Adaptive Plasticity of the Colorblind Brain: A Model for Sensory Compensation

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Abstract

Color vision deficiency (CVD) or color blindness results from X-linked recessive genetic mutation that decreases or impairs the expression of cone cell photoreceptors essential for normal color perception. As a result, individuals with color blindness are unable to distinguish certain colors or hues in the same way as individuals with typical color vision. On a molecular level, the most common forms of CVD arise from the absence or malfunction of one type of cone cell in the retina, which reduces sensitivity to specific wavelengths of light. This disruption in normal color processing leads to altered color perception, often making daily visual tasks more challenging. However, the colorblind brain can adapt to these perceptual differences through neural plasticity. Recent neuroscience research indicates that visual cortical areas V2 and V3 are particularly involved in cortical reorganization in individuals with CVD. Additionally, at the cellular level, structures such as rods, intrinsically photosensitive retinal ganglion cells (ipRGCs), and neurons in the lateral geniculate nucleus (LGN) may contribute to compensatory neuroplastic responses to altered visual input. By using current research on the adaptive plasticity of the brain in color blind people, scientists can further the potential of neural training for rehabilitation and therapeutic strategies targeted to treat brain trauma, injuries, or other visual impairments.

Introduction

Around 1 in 12 men and 1 in 200 women are colorblind (Fareed, 2015), yet we rarely consider how they perceive the world around us. Colorblindness is often dismissed as a minor inconvenience, but it could offer a unique opportunity to study how the brain adapts to sensory deficits.

Color blindness, or color vision deficiency (CVD), is a condition characterized by a decreased ability to perceive color differences under normal light conditions and can be genetic or acquired due to trauma. In CVD, cone cells in the eye retina fail to process color information correctly due to malfunctioning or missing opsin proteins (Simunovic, 2009). Depending on the mutation, colorblind individuals may experience anomalous trichromacy (opsins are present but less sensitive), dichromacy (one of the cone types is missing), or monochromacy (all cones are missing or non-functioning). The most common forms, protanomaly (red-weak) and deuteranomaly (green-weak), result in a shifted perception of color, altering how individuals interact with their environment. Current research suggests that the altered photoreceptor function in individuals with color vision deficiency may influence neural processing at the cortical level (Rina, 2024), introducing changes in brain function, especially its plasticity—the brain's ability to 'rewire' itself due to injury or experience (Puderbaugh, 2023). Unlike sudden sensory loss, congenital color blindness is a lifelong deficiency, allowing researchers to explore how the brain adapts to deficiency from an early age (Isherwood, 2020). As a result, studying these adaptations may offer valuable clues for developing therapies to restore vision or improve recovery after brain injury. This review will discuss neural plasticity associated with colorblindness and how current literature suggests these insights can be used to inform broader neuroscience research on therapies for sensory deprivation and sensory repair.

Mechanisms of Color Vision

The physiology of color vision is thought to be the same across all species, yet scientists still have much to uncover. At its core, color vision relies on our brain's ability to analyze the energy and frequency of light scattered by an object, using opsins—light-activated protein receptors—embedded in cell membranes of photoreceptor cells. So, color processing in humans involves two organs: the retina and the brain, specifically the visual cortex in the occipital lobe. Photoreceptor cells, or photoreceptors, are specialized neurons in the eye retina that detect light. There are two major types of photoreceptors in humans: rods that are used for vision in the dark and cones that are used to detect color via opsin proteins, which are, therefore, crucial for understanding color vision deficiency.

Humans have opsin proteins sensitive to short (S; maximally sensitive to blue wavelength light), medium (M; maximally sensitive to green wavelength light), and long (L; maximally sensitive to red wavelength light) wavelengths, resulting in routine trichromatic vision (Isherwood, 2020; Pasmanter). To understand how opsins can detect specific colors of light and transmit signals to the brain, we need to explore the physics of light and the molecular structure of opsin proteins.

According to the electromagnetic spectrum theory in physics, all electromagnetic radiation, which includes light, can be characterized by its wavelength and energy. The visible portion of the electromagnetic spectrum, which we perceive as colors, has wavelengths ranging roughly from 400 to 700 nanometers. Within this spectrum, different wavelengths correspond to different colors; for example, as shown in Figure 1, longer wavelengths appear red, while shorter wavelengths appear violet or blue (Ailioaie, 2020). When referring to S, M, or L cones (or also S, M, or L opsins in other literature), we mean that each type is activated by a specific range of wavelengths with a corresponding energy. This activation induces a conformational change in the opsin protein, triggering a cascade of biochemical reactions that transmit a signal to the brain. As illustrated in Figure 2, opsins act as G-protein coupled receptors. In their signalling conformation opsins can bind to and activate the G protein by catalysing the exchange of GDP (guanosine diphosphate) to GTP (guanosine triphosphate). The GTP-bound Gα subunit dissociates from the Gβγ subunit exposing its active site and binding to its effector, phosphodiesterase. Phosphodiesterase then starts a cascade of reactions that eventually create a hyperpolarization response in the cones. This membrane hyperpolarization in cones modulates the release of neurotransmitters to ganglion cells which form the optic nerve, finally projecting the signal to the brain (Shichida, 2009).

