LEARNING DOMAINS

CLINICAL PRACTICE

PROFESSIONAL GROUPS

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CPD CODE: C-107458

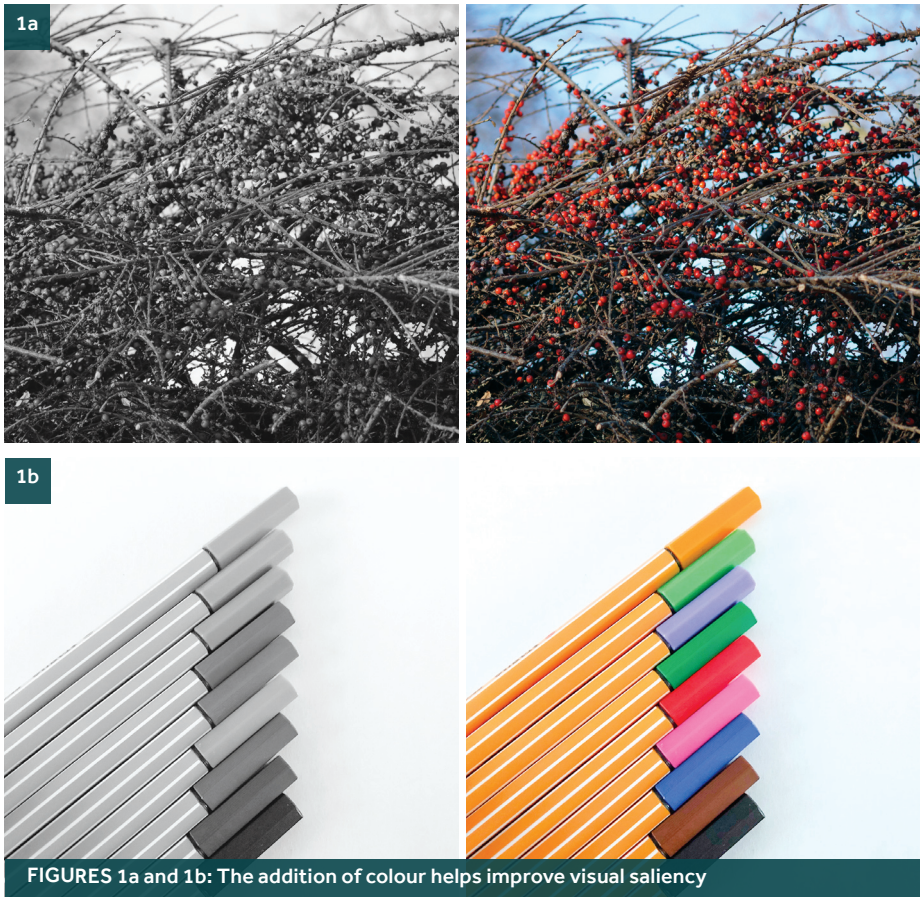
Exploring colour perception

By Mark Hickton BSc (Hons), Cert Ed, FBDO, FHEA

The world we visually experience around us is full of light and colour, and we often take this amazing sensory experience for granted. It may surprise you that there is no such thing *physically* as colour; light that is incident or reflected off objects, and light sources themselves, do not possess colour as a physical property. Only when complex processing within the brain and visual system take place do we perceive colour. This article explores the visual processes relating to human colour perception, including anomalies and pathologies that can affect our perception of colour.

Why do we need colour in our perceptual experience? Visual salience is a perceptual property that determines how much an object in a scene stands out to an observer. Colour, along with other factors such as motion and luminance, helps increase the visual salience of objects within the scene[1].

Figure 1a compares a monochromatic black and white image with a full-colour image of a hedge containing berries. Although the shapes of the berries can be seen in the monochromatic image, they share the same luminosity as other parts of the scene and cannot quickly be discriminated. The application of colour



FIGURES 1a and 1b: The addition of colour helps improve visual saliency

to the image instantly increases the visual salience of the berries in the hedgerow. Similarly, the coloured pens in **Figure 1b** can be individually differentiated when colour is perceived, even when they share the same monochromatic luminance when colour is removed.

Some have suggested that the evolution of colour vision, particularly the red-green system, is an evolutionary enhancement to help animals and humans to detect fruit and ripeness levels more easily, and therefore help discover food sources[2-4]. Colour has also been suggested to aid in courtship by the display of colours in some animals, and even emotional recognition based on blush responses[2,5].

CONES AND WAVELENGTHS

Although colour is a perceptual property, formed by processing within the visual pathways of the brain, there is typically a strong correlation between the colours we experience and the physical wavelength of light. Electromagnetic radiation with wavelengths between 390nm to 760nm represent the human visible part of the electromagnetic spectrum[6], with the longest wavelengths manifesting perceptually as red, and shortest wavelengths perceived as violet. The limits of the visible spectrum are not absolute, and are influenced by factors such as the luminance of light, and the age of the observer[7].

