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Color Perception and Environmentally Based Impairments

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Synonyms

Acquired color vision impairment; Acquired color vision loss; Dyschromatopsia

Definition

Decreased discrimination of colors caused by adverse environment, such as long-term occupational exposure to or consumption of drugs, substances, and food containing neurotoxic chemicals.

Color vision early manifests adverse effects of exposure to an environment that contains neurotoxic substances [1, 2]. The acquired color vision impairments, or dyschromatopsias, can be very subtle (subclinical) but also may vary considerably in severity, increasing or decreasing as long as the responsible agent persists, and can become irreversible under long-term exposure and/or agent dose.

There are several scenarios of exposure to hazardous chemical agents in the environment:

i. Long-term occupational exposure to certain substances (e.g., neurotoxic metals, organic solvents, carbon disulfide, etc.)
ii. Self-administered chronic consumption of substances containing neurotoxic chemicals (e.g., alcohol, tobacco)
iii. Side effects from pharmacological treatment of medical conditions (e.g., cardiovascular, antiepileptic, or antituberculosis drugs)
iv. Consumption of food contaminated by neurotoxic elements through the food chain (e.g., mercury)

General Characteristics of Neurotoxin-Induced Color Vision Impairments

Acquired color vision defects, unlike congenital ones, are noticeable to the observer: recently affected subjects name the stimuli as they see them – in contrast to subjects with congenital color vision defects where there is compensatory adaption of their color naming to that of normal trichromats. Acquired dyschromatopsia may not be identical in the two eyes, which requires testing the two eyes separately.

Neurotoxic substances can affect one or more loci in the color vision system. At the pre-receptor level, hazardous chemicals can accelerate yellowing of the crystalline lens which results in an increase in absorption of blue light and hence decreased discrimination of blue colors. In the retina, the main mechanism of color vision loss is selective damage to specific photoreceptor classes, short-wavelength (S-), middle-wavelength (M-), or long-wavelength (L-) cones [► Cone Fundamentals].

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Most vulnerable among these are the S-cones, damage of which is manifested by blue color vision defects. Post-receptoral processing can also be disrupted – at the level of ganglion cells, optic nerve, optic radiation, or visual cortex – causing color vision impairment. Often the damage is nonselective; i.e., patterns of color discrimination loss are not always specific to one of the color subsystems and differ from those in congenital abnormalities.

Classification of Acquired Dyschromatopsias

The wide variation in acquired color vision defects, according to Verriest [3], can be classified in four major types, I, II, and III and a nonspecific defect. The first two types are associated with impaired color discrimination along the red-green axis in perceptual color space [Cross-Ref. Bimler], much like the patterns found in congenital red-green deficiency, i.e., both involve mild to severe confusion of reds and greens [Cross-Ref. Bonnardel]. Type I is protan-like and reveals little or no loss of blue-yellow discrimination; type II is deutan-like and is manifested by concomitant mild loss of discrimination between blues and yellows. Type III, tritan-like, is manifested by mild to moderate blue-green and yellow-violet confusions, with a lesser or absent loss of red-green discrimination.

According to Köllner's rule [1], impairment of blue-yellow discrimination – the range most frequently affected by exposure to hazardous chemicals – suggests toxic retinopathy, i.e., a more external retinal dysfunction; by contrast, a preponderance of red-green loss is associated with pathology in the optic nerve; finally, complex color vision loss, blue-yellow and red-green, suggests a more advanced stage with a damage to both the retina and the neuro-optic pathway. However, numerous exceptions to Köllner's rule instruct one to be cautious about making a clear-cut attribution of blue-yellow loss to damage at a retinal level and of red-green loss to damage at a neural level. Both color systems appear to be selectively susceptible to damage by various types of neurotoxins.

Tests of Color Vision for Assessing Acquired Dyschromatopsias

In epidemiological studies, color arrangement tests are predominantly used. These can be rapidly administered and easily interpreted, and in addition, they allow color vision ability to be quantified graphically [4]. In an arrangement test, the observer is presented with a set of color caps and requested to arrange them in ("rainbow") sequence. The number of erroneous cap transpositions provides a measure of overall color discrimination; the pattern of the transpositions indicates whether the defect is closer to the blue-yellow or red-green axes or with no discernible pattern [4, 5] (Fig. 1).