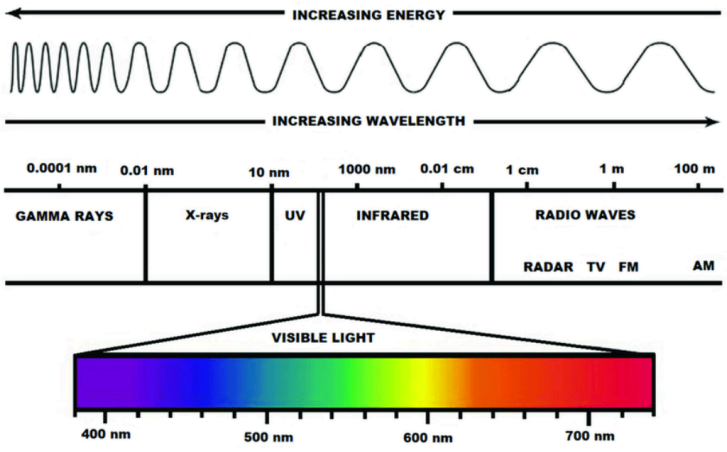

Figure 1. The visible spectrum of light inside the electromagnetic radiation spectrum (Ailioaie, 2020)

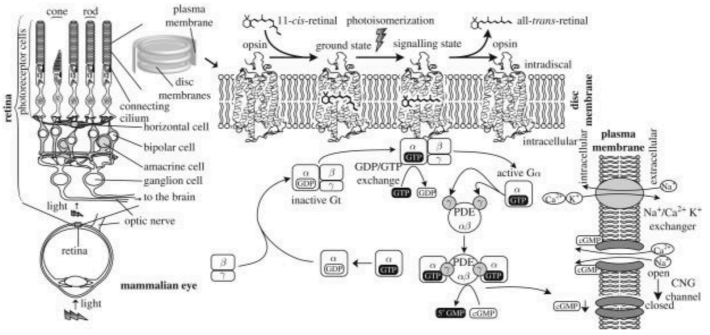

Figure 2. A schematic showing the molecular and physiological mechanism of phototransduction from the retina in mammalian eyes. This schematic uses bovine rhodopsin as an example of opsin protein and its function (Shichida, 2009)

The mechanism of color vision discussed so far is known as trichromatic color theory, which states that our perception of color relies on detecting signal intensities from three types of cones—S, M, and L cones—that correspond to blue, green, and red light. Psychology research also suggests other color vision models, like opponent processing theory, that act in tandem with trichromatic theory to allow us to perceive colors differently (Lee, 2011). Further color encoding (e.g. detecting color hues) depends on neural activity in the visual cortex to compare if a given wavelength, for example, excites M or L cone receptors more (Isherwood, 2020).

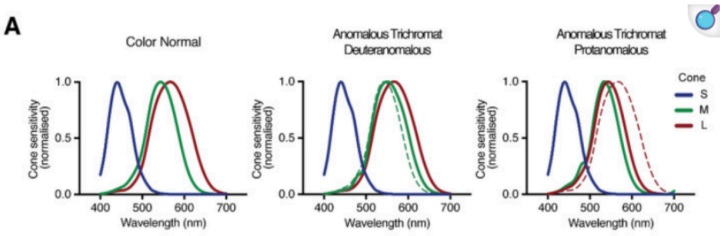

Figure 3. Overlapping sensitivities for different color vision deficiencies (Isherwood, 2020).

Figure 3 from a 2020 review by Isherwood illustrates differences in S, M, and L cone sensitivities in normal color vision as compared to trichromat deuteranomalus or trichromat protanomalus vision. In deuteranomalus vision, the green spectrum(i.e. range of absorbed wavelengths that result in green color) overlaps more with the red spectrum, resulting in green-color weakness. Similarly, the red spectrum shifts closer to the green spectrum sensitivity in protanomalous vision, resulting in a red-color weakness. There are also CVDs where one type of cone is completely missing or non-functioning, resulting in a more drastic change of color vision. When M cones are missing, an individual experiences deuteranopia, leading to the inability to detect green light. Missing L cones leads to protanopia—the inability to detect red light.