From a physiological context, the process of colour discrimination and perception begins with the cone-photoreceptor cells within the retina. Most humans have **trichromatic** vision, which is supported by the presence of three cone-receptor types within the retina containing different types of iodopsin photopigments. Although some informally label these photoreceptors as red, green, and blue cone cells, they should, perhaps, more accurately be described as long, medium, and short (L, M, and S) cells respectively, based on the range of wavelength of light they respond to.

Rod photoreceptor cells are generally considered achromatic, in that they typically do not contribute to our colour perception; however, there is evidence that rod-cell activity can affect colour perception, especially when functioning alongside the other photoreceptor activity in mesopic conditions[8,9].

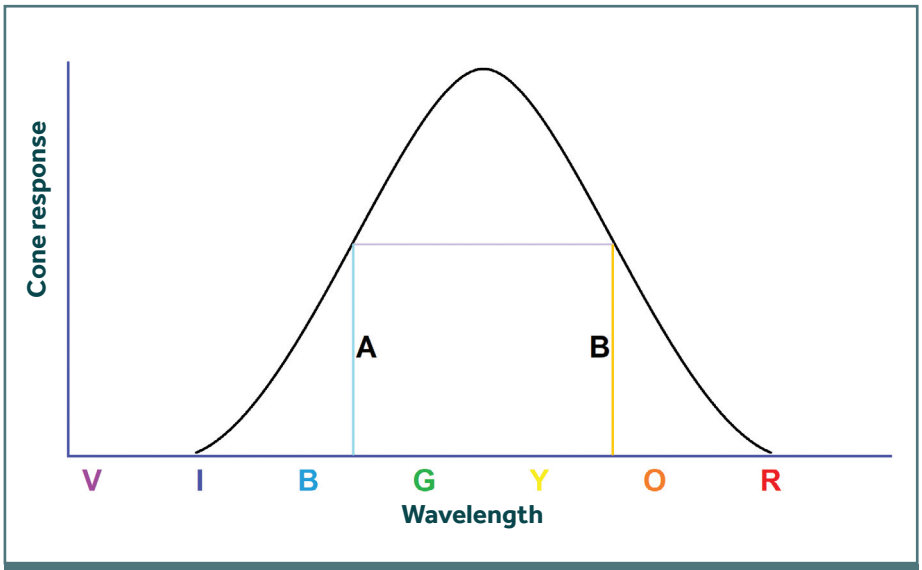


FIGURE 2: An imaginary response curve for a single cone cell, plotting wavelength vs. cell response rate

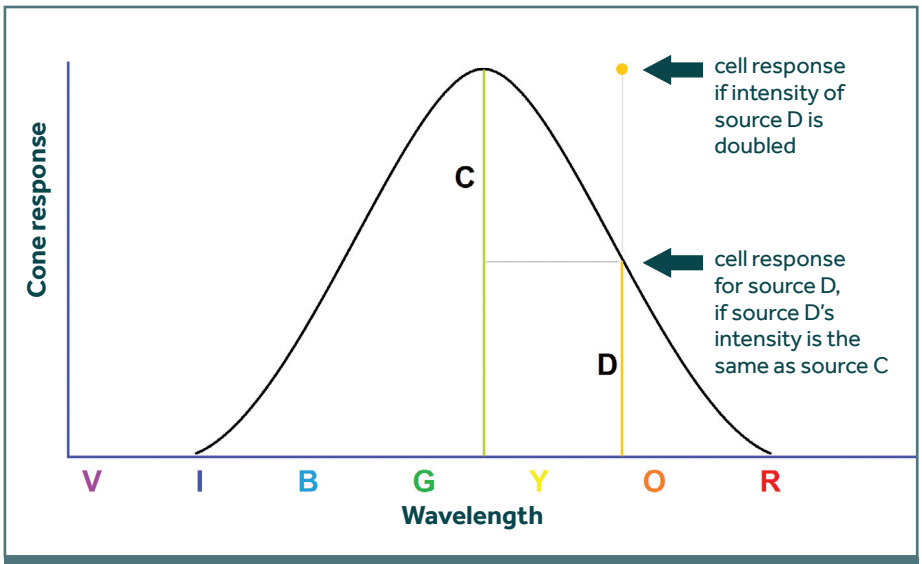


FIGURE 3: An imaginary response curve for a single cone cell, showing the impact of luminance

