



OPEN ACCESS

SUBMITTED 03 January 2025
ACCEPTED 05 February 2025
PUBLISHED 11 March 2025
VOLUME Vol.07 Issue03 2025

CITATION

Kakharova Dildora Maribjanovna. (2025). Color perception disorders. The American Journal of Medical Sciences and Pharmaceutical Research, 7(03), 39–42. <https://doi.org/10.37547/tajmspr/Volume07Issue03-06>

COPYRIGHT

© 2025 Original content from this work may be used under the terms of the creative commons attributes 4.0 License.

Color perception disorders

Kakharova Dildora Maribjanovna

Andijan State Medical Institute, Uzbekistan

Abstract: Daltonism is a vision disorder in which the eye is unable to perceive one or more primary colors. This disorder is caused by a defect in the X chromosome. However, this is not the only cause of the disease. Color perception may be impaired due to eye or nervous diseases, traumatic brain injury, severe flu, stroke, or heart attack.

This pathology was named after the English chemist John Dalton, who also suffered from this disease, like his relatives, discovered and described the pathology in a book.

Keywords: Color perception disorders, color blindness, pathology, treatment, features.

Introduction: Color vision in humans is provided by special light-sensitive receptors located in the retina—cones. Various pigments contained in the cones allow detecting three color spectrums:

- 1) red with a wavelength of 552-557 nanometers
- 2) green with a wavelength of 530 nanometers
- 3) blue with a wavelength of 426 nanometers.

The transmission of the gene for color blindness is linked to the X chromosome and is almost always inherited from a carrier mother to her son. In men, the defect in the X chromosome is not compensated. Today, 2-8% of men and only 0.4% of women suffer from color blindness. Acquired color blindness can develop in an eye if there is damage to the retina or optic nerve. Before the disease develops, difficulties appear in distinguishing yellow and blue colors.

Monochromacy is a change in color perception in which a person sees the world only in one color. The disease is classified as an acquired disorder.

Dichromacy is a change in color perception in which there is a structural disorder of the eye.

Daltonism is a congenital (less commonly acquired) disease that manifests in various defects in color perception. This feature of vision appears not only in humans but also in primates.

There are some theories of color vision, and one of them is the trichromatic theory of vision. It was first proposed

by Mikhail Vasilyevich Lomonosov in 1756 when he wrote "On the Three Matters of the Eye's Depth." In 1802, Thomas Young suggested that the human eye has three types of color receptors, each of which is sensitive to red, green, and violet. Half a century later, in 1853, the theory of T. Young was supplemented by the German biologist and physicist Hermann Helmholtz. He suggested that to obtain different shades, three primary, pure colors are necessary: red, green, and blue. As a result of their mixing, intermediate colors are produced.

Another theory is the opponent-process theory. In 1870, the German physiologist Ewald Hering interpreted the results of color mixing, concluding that the visual system contains three substances, each of which perceives the colors of its opponent pair: red and green, blue and yellow, black and white. Depending on the spectral composition of the perceived light, either synthesis or decomposition of these substances occurs, creating the sensation of color.

There are also other theories of color vision. For example, in 1975, the Soviet scientist Sergey Dmitrievich Remenko proposed a nonlinear two-component theory of vision, according to which the human visual system contains only two types of light-sensitive elements—uniform rods and cones, which contain pigments highly sensitive to multiple regions of the spectrum. This theory suggests the nonlinearity of the processes of color signal formation. The nonlinear theory of S.D. Remenko describes the mechanisms of signal processing by receptors, maintaining white balance, and models the overall function of the eye. However, it did not gain widespread acceptance. There are various opinions on this topic. It is also important to note that the listed theories and hypotheses are not agreed upon and often contradict each other.

Problems with color vision disorders were covered in the works of the English physicist John Dalton. According to statistics, the percentage of people with normal color vision is 90-92%, while about 8-10% are partially or completely "color blind." In 1794, J.

Dalton first conducted research and described the vision defect, which was named daltonism—complete or partial color blindness. This disease is most often diagnosed in men. Women, as a rule, only pass the pathology on by inheritance.