Three tests, whose caps sample a color circle at even intervals, are traditionally used for the purpose: the Farnsworth-Munsell 100-hue test, the Farnsworth Dichotomous panel D-15 test, and the Lanthony Desaturated Panel D-15d test. The FM 100-hue test consists of 85 caps and takes 20–30 min to perform; it is designed so that error scores will be concentrated in the region of the poorest discrimination. The D-15 test contains a sample of the latter, including a fixed cap and 15 movable ones; it requires ca. 5 min to complete and is designed to diagnose moderate to severe color defects. The Lanthony D-15d test is similar in design and identical to the D-15 test in administration but consists of color samples that are lighter and paler [5]. The D-15d test was designed specifically to capture mild or subclinical color defects in observers who pass the standard D-15 test. The two tests are often used in conjunction, though the more sensitive D-15d is widely

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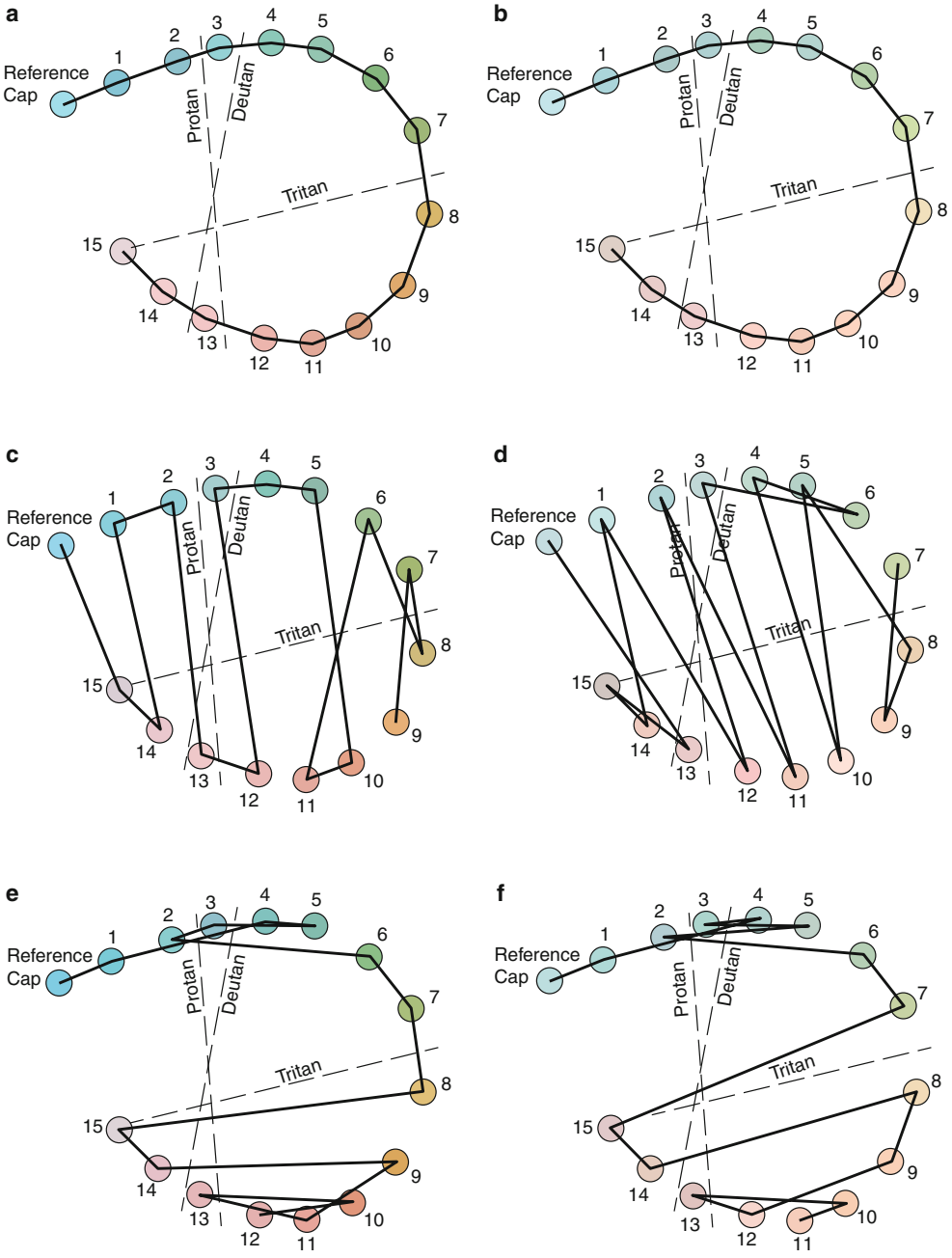

Fig. 1 Scoring sheet for the D-15 (**a**) and the D-15d (**b**); for illustrative purposes, the numbers are accompanied by colors simulating those of the original test caps. At perfect color arrangement of a normal trichromat, lines connect the "reference cap" through 1–15; CCI = 1.0. (**c**–**d**) Lines are drawn between consecutive caps as placed by a protanope, observer with a congenital red-green deficiency; (**c**) CCI = 2.48; (**d**) CCI = 3.11. (**e**, **f**) Mild acquired tritan type of color (*blue-yellow*) discrimination impairment; (**e**) CCI = 2.06; (**f**) CCI = 2.19. Note that the low-saturated stimuli of the D-15d result in a more prominent color confusion (**d**, **f**)

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employed for early detection of mild neurotoxin-induced dyschromatopsias. Outcomes of both tests are reported via a Color Confusion Index (CCI), where 1.0 corresponds to color perfect arrangement; CCI values greater than 1.0 indicate progressive impairment of color discrimination [4, 5].