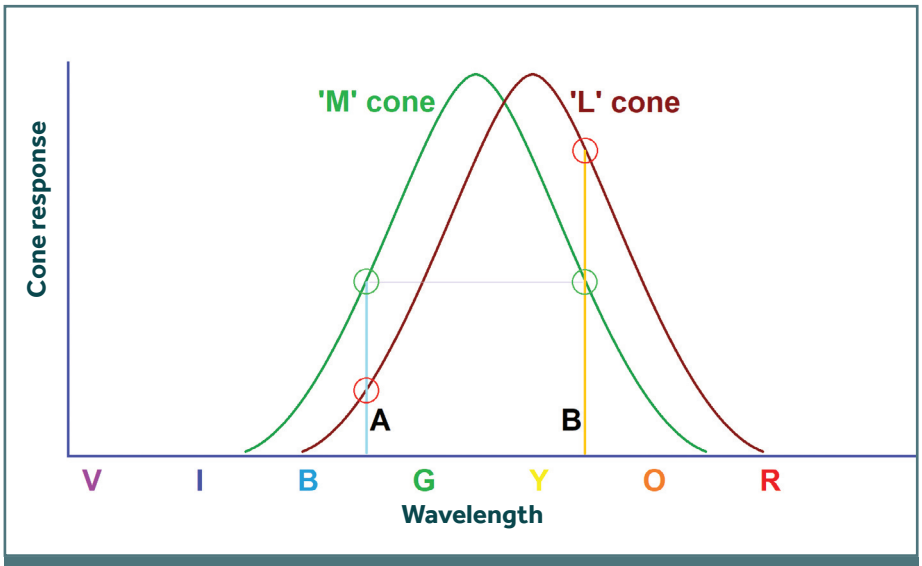


FIGURE 4: An imaginary set of response curves for a dichromatic visual system

	CVD TYPE	DESCRIPTION	APPROXIMATE PREVALENCE	IMPACT
Dichromats	Protanopia	Absence of L-cone 'red' photoreceptors	~ 1% male ~ 0.01% female[10]	Inability to perceive reds and greens. Colour vision consists of blues, yellows, and greys, with restricted long-wavelength perception[2,14]
	Deuteranopia	Absence of M-cone 'green' photoreceptors	~ 1% male ~ 0.01% female[10]	Inability to perceive greens AND reds. Colour vision consists of blues, yellows, and greys. Middle wavelength regions are likely to be perceived more grey than protanopes[2,14]
	Tritanopia	Absence of S-cone 'blue' photoreceptors	<0.01% male and female[10]	Longer wavelengths perceived as red, shorter wavelengths perceived as blue, with the middle wavelengths perceived as grey[2,14]
Anomalous Trichromats	Protanomaly	A dysfunction of the L-cone 'red' photoreceptors	~ 1% male ~ 0.03% female[10]	Confusion with colours relating to medium-long wavelengths and the red-green channel, with more red light required to make colour matches[10,12]
	Deuteranomaly	A dysfunction of the M-cone 'green' photoreceptors	~ 5% male ~ 0.35% female[10]	Confusion with colours relating to medium-long wavelengths and the red-green channel, with more green light required to make colour matches[10,12]
	Tritanomaly	A dysfunction of the S-cone 'blue' photoreceptors	~ 0.2%[20]	Confusion with colours relating to medium-short wavelengths and the yellow-blue channel, with more blue light required to make colour matches[6]
Monochromats	Blue cone monochromat	The patient possesses only a S-cone and rod photoreceptors	~ 0.001%[21-22]	Poor levels of vision, but some colour discrimination between yellows and blues[6]
	Rod monochromat	A complete absence of cone photoreceptors	~ 0.0003%[10]	No colour vision (achromatic), alongside extremely poor levels of vision (especially in photopic conditions)[6,23]

TABLE 1: Forms of CVD and associated perceptual impact

Multiple cone cell types are required to differentiate perceptual colours or hues[4,9,10]; consider an imaginary single medium-wavelength cone cell – as per **Figure 2**.

Figure 2 shows the sensitivity of the cone to varying wavelengths of light, with medium wavelengths initiating the highest response from this cell. Even though this photoreceptor responds best to medium-wavelength light (such as yellow-green), it will also respond when excited by light of other wavelengths within the cell's bandwidth. It can be seen from **Figure 2** that light from source A and light from source B, which have different wavelengths, will produce the same response from this single cell and therefore the light sources will appear perceptually identical.

Additionally, the luminance of light affects our perception of colour; for example, orange light with a fixed wavelength will appear brown at low luminance levels. Returning to our single cone cell (**Figure 3**), we can see that light from source C will produce the maximum cell response, and light from source D will achieve half the maximum response. However, if we double the intensity of source D, we will end up with a similar cell response; again, the single cell in this situation will not be able to support perceptual discrimination between the colour of these sources, despite the sources having different wavelengths.

The inability of a single cone cell to discriminate colour within its bandwidth due to differing wavelengths and luminances is known as the principle of univariance[2,9,10]. Humans and animals that possess visual systems comprising of one type of cone-cell and rod cells, or just rod photoreceptors, are known as **monochromats**. Such visual systems will result in extremely limited colour perception (**see Table 1**). From these discussions, it is clear that possessing only one type of cone-receptor is not sufficient for disentangling different wavelengths and luminances for colour perception[10,11].