Scientists can use computational tools to visualize how individuals with these conditions experience color, see Figure 4 (Wong, 2011). Figure 3 displays a simulation of dichromat percept with decreased L-M light spectrum comparison, assuming this image is a close representation of how protanopes and deuteranopes perceive color.

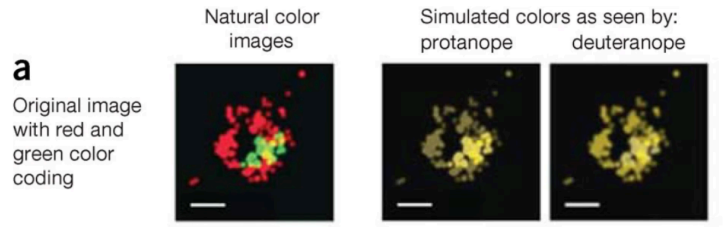

Figure 4. Immunofluorescence image in original color (red and green) and simulated images as seen by protanopes and deuteranopes (Wong, 2011)

Although color blindness might result from a brain injury or eye disease (Cowey, 1997), it is most commonly diagnosed as a congenital condition. The genes that code for opsins are located on the X chromosome. This explains why men are more likely to be color blind than women because mutation on the X chromosome is guaranteed to be expressed in the male population with only one X chromosome copy and not two copies like in females. The genetic designations for the L and M opsin genes are OPN1LW and OPN1MW, respectively. Mutations in these genes, therefore, lead to color vision deficiencies, such as protanopia or deuteranopia, when affecting either OPN1LW or OPN1MW. On a physiological level, mutation in these genes leads to decreased or absent expression of opsin receptors, affecting visual perception of contrast sensitivity, color discrimination, and object recognition. As a result of these genetic changes, colorblind individuals often rely on several post-receptoral adaptations (Isherwood, 2020)—processes in the neural activity of the colorblind brain that help individuals with CVD compensate for receptoral malfunction. Such adaptations are of great interest in neuroscience because they pose questions about mechanisms of neuroplasticity and how the brain adjusts to CVD on the level beyond the eye retina.

Figure 5: Simulated dichromat percept of color (Isherwood, 2020)

Neural Plasticity in Color Vision Deficiencies

Color blindness is a unique "natural experiment" (Isherwood, 2020) to study neural plasticity due to two main reasons. Firstly, CVDs arise from a discrete change on the first step of color vision, which allows us to study brain reorganization as a result of a constant and simple change in informational input like the altered light detection (Isherwood, 2020). Secondly, because each color blind individual spends a lifetime experiencing a defective color vision, which serves as a valuable opportunity to study brain plasticity on timescales much larger than scientists can afford in the lab (Isherwood, 2020).

It is obvious that CVD causes perceptual changes at the retinal level of color vision, but neuroscientists are also interested in studying how deficient light input changes cortical function. Some studies hypothesize that neuroplasticity adapts to CVD through cortical reorganization, where the brain assigns a function to a cortical area that it does not normally have due to altered sensory input.

There is evidence of strong compensation for color losses in anomalous trichromacy via amplification of cortical responses to chromatic contrast in the V1 (primary visual cortex), V2 (secondary visual cortex), and V3 (V1 and V2 signal processing; motion processing) areas of the visual cortex (Tregillus 2021; Huff; Arcaro, 2015). Interestingly, in an fMRI study examining brain activity in colorblind and healthy subjects performing tasks requiring attention fixation on image contrast sensitivity, researchers found that V1 activity was decreased in colorblind individuals, while V2 and V3 activity remained unchanged, meaning that these parts of the visual cortex might play a role in color processing and associations in color blind individuals, see Figure 4 (Tregillus 2021).