Occupational Exposure to Neurotoxic Substances

A number of occupations involve exposure to volatile neurotoxic substances (e.g., printers, aircraft maintenance workers, dry cleaners in automotive and metalworking industries, viscose rayon workers, microelectronics assembly workers, gold miners, dentists, etc.). Such substances include organic solvents (toluene, styrene, benzene, perchlorethylene, *n*-hexane, carbon disulfide), solvent mixtures, and metals like mercury (in its elemental or methyl forms). Even when neurotoxic substances are applied within the occupational limits, long-term exposure has been shown to result in mild impairment of color discrimination [6–8].

Using the D-15d test, the degree and pattern of color vision loss was intensively investigated with regard to exposure to organic solvents, in particular, toluene and styrene [2, 6–9], and to mercury [10]. The main finding across these studies is significant increase of the CCI in the occupationally exposed observers (compared to age-matched controls). For example, in a meta-analysis of 15 sample studies of the effects of toluene, styrene, and solvent mixtures [8], the grand mean CCI for the exposed workers was 1.22 ± 0.08, significantly greater than 1.13 ± 0.06 for the controls ($p = 0.003$). Similarly in [10], for workers of fluorescent lamp production exposed to mercury vapor CCI $= 1.14 \pm 0.14$ was significantly greater than 1.04 ± 0.06 for controls ($p = 0.002$). The impairment of color discrimination was shown to be subject to cumulative exposure, i.e., product of duration and current level of exposure [2, 6–9], and may become irreversible even when the hazardous agent is withdrawn [10].

Neurotoxin-induced dyschromatopsias are predominantly of type III, i.e., tritan-like pattern. Less common are types I and II, red-green dyschromatopsias. In comparison, the nonspecific type of dyschromatopsia, which implies difficulty in discriminating colors along both the red-green and blue-yellow axes of color space, is also highly prevalent.

Self-Administered Consumption of Substances Containing Neurotoxic Chemicals

Chronic excessive consumption of alcohol (ethanol) affects color discrimination capacity [5, 6]. When assessed by the D-15d test, the prevalence of dyschromatopsia was shown to increase with alcohol intake. Further, heavy drinkers (with an intake larger than 750 g/week) manifested primarily loss of blue-yellow discrimination, whether or not they were undergoing treatment in a detoxification center. However, 25 % of persons undergoing detoxification revealed dyschromatopsia of the nonspecific type, including red-green loss [11].

Tobacco smoke contains a range of compounds including nicotine, cyanide, and carbon monoxide and, when consumed excessively, can affect color vision [1]. When tested by means of the color arrangement tests, chronic smokers (>20 cigarettes/day, for at least a year) revealed a subtle but statistically significant reduction in sensitivity to red-green differences compared to nonsmokers [12] or showed a diffuse character of color vision disturbance, without a particular dyschromatopsia axis [13].

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Side Effects from Pharmacological Treatments

A number of medications (e.g., cardiovascular, antiepileptic, antituberculosis, and antirheumatism drugs, oral contraceptives, etc.) are known to produce measurable color vision disturbances, some of which affect color vision even at therapeutic levels, most commonly as type III, tritan-like blue-yellow dyschromatopsia [1, 5, 6, 14]. For instance, patients taking antiepileptic drugs develop mild blue-yellow deficiency which may show signs of progression with lasting intake of the drugs. Intake of Viagra was shown to cause transient adverse visual events described as a blue color tinge to vision, accompanied by mild blue-yellow deficiency in about 11–14 % of those taking the medication, the disturbance being reversible after the medication has been discontinued. Treatment by the tuberculostatic ethambutol shows mild blue-yellow deficiency as the earliest sign of the drug's neurotoxicity, but this can also develop as type II, deutan-like red-green, or a nonspecific dyschromatopsia; these defects are transient and reversible after stopping the therapy.

Consumption of Food Contaminated by Neurotoxic Elements

Certain industrial activities, like gold mining or mercury mining, are associated with pollution of mercury which bioaccumulates mainly through the aquatic food chain (seafood and fish). Even at low levels of dietary exposure, using the FM 100-hue test, and the Cambridge Colour Test (► Paramei & Bimler, Deuteranopia), mercury was shown to chronically reduce color discrimination, with the error pattern indicating that both blue-yellow and red-green systems are affected [15].

Cross-References

► Color Categorization and Naming in Inherited Color Vision Deficiencies
► Cone Fundamentals
► Deuteranopia
► Psychological Color Space and Color Terms

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