Consider now an example of **dichromatic** vision, achieved with the presence of two different cone cell types with different response curves, as per **Figure 4**. In this system, light from sources A and B will produce the same response from the 'M' cone, but a differing response from the 'L' cone. Thus, through comparative signal analysis from multiple cone cells, differentiation of wavelengths and luminance vastly improves colour perception and discrimination.

The typical human trichromatic colour system, supported by the presence of the three cone types, allows for greater colour discrimination compared to a dichromatic system, allowing the perception of more than two million individual colours[2]. Each of the cone cells have peak sensitivities in different parts of the visual spectrum based on the type of iodopsin (cyanolabe (S), chlorolabe (M), and erythrolabe (L)) present within each of the three cone types (**Figure 5**).

The human retina contains approximately six to seven million cone cells, and approximately 120 million rod

cells[2,4] and the distribution of these photoreceptors is not uniform. Most of the M ('green') and L ('red') cone cells tend to be in the macula region, with the greatest concentration in the fovea, whilst the S wavelength ('blue') cells, comprising only around five to 10 per cent of the total cone cells, tend to be absent in the central fovea and scattered more in the peripheral retina[10-14].

Although some animals have more than three cones types (allowing perception of wavelengths invisible to humans) additional cone types are thought to have an adverse effect on visual acuity. It is suggested that the absence of S cone cells in the central fovea helps improve acuity in that region[10] and offsets unwanted chromatic aberration effects[12,13].

In the late 19th century, Hering proposed an opponent-processing theory based on the perception of colour, rather than the physiology of cone cells, showing that colour processing can be decomposed into three colour 'channels': a blue-yellow colour channel, a more evolutionary-recent red-green colour channel, and a third channel relating to achromatic luminosity (or, simplified, white-black)[2,12,14]. Opponent processes are present in the retina and lateral geniculate nucleus[12,15], utilising neural mechanisms that can show excited responses to some wavelengths (i.e. red light) and inhibitory responses to other spectral wavelengths (i.e. green light).

Although the exact neurophysiology that supports colour vision is still debated[16], it is generally agreed that the trichromatic cone physiology and the summation of opponent-channel outputs, along with further processing in the cortex, mediates our perception of colour and allows us to see the multitude of colours in our everyday experience[2,9,17].

HIGHER-ORDER COLOUR PROCESSING

Although it seems from the discussion so far that our perception of colour is purely stimulus-driven, with the wavelength and luminosity influencing photoreceptor activity, there are also higher-order neurological processes that exhibit a top-down influence. Our perceptual system is wired to try to 'see' objects with consistent features regardless of the illumination, distance, and viewing angle.

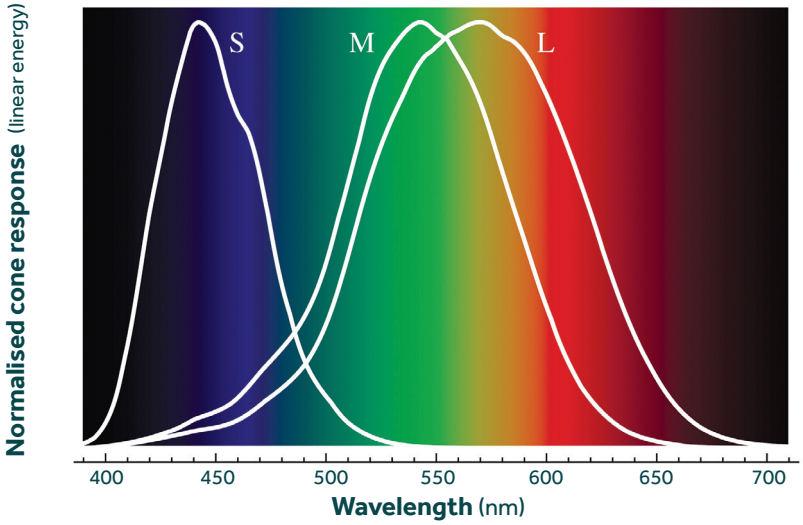


FIGURE 5: Typical cone response curves for human cone photoreceptors in a trichromatic visual system

As an example, consider a white car illuminated by the midday sun. This car will naturally appear white, as it is reflecting white light. However, imagine the same car illuminated by the orange-red light of a sunset. The spectral profile of the reflected light will consist more of the red-orange longer wavelengths, implying that we should see the colour of the car as orange, but this is not the case; most of the time, we will still perceive the car as white, no matter the colour of the light illuminating the object.

Our visual system uses **constancy** mechanisms to allow us to perceive objects normally; colour constancy enables our perception of colour to typically remain relatively stable regardless of the colour of the light illuminating a scene[2,18]. It is believed that the visual system analyses the overall lighting in the scene and, alongside cone chromatic adaption mechanisms[1,10,12], uses this to normalise colour perception of objects within the scene to achieve colour constancy.

