principle of confusion of the pigment colour in red-green colour defectives. This is easy and rapid

test used most commonly for screening purposes. The 10th edition of Ishihara has 38 plates. Five different design formats used in 38 plates Ishihara (Table 4) (Figure1).^{2,11,12}

Sub-types	Plate no. (Numerals)	Plate no. (Pathways)	Intended design
1 Introductory	1	38	Seen correctly by all subjects. Identifies malingering.
2 Transformation	2-9	34-37	A number seen by a colour normal appear different to colour deficient subject.
3 Vanishing	10-17	30-33	A number is seen by a colour normal, but cannot be seen by a colour deficient subject.
4 Hidden digits	18-21	28-29	A number cannot be seen by normal, but is seen by a colour deficient subject.
5 Classification	22-26	26-27	Type of deficiency (red or green) is obtained from comparing the relative contrast of the paired numbers.



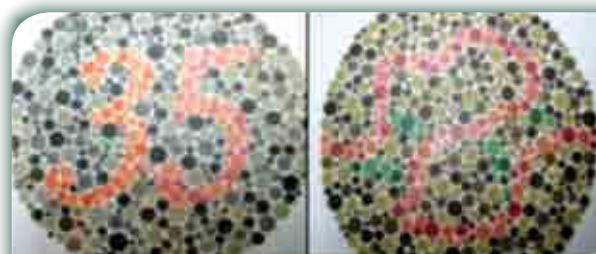
Introductory Plate

Transformation Plate



Vanishing Plate

Hidden Plate



Classification plate

Pathway Plate

Figure 1: Different types of Ishihara plates

The plates are designed to be appreciated correctly in a room which is lit adequately by daylight. The plates are held at a distance of 75 cm perpendicular to the line of sight. Out of initial 21 plates, if 17 or more plates are read correctly by an individual his colour sense should be regarded as normal. If 13 or less plates are correctly read then the

person has a red-green colour defect.¹³ Plates 22 to 25 are for differential diagnosis of Protans and Deutans. Disadvantage of this test is that it neither test for tritanope nor grade the degree of deficiency. American Optical Hardy-Rand-Ritter (HRR) is the test of choice for quantitative diagnosis and the Standard Pseudoisochromatic plates volume 2 for acquired colour deficiency.^{11,14}

Spectral Anomaloscope

The following include types of spectral anomaloscopes:

Nagel anomaloscope

Oculus HMC (Heidelberg Multi Colour) anomaloscope

Neitz anomaloscope

Pickford-Nicolson anomaloscope

Nagel Anomaloscope

The Nagel Anomaloscope (Figure 2) is based on Rayleigh equation, which states that a yellow hue is obtained by mixing red and green colour.² The instrument consists of a source of white light which is split into its spectral colours by a prism in a circular split field. In the lower half, spectral yellow appears. The colour in the upper half is adjusted to match the lower half. A normal subject can achieve a good colour match between two halves of field at 40-50 units of red-green mixture and 15 units of yellow. The test should be performed in semi dark environment. The anomalies quotient is a common method of presenting the midpoint of the red-green equation. It involves calculating an individual observer's match relative to the mean of normal observers. Anomalous quotients for normal trichromats fall between about 0.74 and 1.40. Deuteranomaly 1.7-20, Extreme Deuteranomaly 1.0-∞, Protanomaly 0.6-0.11, Extreme Protanomaly 1.0-0, and Protanopia – a very bright yellow



Figure 2: Heidelberg Anomaloscope

(around scale value 30) is matched to green end-point and a very dark yellow (around scale value 5) is matched to the red end-point. Deuteranopia – both end-points is matched to a brightness value around.¹⁵

Advantages

Excellent test for screening, classification and grading colour vision deficiencies.

