H 13: Impaired Colour Vision

The first detailed account of impaired colour vision was by the chemist John Dalton and his brother in 1798; as a result it is often called Daltonism. Impaired colour vision can be hereditary or acquired as a symptom of disease or an injury to the head.

The gene responsible for inherited impaired colour vision is carried as part of the X chromosome, which is dominant in the male and recessive in the female. Both parents must be carriers of the defective gene for the daughter to have impaired colour vision but if either parent is a carrier then the son will have impaired colour vision. As a result around 8% of the male population have impaired colour vision and only 0.5% of the female population. Because of the maths involved it follows that 16% of the female population are carriers of the defective chromosome.

Types of colour vision

The words used to describe the types of colour vision are based on the trichromatic theory of colour vision.

Trichromat

A person with normal colour vision has the normal sensitivity of the three cone cell responses and is termed a trichromat and every part of the light spectrum appears coloured to person with this type of vision.

Anomalous trichromat

There is a further class of impaired colour vision where the three cone responses are present however one of them is different sensitivity to that of a standard observer.

Name	Cause	Symptoms
Protanomaly M: 1.0% F: 0.02%	Red cone response curve moved towards the blue end of the spectrum	Red-green discrimination reduced, red colours appear dimmer than normal
Deuteranomaly M: 4.9% F: 0.38%	Green cone response curve moved towards the red end of the spectrum	Red-green discrimination reduced, no abnormal dimming of colours.
Tritanomaly M: unknown F: unknown	Blue cone response curve moved towards the red end of the spectrum.	Blue-yellow discrimination reduced, no abnormal dimming of colours.

The occurrence of these different types of impaired colour vision varies greatly; however, the average occurrence for western races are as follows.

Type of impairment	Men %	Women %
Protanopia	1.0	0.02
Deuteranopia	1.1	0.01
Tritanopia	0.002	0.001
Protanomaly	1.0	0.02
Deuteranomaly	4.9	0.38
Tritanomaly	unknown	unknown
Monochromat	0.003	0.002
Total	**8.01**	**0.43**

Dichromat

A person who is very weak or deficient in one of cone cell responses is termed a dichromat. There are three types of dichromat depending on which cone cell response is impaired. All three types will confuse a certain range of colours and will have a ***neutral point***, a certain narrow range of wavelengths that appear colourless.

Protanopia (M: 1.0%, F: 0.02%)
A person with protonopia displays a lack of the red(long) cone response.
Red and green colours are confused.
The luminous response at long wavelengths normally comes from this signal, hence bright red colours will appear dark .
Equal green and blue cone response occurs at around wavelengths 490 nm to 495 nm hence light of these wavelengths appears colourless.

Deuteranopia (M: 1.1%, F: 0.01%)
A person with deuteranopia displays a lack of the green(medium) cone response.
Red and green colours are confused
The luminous response is essentially normal as the blue and red cone response curves overlap to cover the complete spectrum.
Light of wavelengths from 500 nm to 505 nm appears colourless.

Tritanopia (M: 0.002%, F: 0.001%)
A person with tritanopia displays a lack of the blue(short) cone response.
Yellow and blue colours are confused
The luminous response is essentially normal as the green and red cone response curves overlap to cover the complete spectrum.
Light of wavelengths 568 nm to 570 nm appears colourless.

Monochromats (M: 0.003%, F: 0.002%)
A person who lacks all the cone responses and only has a rod response.
Cannot discriminate between colours, only lightness/darkness.
The luminous response is that of scotopic (rod vision).
All of the spectrum appears colourless.

Effects of ageing

As described earlier, the fovea of the eye contains a yellow pigment which reduces the amount of blue light transmitted to the cones. The amount of pigmentation varies from one individual to another from negligible to an amount that causes a considerable reduction in the blue light reaching the cones. In addition the lens yellows with age. Young children have relatively pigment free lenses; with time the lens develops a yellow-brownish pigment that absorbs ultra-violet and blue light entering the eye. The difference in vision between a group of 16 and 65 year olds due to lens yellowing is twice that due to variation of fovea pigmentation.

The visual process appears to adapt to these gradual changes and differences in judgements by observers with different yellowness of vision only occurs when comparing metameric pairs of samples.

Eating carrots!
Proper nutrition is important to preserve good eyesight. The rod and cone cells use a form of vitamin A to help convert light into nerve signals. The vitamin combines with proteins to make the light-sensitive photoreceptor chemical in the rods and colour sensitive chemicals in the cones. People who are get too little vitamin A can not see well at night.

Testing colour vision

The Ishihara Colour Confusion charts are one of the most common methods of testing for impaired colour vision. The normal format is a chart in which a number is outlined by coloured dots of various sizes against a background of a random pattern of coloured dots. The colours of the number and background are chosen to highlight any confusion between colours caused by impaired colour vision. The test is able to distinguish between a normal trichromat, dichromat, monochromat and anomalous trichromat . However, the test is not able to assess the degree of impairment of colour vision of an anomalous trichromat, more sophisticated tests such as the Farnsworth hundred hue test need to be used.

The Davidson and Hemmendinger Color Rule may be used to demonstrate the influence of yellowness of vision on the judgement of metameric pairs of colours.

Further Information

R Fletcher and J Voke, "Defective Colour Vision, Fundamentals, Diagnosis and Management", Adam Hilger, Bristol, 1985.

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