Chromatic adaption mechanisms also account for the typical normalisation of colour perception after wearing tinted sunglasses for a time[19]. Although this colour constancy mechanism is reliable under natural viewing conditions, images in which the nature of the lighting is uncertain or ambiguous can lead to mis-interpretation of colour. A famous example of this is the blue-black/white-gold dress, in which multiple observers viewing the same scene will see very different colours, despite being exposed to the same light and wavelengths[1,2].

Experiential knowledge has also been shown to provide a minor contribution to the perception of colour. When subjects have been shown grey-scale images of familiar objects, top-down processing has been found to induce memory colour when viewing such objects[1,2]; for example, grey-scale images of bananas have been shown to elicit a yellow perception of the banana under certain viewing conditions.

COLOUR VISION DEFICIENCY

Congenital colour-vision deficiency (CVD) – sometimes referred to as dyschromatopsia – can present as either an absence or an anomaly of one or more cone receptor types. Patients with only two cone receptor types would possess dichromatic vision, whereas patients possessing only one cone type would present with monochromatic vision. Anomalous trichromatic patients possess all three cone photoreceptor types, however, there is an anomalous shift in the wavelength sensitivity of the photoreceptor response curves which affects colour discrimination[11,20].

The extent of anomalous trichromacy can vary significantly, with some patients experiencing very mild CVD to others that almost experience full dichromacy[6]. There are also extremely rare congenital conditions in which a patient presents with no cone photoreceptor cells, a condition known as rod monochromacy (also known as congenital achromatopsia). **Table 1** details the various forms of CVD, and associated perceptual impact.

CVD corresponding to the absence/abnormality of the L and M cone cells typically have their aetiologies based on X-linked gene mutations. Females have two X chromosomes, and therefore a mutation in one of these may not manifest if the other X chromosome is genetically normal. Males (with only one X chromosome) have a higher prevalence of CVD in relation to L and M cone-cell function, and this reflects in the overall prevalence with around eight per cent of the male population presenting with some form of CVD compared to 0.4-0.5 per cent of the female population[10,24].

Perceptual symptoms in CVD reinforce Hering's opponent-process theory, as typically most CVDs affect either the red-green colour channel, or the blue-yellow colour channel, and not just colours of specific wavelengths. **Table 1** also demonstrates why we should perhaps avoid referring to cone cells by the labels red/green/blue, as blue can be perceived with the absence of S cones in tritanopia, and the perception of green can be absent despite the presence of M cones in protanopia[10,14].

It should be noted that even subjects with normal colour vision can show minor variations in colour perception when asked to match unique colour hues; some suggest this is due to the mosaic distribution of L and M cone cells, and the variable ratio in their numbers[9,15].

There are also forms of *acquired* CVD. Perhaps the most severe is cerebral achromatopsia, a rare condition typically resulting from lesions caused by pathology or trauma in areas of the brain relating to colour processing (such as areas V4 and V8)[10,12,17,25,26]. Such lesions can result in a complete loss of colour perception, in the full or partial visual field, with patients often describing the resulting perception as a monochromatic grey-scale or a sepia-style experience[13,27,28].

As the crystalline lens within the eye ages, it begins to yellow; this reduces the amount of short-wavelength light incident on the retina, affecting the perception of colour and contrast[29]. Additionally, further disruption of colour perception can be attributed to the formation of cataract opacities[29]. As the ageing of the lens is a typically gradual process, patients are unlikely to perceive this slow deterioration due to chromatic adaption mechanisms[19].

After intraocular lens surgery, the sudden change to a clearer optical media often results in patients describing colours as richer and more vivid. Whilst the visual system renormalises to the increased spectral transmission, patients may also experience a blue-tinge to their colour perception (known as cyanopsia) for a few weeks before constancy mechanisms adapt[29,30].

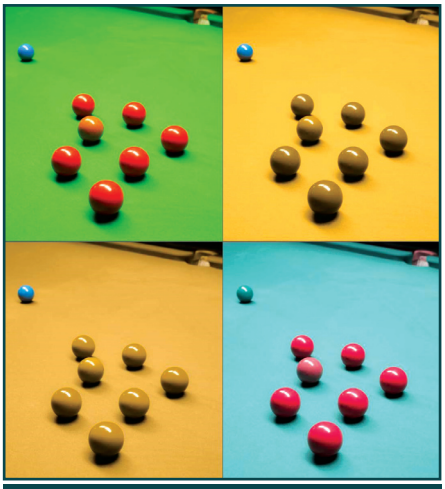


FIGURE 6: Perceptual changes due to CVD[37]
TOP-LEFT: normal colour vision;
TOP RIGHT: protanopia;
BOTTOM LEFT: deuteranopia;
BOTTOM RIGHT: tritanopia.

Other ocular pathologies can have associated CVD symptoms, for example, optic neuritis typically presents as a unilateral inflammation and demyelination of the optic nerve, related to multiple sclerosis, auto-immune deficiencies and infections[31-33]. As well as significant deterioration of visual acuity, acquired CVD typically accompanies optic neuritis. Red desaturation tests can be used to diagnose and monitor optic neuritis by comparing the perceived saturation of colours between the patient's eyes, with the affected eye typically experiencing more desaturation of colour compared to the healthy eye. Other conditions that may present with CVD symptoms include glaucoma, age-related macular degeneration and diabetic retinopathy[24,34].