Disadvantages

Anomaloscopes are expensive and comparatively difficult to administer.^{2,11}

Arrangement Tests

The following comprise arrangement tests:

- Farnsworth-Munsell 100 hue test
- Farnsworth-Munsell Dichotomous D-15 or Panel D-15 test
- Lanthony Desaturated D-15
- Adams Desaturated D-15

Farnsworth-Munsell 100 hue test

Farnsworth-Munsell 100 hue test is a very sensitive, reliable and effective method of determining colour vision defect. The test consists of 85 movable colour samples arranged in four boxes of 22 colours in the first box and 21 colours in rest. (Figure 3a) They are numbered on the back according to the correct colour order of the hue and scoring sheets are provided. (Figure 3b) The examiner prearranges the caps in random order and observer is instructed to arrange the caps as per hue variation taking first and last fixed caps as reference. Generally recommended time for arranging each panel is 2 minutes. Errors are made whenever caps are misplaced from the correct order. Error scores are calculated according to the distance between any two caps (Table 5). The score of each cap are plotted on a circular graph provided. By plotting the scores in a graph, it is seen that characteristic patterns are obtained in specific defects. (Figure 3c) The error score is the score with 2 subtracted. Sum of the error scores of the entire set of caps goes to make the total error score (TES).¹⁵



Figure 3 (a): FM 100 hue test consists of 85 movable colour caps arranged in four boxes



Figure 3 (b): Caps are on the back according to the correct colour



Figure 3 (c): Error score are plotted on scoring sheet

Colour Discrimination	Total error scores
Superior	0-20
Average	20-100
Low	>100

Thus it is considered capable of detecting severity and type of colour deficiencies.

Lantern Test

The following include types of lantern tests.

- Edridge-Green Lantern
- Farnsworth Lantern
- Holmes-Wright Lantern
- Martin Lantern

Edridge-Green Lantern

The Edridge-Green Lantern is designed to produce a range of colours and tints. It was built to simulate the light of railway signals, as they are visible from a distance. It has five rotating discs;

Disc 1 – aperture sizes varies 1.3 to 13 mm.

Disc 2-4 – Eight colour filters (2 red, 2 green, white, yellow, blue, purple)

Disc 5 – a clear aperture, 5 neutral density filters, a ribbed glass (simulate rain), frosted glass (simulate mist)

The test is performed in a dark room at 6 meters distance. Set of filters showing signal red, yellow, green and blue colours are shown, each colour being shown twice for each aperture size. The five rotating discs (containing the coloured filters, the modifying filters, and the apertures) can be rotated singly or jointly, making hundreds of combination possible.

The recommendations of the test state that a candidate should be rejected if he calls:

- Red as Green
- Green as Red
- White light as Green or Red or vice versa
- Red-Green or White light as black.^{2,16}

Computer based Farnsworth-Munsell 100 Hue test

Computer based Farnsworth-Munsell 100 Hue test¹⁵ test is based on the Farnsworth-Munsell 100 Hue test. To the best of their abilities, subjects are required to arrange four sets of tiles on the basis of colour. Tiles are dragged into place by using the mouse. Each tile set has two reference endpoints; therefore only one valid arrangement of the tiles is possible. Once all tiles are in place, the option to graph the results will be enabled. The software scores the tile arrangements and plots them on a colour wheel. The resultant graph will indicate areas of colour deficiency. Scoring is computed for each chip in the manner similar to manual FM 100 hue test. Total error scores (Table 6) are obtained and these scores are displayed in a graphical manner. A higher score indicates greater inaccuracy in placing chips in the correct order.

Interpretation

- The major rings of the graph indicate 2,5,10,15, and 20 error points.
- The perfect graph will be a circle on the 2 ring (total score of 0).
- Minor errors (within the 5 ring) are generally acceptable
- Large imbalances/spikes towards a quadrant are an indicator of color vision weakness in those colours.