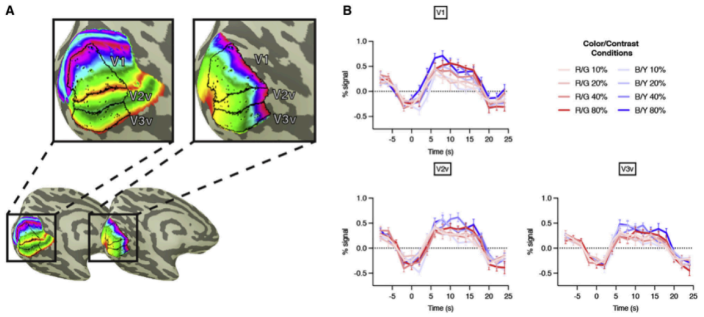

Figure 6. fMRI data on V1, V2, and V3 cortical activity during attention heavy experiments. A. CVD brain activity is on the right of A being compared to normal brain function on the left of A. Retinotopic polar-angle and eccentricity maps (0–9.5°) overlaid on an inflated left hemisphere for V1, V2v, and V3v. The central 0–0.95° (fixation zone) was excluded. B. Elucidation of the results in A heat maps with plots of fMRI signal change. Mean % signal change in V1, V2v, and V3v for one subject, averaged over 6 runs (12 repeats per condition). Each block had 14 s of stimulus followed by an 8 s gray-screen rest. (Tregillus, 2021).

Additional research supports the idea that chromatic adaptation occurs at the retinal level and within central visual pathways. Studies on the McCollough effect (a visual illusion that causes color aftereffects) suggest that some chromatic adaptation mechanisms operate at an early monocular stage (Stromeyer, 1978). However, further experiments indicate that normalization mechanisms extend beyond the retina. Electroretinography (ERG) recordings showed no significant differences in spectral sensitivity before, during, or after chromatic alteration, suggesting that these effects are mediated at a post-receptoral level (Neitz, 2002). Furthermore, monocular chromatic alteration experiments demonstrated interocular transfer of color perception shifts, supporting that chromatic adaptation occurs within central visual pathways at a postsynaptic locus where chromatic information from both eyes has already been integrated (Neitz, 2002). Other studies also suggest that the cortex shows much more plasticity related to color-contrast adaptation compared to the lateral geniculate nucleus (LGN), which is a part of the thalamus that relays information from the retina to the visual cortex or retinal cells (Isherwood, 2020). These findings further raise the significance of cortical plasticity as the cortex seems more adaptive to changes in light input and not anatomical areas that come first in relaying visual information.

On a cellular level, current research suggests that intrinsically photosensitive retinal ganglion cells (ipRGCs) mediate color processing (Raja, 2023), and can be considered a part of adaptive neuroplasticity in color blindness. While traditionally associated with non-image-forming functions such as circadian regulation, sleep, mood, and cognition, ipRGCs also receive input from cones and rods, and project to visual pathways, influencing both brightness and color percepts (Isherwood, 2020). This raises intriguing possibilities for their role in color vision deficiencies, particularly in dichromats lacking one cone type. Despite the absence of a full trichromatic signal, dichromats can still reliably categorize colors in ways that align with trichromats, a phenomenon attributed to sensory mechanisms and learned associations. Some studies suggest that dichromats can achieve a form of functional trichromacy over large visual fields by utilizing variations in spectral sensitivity across the retina or by incorporating rod-based signals (Isherwood, 2020). Although rods are generally considered "color blind," they have been shown to contribute to color perception under certain conditions. Given that ipRGCs integrate inputs from cones and rods, they may play an unrecognized role in color perception, particularly in individuals with color vision deficiencies who rely more heavily on alternative visual pathways (Isherwood, 2020). While the specific contributions of ipRGCs to color processing in dichromats remain unexplored, their distinct signaling properties may offer a valuable test case for investigating alternative mechanisms of color coding in the visual system.

Rehabilitation and Assistive Technologies

Given recent advancements in neuroscientific research on brain plasticity in colorblind individuals, this knowledge can be applied to developing rehabilitation and assistive technologies for optic injuries. Understanding neuroplasticity mechanisms in the visual cortex allows for creating personalized treatment approaches based on an individual's plasticity pattern. One common approach to aiding color blindness is using color-correcting glasses tailored to a person's specific receptor sensitivity. However, some researchers are also exploring gene therapy as a potential method to alleviate or even cure color blindness (Dougherty, 2024). More importantly, insights into brain plasticity extend beyond color blindness and can aid in treating brain injuries. Recent studies suggest that doctors can use neural training techniques, such as virtual reality therapy and constraint-induced movement therapy, to help the brain recover from damage (Zotey, 2023). A deeper understanding of neuroplasticity could make neural training a key component of non-invasive rehabilitation therapies. Additionally, research on the plasticity of the visual cortex may provide valuable insights into treating vision-related injuries (Barton, 2020) and conditions such as myopia (Tan, 2008).