Köllner's rule can be used to predict the type of acquired CVD based on the pathophysiology; diseases affecting the ocular media and outer retinal layers tend to produce blue-yellow channel colour defects, whereas optic nerve and visual pathway pathology result in colour defects relating to the red-green channel[34]. There are exceptions to Köllner's rule, however, and some pathologies will present with CVDs affecting both colour channels.

Certain medications can also have side-effects that may impact on a patient's colour perception. Digoxin, Hydroxychloroquine, Sildenafil, Tamoxifen, Ethambutol, and Carbamazepine are examples of drugs that can have acquired CVD as a side-effect[24,31,35,36].

CVD TESTING AND CAREER IMPACT

The impact of CVD on quality-of-life and vocation can be highly variable depending on the severity; Dichromatic patients will have significant difficulties with colour discrimination (**Figure 6**), whereas anomalous trichromacy may not impact vastly on the early years of life, and may only present with minor perceptual confusion of colours. The patient, of course, will not realise they have any congenital colour deficiency through their own observations, as they will never experience the full spectral range of a trichromat and assume their perception of the world is 'normal'.

Colour labelling by children may seem intact (i.e. they would still describe red objects as 'red', even though they may experience a different percept of red) and this may often mask the condition from the parent's perspective; thus, it is important to ensure that colour-vision screening is embedded into paediatric development checks.

Whilst colour confusions may raise suspicions with parents or carers of CVD children, it is often the case that CVD is more likely to be detected through screening within the school setting or within an optical practice as part of a sight test. **Table 2** highlights a few experiences, which suggest CVD is present. CVD screening may be overlooked, however, if colour vision confusion is not reported by the child or parent and, as such, CVD may not be diagnosed until well into childhood, or even into the teenage years. A review by Stoianov *et al.*[38] highlights the wide-ranging impact on quality of life that CVD can have, with occupational, educational, emotional, and social detriments included. More information on CVD, its impact and support information, can be found on the Colour Blind Awareness site, ***www.colourblindawareness.org***[39].

"I cannot discern whether a banana is ripe or unripe"
"I have difficulty discriminating between the brown and red balls in snooker"
"Kit clashes whilst I watch football is an issue"
"I did not want to use colour in art at school"
"I have had difficulty with shopping for clothes, and have to ask what matches"

TABLE 2: Examples of indicators for potential CVD

Although CVD is detrimental in most cases, it is suggested that some dichromats are less susceptible to colour-camouflage effects. The reduction of colour saliency allows shape and texture to become more apparent when viewing camouflaged objects and, as such, dichromats have been recruited into military roles in relation to image analysis[10,34,38].

SUGGESTED CVD 'SOLUTIONS'

Whilst there is no cure for human CVD, and many live with this perceptual disability without any corrective optical appliance, there are spectacle and contact lens products available designed to improve colour discrimination for CVD patients[20]. Many of these devices utilise a 'notched' spectral filter, which inhibits the transmission of light wavelengths that generate confusion between colours (often filtering out the wavelengths where the M and L cone response curves overlap).

It is important to note that such products, despite some claims, cannot allow CVD patients to experience normal colour vision[20,40]; rather they may help improve colour-contrast and differentiation of colours for the wearer. There is also the need to have different filters for different classifications of CVD, with many products specifically targeting the more common CVDs, such red-green related CVDs.

Due to the wide range of cone-response shifts that can be encountered, studies have shown the efficacy of these devices can be highly variable[20,40-44]. Additionally, the lenses present with vividly coloured tints, and thus the cosmetics of such devices should be discussed with the patient beforehand when managing their expectations.

Many modern digital devices, operating systems and browsers (such as Microsoft Windows, iOS, and Google) now have settings that allow display colours to be altered to support patients with CVD (**Figure 7**), though visibility and public awareness of these assistive technologies is limited[20,45]. Research into potential solutions is ongoing, and directions in this field include live-feed augmented reality glasses, allowing confusion-causing wavelengths to be controlled in the live viewing scene, and gene-therapy, which has had some success in mice and squirrel dichromacy[20,43,45,46].

IN SUMMARY

Our subjective perception of colour seems effortless in our everyday experience, but it has been seen that numerous processes contribute to our experience of colour. Though this article has covered several mechanisms that contribute to our perception of colour, it does not tell the full story.

Research into colour perception is ongoing, with many other mechanisms proposed to influence our perception of colour, including chromatic adaption, lateral inhibition, and inference based on context and frequency of occurrence[2,26,47]; although this is beyond the scope of this article, it clearly shows the complexity behind the processes responsible for our visual experience, and how much more we need to learn about perceptual mechanisms that form our conscious view of the world around us.