Colour discrimination	Score
Superior	0-16
Average	16-80
Low	>80

Rehabilitation of Colour defective individuals

It is advisable to screen school children for colour deficiency in order to help them select suitable vocation. Rubin LR et al. used color and grayscale images to teach histology to color-deficient medical students. They concluded that using this approach, color-deficient students have quickly learned to compensate for their deficiency by focusing on cell and tissue structure rather than on color variation.¹⁷

Tinted contact lenses have been designed for colour deficient individuals. Mutilab HA et al. concluded that

special tinted contact lens used in their study did not cause a reduction of visual acuity and contrast sensitivity for the colour defects. Stereopsis was also not reduced with except when tested with the TNO test. Colour vision defects became difficult to detect using the Ishihara plates but FM100Hue test did not show any improvement with contact lenses.¹⁸

Schornack et al.¹⁹ used tinted spectacle or contact lenses in relieving photophobia associated with a number of cone disorders, including achromatopsia. In addition to decreasing light sensitivity, tinted lenses improve visual acuity, decrease the size of central scotomata, enlarge peripheral visual field, and enhance visibility of long wavelength stimuli in bright illumination

Financial & competing interest disclosure

The authors do not have any competing interests in any product/ procedure mentioned in this study. The authors do not have any financial interests in any product / procedure mentioned in this study.

References

- Swanson WH, Cohen JM. Color vision. *Ophthalmol Clin N Am* 2003; 16:179-203.
- Birch J. Diagnosis of defective colour vision. 2nd ed. Oxford: Butterworth-Heinemann; 1993. P. 125-32.
- Duke-Elder S. Colour vision. In: System of Ophthalmology. 2nd ed London: Henry Kimpton; 1968. p. 617-51.
- Deeb SS. Molecular genetics of color-vision deficiencies. *Vis Neurosci* 2004; 21:191-6.
- Spalding JA. Colour vision deficiency in the medical profession. *Br J Gen Pract* 1999; 49:469-75.
- Katz B. The dyschromatopsia of optic neuritis: a descriptive analysis of data from the optic neuritis treatment trial. *Trans Am Ophthalmol Soc* 1995; 93:685-708.
- Jaeger W. Acquired colour-vision-deficiencies caused by side-effects of pharmacotherapy. *Klin Monbl Augenheilkd* 1977; 170: 453-60.
- No authors listed. Drug-induced colour vision disorders. *Prescribe Int* 2012; 21:126-8.
- Attarchi MS, Labbafinejad Y, Mohammadi S. Occupational exposure to different levels of mixed organic solvents and colour vision impairment. *Neurotoxicol Teratol* 2010; 32:558-62.
- Tan X, Aoki A, Yanagi Y. Color vision abnormality as an initial presentation of the complete type of congenital stationary night blindness. *Clin Ophthalmol* 2013; 7:1587-90.
- Dain SJ. Clinical colour vision tests. *Clin Exp Optom* 2004; 87:276-93.
- Birch J. Efficiency of the Ishihara plate for identifying red-green colour deficiency. *Ophthalm Physiol Opt* 1997; 17:403-8.
- Kakajima A, Ichikawa H, Nagagawao O, Majima A, Watanabe M. Ishihara in colour vision defects. *Am J Ophthalmol* 1960; 49: 921-9.
- Hart WM, Adler FH editors. Colour vision. In: Adler's Physiology of the Eye. 9th ed. Saint Louis (MO). Mosby: 1992; p. 708-27.
- Kinnear PR. Proposal for scoring and assessing the Farnsworth-Munsell 100 hue test. *Vision Res* 1970; 10:423-33.
- Duke-Elder S. Congenital colour defects. In: System of Ophthalmology. 2nd ed. London: Henry Kimpton; 1964. p. 661-8.
- Rubin LR, Lackey WL, Kennedy FA, Stephenson RB. Using color and grayscale images to teach histology to color-deficient medical students. *Anat Sci Educ* 2009; 2:84-8.
- Mutilab HA, Sharanjeet-Kaur, Keu LK, Choo PF. Special tinted contact lens on colour-defects. *Clin Ter.* 2012; 163: 199-204.
- Schornack MM, Brown WL, Siemsen DW. The use of tinted contact lenses in the management of achromatopsia. *Optometry* 2007; 78:17-22.