Thus, it can be concluded that light-sensitive cones responsible for color perception are located on the X chromosome.

There is another alternative theory presented in the book of the famous American neuroscientist Mark Changizi "The Vision Revolution: What, How, and Why

We See the Way We Do." In his work, he outlines the essence of his proposed "skin hypothesis." Mark Changizi believes that human color vision evolved for observing significant and minor changes in skin tone, which are determined by two physiological indicators: excess/lack of blood and excess/lack of oxygen in the blood. When blood accumulates under the skin, as in a bruise, a blue color appears; if there is a lack of blood, a yellowish tint appears. With excessive oxygenation, the skin turns red; with oxygen deficiency, it slightly turns green. Each of these processes helps determine a person's physical and emotional state, which is particularly important in sexual signaling and disease recognition. The neuroscientist's ideas allow several conclusions to be drawn. Our skin, besides its thermoregulatory, water-repellent, and elastic properties, has remarkable, almost magical, color properties—it can become colorful.

To diagnose color perception disorders in medicine, various methods are used. The most well-known and popular diagnostic tool is the Ishihara test. It consists of polychromatic plates displaying numbers or geometric figures composed of dots of varying color and brightness. Against the background of these dots, different colors are used to highlight numbers or shapes. A person with normal vision can easily distinguish them from the rest. If there are color perception disorders, the patient will not be able to see the hidden figures. Using the Ishihara test, the type of color blindness is determined based on the colors and number of figures identified by the patient.

The images are placed one meter from the eyes at the same level as the patient, in good even lighting. The patient examines the image for 7–10 seconds and then describes what they see. The plates include trick images, which only colorblind individuals with a specific type of color blindness can see. The doctor can determine which pigment is missing in the retina using these tables.

To identify those who may try to cheat the test for some reason, special control images are created. These plates display figures that are clearly and distinctly visible both to people with normal vision and those with any form of color blindness. A total of 48 plates are used. The first 27 are the main ones, while the remaining 21 are additional, allowing for a more in-depth analysis of vision disorders. They are mandatory for professional examinations of drivers, train operators, and pilots.

Color blindness testing with Ishihara plates is used worldwide and provides the most accurate results regarding the type and severity of the disorder. The control images refine the diagnosis.

Another diagnostic method for color blindness is the use

of the Ishihara plates. This method also consists of cards. They help identify which specific color perception is impaired. There are four sets of cards, each revealing a pathology related to one of the primary colors.

The cards in the first set are used to detect impairments in the perception of red and its shades. The second set identifies pathologies related to the inability to perceive green shades, the third set is for blue shades, and the fourth set contains black-and-white text for familiarization purposes [11].

For diagnosing this condition, the Polychromatic Rabkin plates are used. There are 27 pages with images in the form of numbers or geometric shapes made up of circles and dots of equal brightness against a background of pale-colored circles.

A person with normal color vision (trichomats) can identify the numbers in this form. A color-blind person, with blindness to one or more colors, will not see the numbers or shapes on the pages. The table helps determine which color the vision cannot perceive. Doctors also use the Ishihara test to assess background perception disorders. In photographs with patches of different colors, some of them, with a uniform shade, form a number, letter, or shape. A person with color blindness will not see the image.

The disease can be diagnosed in children from the age of three. Before this age, children do not see many colors due to physiological reasons. If this pathology is present at birth, the child should be examined by an ophthalmologist once they reach the age of three.

Color blindness that is inherited cannot be treated. Special corrective lenses with a special coating exist that enhance certain colors, but they distort surrounding objects. Ophthalmologists recommend tinted glasses, which help improve color perception in dim lighting.

Types of Color Blindness

- 1) Achromacy – the inability to distinguish colors: in this case, a person perceives only shades of gray.
- 2) Monochromacy – the inability to see the full range of colors. Only one color and its spectrum of shades are available.
- 3) Dichromacy – two colors are available. It is divided into: protanopia – the inability to see red; deutanopia – problems with perceiving green; tritanopia – the inability to perceive blue-violet colors.
- 4) Trichromacy – all three primary colors are perceived well. It can be normal and abnormal.