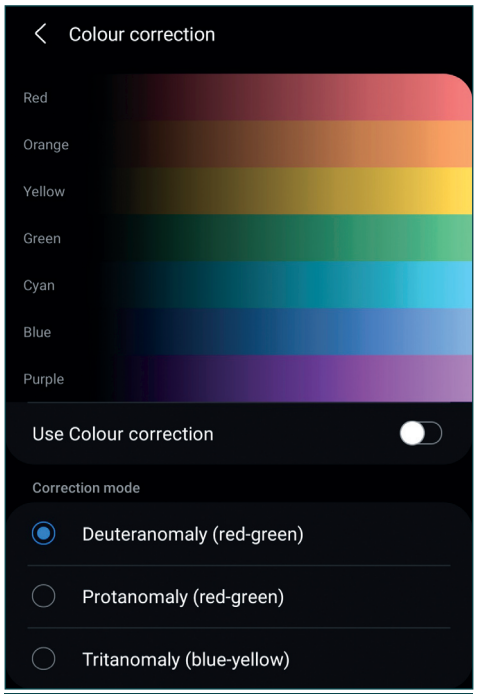


FIGURE 7: Example of digital device colour correction function

REFERENCES

References can be found when completing this CPD module. For a PDF of this article with references, email abdocpd@abdo.org.uk

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LEARNING OUTCOMES FOR THIS CPD ARTICLE

DOMAIN: Clinical Practice

5.3: *Keep up-to-date with the latest information relating to colour perception and consider how you may apply this knowledge to the care you provide.*

7.1: *Conduct an adequate assessment for the purposes of the optician consultation, including within the area of colour vision deficiency.*

7.5: *Provide effective care and treatments for patients with colour deficiency, based on current good practice.*

References

1. Witzel C and Gegenfurtner KR. Color perception: Objects, constancy, and categories. *Annual Review of Vision Science* 2018;4:475-499.
2. Goldstein EB and Cacciamani L. *Sensation and Perception*. 11th ed. Boston: Cengage Learning, 2021.
3. Kremers J, Baraas RC and Marshall NJ (eds.). *Human Color Vision (Vol. 5)*. Cham: Springer, 2016.
4. Bruce V, Green PR and Georgeson MA. *Visual Perception: Physiology, Psychology, and Ecology.* 4th ed. Hove: Psychology Press, 2003.
5. Vorobyev M. Ecology and evolution of primate colour vision. *Clinical and Experimental Optometry* 2004;87 (4-5):230-238.
6. Tunnacliffe AH. *Introduction to Visual Optics*. 4th ed. Canterbury: ABDO, 1993.
7. Sliney DH. What is light? The visible spectrum and beyond. *Eye* 2016;30(2):222-229.
8. Stabell B and Stabell U. *Duplicity Theory of Vision – From Newton to the Present.* New York: Cambridge University Press, 2012.
9. Thoreson WB and Dacey DM. Diverse cell types, circuits, and mechanisms for color vision in the vertebrate retina. *Physiological Reviews* 2019;99(3):1527-1573.
10. Snowden R, Thompson P and Troscianko T. *Basic Vision – An Introduction to Visual Perception.* Revised ed. Oxford: Oxford University Press, 2012.
11. Solomon SG and Lennie P. The machinery of colour vision. *Nature Reviews Neuroscience* 2007;8(4):276-286.
12. Carpenter R and Reddi B. *Neurophysiology – A Conceptual Approach.* 5th ed. London: CRC Press, 2012.
13. Stone JV. *Vision and Brain – How We Perceive the World.* Cambridge: Massachusetts Institute of Technology, 2012.
14. Pashler H and Yantis S (eds.). *Stevens' Handbook of Experimental Psychology.* Sensation and Perception (Vol. 1). USA: John Wiley and sons, 2002.
15. Schmidt BP, Neitz M and Neitz J. Neurobiological hypothesis of color appearance and hue perception. *JOSA A* 2014;31(4):A195-A207.
16. Schmidt BP, Boehm AE, Tuten WS and Roorda, A. Spatial summation of individual cones in human color vision. *PloS one* 2019;14(7):e0211397.
17. Gegenfurtner KR. Cortical mechanisms of colour vision. *Nature Reviews Neuroscience* 2003;4(7):563-572.
18. O'Dea J. Perceptual constancy and the dimensions of perceptual experience. *Phenomenology and the Cognitive Sciences* 2022;21(2):421-434.
19. Tregillus KE and Engel SA. Long-term adaptation to color. *Current Opinion in Behavioral Sciences* 2019;30:116-121.
20. Salih AE, Elsherif M, Ali M, Vahdati N, Yetisen AK and Butt H. Ophthalmic wearable devices for color blindness management. *Advanced Materials Technologies* 2020;5(8):1901134.
21. Sechrest ER, Chmelik K, Tan WD and Deng WT. Blue cone monochromacy and gene therapy. *Vision Research* 2023;208:108221.
22. Patterson EJ, Kalitzeos A, Kane TM, Singh N, Kreis J, Pennesi ME, Hardcastle AJ, Neitz J, Neitz M, Michaelides M and Carroll J. Foveal cone structure in patients with blue cone monochromacy. *Investigative Ophthalmology & Visual Science* 2022;63(11):1-9.
23. Dickinson C. *Low Vision: Principles and Practice.* Edinburgh: Butterworth-Heinemann, 1998.
24. Freeman S. Colour vision in the modern environment. *Dispensing Optics* 2019;34(7):16-22.
25. Carter R. *The Human Brain Book: An Illustrated Guide to its Structure, Function, and Disorders.* 3rd ed. London: Penguin, 2019.
26. Strong S and Fulcher C. High-level processing and visual illusions. *Optometry Today* 2018;58(6):64-68.
27. Bartolomeo P. Color vision deficits. *Current Neurology and Neuroscience Reports* 2021;21:1-7.
28. Goodale M and Milner D. *Sight Unseen: An Exploration of Conscious and Unconscious Vision.* Oxford: Oxford University Press, 2013.