Conclusion

In conclusion, color blindness offers a unique perspective on adaptive neuroplasticity, especially in the brain's visual cortex. Interestingly, the brains of individuals with different types of color blindness are more likely to experience structural and functional changes in the cortex and not in the retina. Hence, studying color blindness brings more attention to cortical neuroplasticity because these areas seem to play a more significant role in adaptation to vision impairment when compared to neurons and receptors involved in the first steps of color vision. Brain areas most involved in color vision adaptation are V1, V2, and V3 areas of the visual cortex, and research shows that V2 and V3 play a role in cortical reorganization of colorblind individuals. However, some studies also explore the extent to which cellular retinal structures like rods, ipRGCs, or neurons in LGN contribute to adaptive neuroplasticity as a response to altered light input. By using the knowledge about the adaptive neuroplasticity of the colorblind brain, scientists are looking to study neural training for rehabilitation and therapeutic technologies to treat brain injuries or visual impairments.

References

1. Ailioaie, Laura & Litscher, Gerhard. (2020). Molecular and Cellular Mechanisms of Arthritis in Children and Adults: New Perspectives on Applied Photobiomodulation. International journal of molecular sciences. 21. 10.3390/ijms21186565.

2. Andriani, R. (2024). Investigating neural processing of color in normal and impaired vision. medRxiv. https://doi.org/10.1101/2024.12.22.24319498

3. Arcaro, M J, and S Kastner. "Topographic organization of areas V3 and V4 and its relation to supra-areal organization of the primate visual system." Visual neuroscience vol. 32 (2015): E014. doi:10.1017/S0952523815000115

4. Barton, J. J. S., & Ranalli, P. J. (2020). Vision therapy: Ocular motor training in mild traumatic brain injury. Annals of Neurology, 88(3), 453–461. https://doi.org/10.1002/ana.25820

5. Dougherty, E. (2024). Color therapy. Harvard Medical School Magazine. Retrieved from https://magazine.hms.harvard.edu/articles/color-therapy

6. Fareed, M., Anwar, M. A., & Afzal, M. (2015). Prevalence and gene frequency of color vision impairments among children of six populations from North Indian region. Genes & Diseases, 2(2), 211–218. https://doi.org/10.1016/j.gendis.2015.02.006

7. Huff T, Mahabadi N, Tadi P. Neuroanatomy, Visual Cortex. [Updated 2023 Aug 14]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK482504/

8. Isherwood, Z. J., Joyce, D. S., Parthasarathy, M. K., & Webster, M. A. (2020). Plasticity in perception: Insights from color vision deficiencies. Faculty Reviews, 9, 8. https://doi.org/10.12703/b/9-8

9. Lee, B. B. (2008). The evolution of concepts of color vision. Neurociencias, 4(4), 209–224.

10. Neitz, J., Carroll, J., Yamauchi, Y., Neitz, M., & Williams, D. R. (2002). Color perception is mediated by a plastic neural mechanism that is adjustable in adults. Neuron, 35(4), 783–792. https://doi.org/10.1016/s0896-6273(02)00818-8

11. Pasmanter N, Munakomi S. Physiology, Color Perception. [Updated 2022 Sep 12]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK544355/

12. Puderbaugh M, Emmady PD. Neuroplasticity. [Updated 2023 May 1]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK557811/

13. Raja, S., Milosavljevic, N., Allen, A. E., & Cameron, M. A. (2023). Burning the candle at both ends: Intraretinal signaling of intrinsically photosensitive retinal ganglion cells. Frontiers in Cellular Neuroscience, 16. https://doi.org/10.3389/fncel.2022.1095787

14. Shichida, Yoshinori, and Take Matsuyama. "Evolution of opsins and phototransduction." Philosophical transactions of the Royal Society of London. Series B, Biological sciences vol. 364,1531 (2009): 2881-95. doi:10.1098/rstb.2009.0051

15. Simunovic, M. (2010). Colour vision deficiency. Eye, 24, 747–755. https://doi.org/10.1038/eye.2009.251

16. Tan, D. T., & Fong, A. (2008). Efficacy of neural vision therapy to enhance contrast sensitivity function and visual acuity in low myopia. Journal of Cataract and Refractive Surgery, 34(4), 570–577. https://doi.org/10.1016/j.jcrs.2007.11.052

17. Tregillus, K. E. M., Isherwood, Z. J., Vanston, J. E., Engel, S. A., MacLeod, D. I. A., Kuriki, I., & Webster, M. A. (2021). Color compensation in anomalous trichromats assessed with fMRI. Current Biology, 31(5), 936–942.e4. https://doi.org/10.1016/j.cub.2020.11.039

18. Zotey, V., Andhale, A., Shegekar, T., & Juganavar, A. (2023). Adaptive neuroplasticity in brain injury recovery: Strategies and insights. Cureus, 15(9), e45873. https://doi.org/10.7759/cureus.45873

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