The last type lies between normal trichromacy and dichromacy. If in typical dichromacy there is no ability

to distinguish between two colors, then in anomalous trichromacy, a person does not struggle with the colors themselves but with their shades. Just like in dichromacy, here we also distinguish prot-, deuter-, and tritanomaly, in which the perception of the red, green, and blue parts of the spectrum is weakened, respectively.

Sometimes, when certain shades are not perceived, there is a compensatory enhancement of the perception of other colors, thereby balancing the existing deficiency. For example, patients who cannot distinguish red tones from green ones can perfectly distinguish khaki shades in a quantity inaccessible to healthy people.

Molecular Mechanisms

The light-sensitive pigment rhodopsin consists of the protein opsin and the chromophore 11-cis-retinal. Photoisomerization of retinal into the trans-form activates the G-protein transducin, which binds to phosphodiesterase, catalyzing the hydrolysis of cGMP. This leads to the closure of cGMP-dependent sodium channels. The potential difference across the membrane increases, and hyperpolarization spreads to the synaptic terminal, where a nerve impulse is generated and transmitted to the brain.

The regeneration of the photoresponse is ensured by the enzyme guanylate cyclase, which restores the initial concentration of cGMP in the cell, leading to the opening of ion channels and the restoration of the initial potential.

The perception of all shades (trichromacy) is ensured by three types of cones with different spectral sensitivity, which is determined by the combination of retinal with a specific type of opsin. Genetic disorders leading to the absence of a specific type of cones (dichromacy, monochromacy) cause color blindness – daltonism.

CONCLUSION

Thus, daltonism is inherited from the mother to the sons. Girls rarely suffer from it, mainly if both parents are affected by daltonism. Girls, being carriers of the gene, only pass it on by inheritance. Only acquired daltonism can be treated. Inherited daltonism cannot be cured. Inherited daltonism remains with a person for life.

Along with the inability to perceive colors, people often experience decreased visual acuity. Methods for correcting and treating daltonism are being developed, but as of today, scientific advancements in this field are still not large-scale enough.

REFERENCES

Dubrov D.I., Grigoriev D.S. Modern studies of intergroup ideologies: assimilationism, ethnic daltonism, multiculturalism, polyculturalism // Social Sciences and Modernity. – 2019. – No. 1. – P. 143.

Klimov A.V., Lifantieva A.A. Color perception disorder: causes, diagnosis, correction // NovalInfo. Ru. – 2018. – Vol. 1. – No. 93. – P. 201-205.

Makarov I.A. The prevalence of hereditary color perception disorders // Ophthalmology. – 2020. – Vol. 17. – No. 3. – P. 414-421.

Ovchinnikov N.D. A method for diagnosing color perception disorders. – 1988.

Savinova A.D. Daltonism and its correction // Current Issues in Medical Science. – 2019. – P. 219-219.

Skvortsova T.A. Daltonism in painting // Issues of Sustainable Development of Society. – 2020. – No. 9. – P. 277-280.

Taranova L.S. Correction of daltonism with color filters // Regional Student Scientific Conference dedicated to the 85th anniversary of SGUGiT. – P. 20.

Taranukha O.A. Color perception disorders (Review) // Experimental and Clinical Medicine. – 2015. – No. 1. – P. 174-177.

Tarasova T.A. Molecular mechanisms of vision. Daltonism // Forcipe. – 2019. – No. Supplement. – P. 252-252.

Faustova Yu.P. et al. Daltonism // Alley of Science. – 2021. – Vol. 1. – No. 5. – P. 165-167.

Khrantsov D.A. et al. Basic concepts of color perception and diagnosis of daltonism // Avicenna. – 2019. – No. 45. – P. 17-24.

Tsitskieva M.M., Plieva A.M. Inheritance of the daltonism trait in the human genotype // Scientific Electronic Journal Meridian. – 2020. – No. 6. – P. 66-68.

Shatukhina M.D. Daltonism // Breakthrough Scientific Research: Problems, Patterns, Perspectives. – 2017. – P. 214-216.