29. Ao M, Li X, Qiu W, Hou Z, Su J and Wang W. The impact of age-related cataracts on colour perception, postoperative recovery and related spectra derived from test of hue perception. *BMC Ophthalmology* 2019;19:1-9.

30. Kitakawa T, Nakadomari S, Kuriki I and Kitahara K. Evaluation of early state of cyanopsia with subjective color settings immediately after cataract removal surgery. *JOSA A* 2009;26(6):1375-1381.

31. Salmon JF. *Kanski's Synopsis of Clinical Ophthalmology.* Elsevier Health Sciences, 2022.

32. Toosy AT, Mason DF and Miller DH. Optic neuritis. *The Lancet Neurology* 2014;13(1):83-99.

33. Bruegger D, Koth AL, Dysli M, Goldblum D, Abegg M, Tschopp M and Tappeiner C. Evaluation of the Reddesa Chart, a new red desaturation testing method for optic neuritis screening and grading in clinical routine. *Frontiers in Neurology* 2022:13.

34. Hasrod N and Rubin A. Defects of colour vision: a review of congenital and acquired colour vision deficiencies. *African Vision and Eye Health* 2016;75(1):1-6.

35. Li J, Tripathi RC and Tripathi BJ. Drug-induced ocular disorders. *Drug Safety* 2008;31:127-141.

36. Rassi SZ, Saint-Amour D and Wittich W. Drug-induced deficits in color perception: Implications for vision rehabilitation professionals. *Journal of Visual Impairment & Blindness* 2016;110(6):448-453.

37. Asada K. *Chromatic Vision Simulator* (Version 3.2) [Mobile app]. (2019). Available at Google Play (Downloaded: January 2023).

38. Stoianov M, de Oliveira MS, dos Santos Ribeiro Silva MCL, Ferreira MH, de Oliveira Marques I and Gualtieri M. The impacts of abnormal color vision on people's life: an integrative review. *Quality of Life Research* 2019;28:855-862.

39. Colour Blind Awareness [Internet]. 2023. Available from: *https://www.colourblindawareness.org*

40. Pattie C, Aston S and Jordan G. Do EnChroma glasses improve performance on clinical tests for red-green color deficiencies? *Optics Express* 2022;30(18):31872-31888.

41. Male SR, Shamanna BR, Bhardwaj R, Bhagvati C and Theagarayan B. Color vision devices for color vision deficiency patients: a systematic review and meta-analysis. *Health Science Reports* 2022;5(5):e842.

42. Almutairi N, Kundart J, Muthuramalingam N, Hayes J, Citek K and Aljohani S. Assessment of EnChroma filter for correcting color vision deficiency. *Pacific University* (Oregon) 2017.

43. Patterson EJ, Mastey RR, Kuchenbecker JA, Rowlan J, Neitz J, Neitz M and Carroll J. Effects of color-enhancing glasses on color vision in congenital red-green color deficiencies. *Optics Express* 2022;30(17):31182-31194.

44. Álvaro L, Linhares JM, Formankiewicz MA and Waugh SJ. (2022) Coloured filters can simulate colour deficiency in normal vision but cannot compensate for congenital colour vision deficiency. *Scientific Reports* 2022;12(1):11140.

45. Sutton J, Langlotz T and Plopski A. Seeing colours: addressing colour vision deficiency with vision augmentations using computational glasses. *ACM Transactions on Computer-Human Interaction* 2022;29(3);1-53.

46. Michalakis S, Gerhardt M, Rudolph G, Priglinger S and Priglinger C. Achromatopsia: genetics and gene therapy. *Molecular diagnosis & Therapy* 2022;26(1):51-59.

47. Lotto RB and Purves D. The empirical basis of color perception. *Consciousness and Cognition*, 2002;11(4):609-629.