Color blindness and its illuminations

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In 1974, not long after an antenna was implanted in his skull, the British artist Neil Harbisson became the first person in history to be officially recognized as a cyborg.[1] A cyborg—short for cybernetic organism—is a living thing formed from the integration of organic and mechanical parts.

Before becoming a cyborg, Harbisson struggled as an art student afflicted with achromatopsia, an extremely rare, congenital form of color blindness that allowed him to see only black, white, and shades of gray. After an antenna was implanted in his occipital bone, Harbisson, at age 22, acquired the ability to receive electromagnetic waves from the surrounding air, and transform them using an implanted computer chip into carefully programmed tones. Harbisson is able to hear colors, including those that people with normal vision cannot see.

Harbisson regards his antenna as no less a body part than any bone or tendon. When he was caught up in a public demonstration during which jostling by the police caused damage to his antenna, he felt manhandled, the victim of a particularly grievous form of personal injury.

Harbisson's success as a highly innovative color blind artist is meritorious, not only in its own right, but for the way it illuminates the under-appreciated nature of color

Contemporary artist and cyborg activist Neil Harbisson.

Photo by Rosdiana Ciaravolo/Getty Images

blindness. Harbisson has spearheaded a highly visible campaign to prove that color blindness is no obstacle to achievement in the arts. His efforts are just the latest example of a long struggle to prove that its reputation as a disability, that squelches careers and shrinks the lives of its victims, is a grave oversimplification of a complex reality.

200 years of understanding color blindness

If color blindness is indeed a serious disability, it is curious that its existence was not suspected, or at least not commented upon, until 200 years ago, when the disorder struck John Dalton. In his youth, Dalton, a student of nature, had great difficulty identifying wildflowers, especially reddish ones.[2] He described his defect to the Manchester Literary and Philosophical Society in 1794, at age 28, hypothesizing that his vitreous humor contained an abnormal blue pigment. But with the exception of his youthful botanical blundering (which was duplicated in his brother), Dalton's color blindness seems to have posed very few impediments. Dalton was a genius who regarded his visual defect as a mere curiosity, a bit of impersonal exotica, like a rare plant.

A devout Quaker who had taken a vow of sartorial modesty, Dalton at first declined the honorary Doctorate bestowed on him in 1832 by Oxford University, raising a religious objection to the ostentatious scarlet of the graduation gown. But the hectoring of his conscience was, in the end, forced to yield to the testimony of his vision. The gown was not scarlet. To his eye, it was the color of dried mud.[3]

Dalton's theory of color blindness—that it arises from tinting of the vitreous humor—was met with general skepticism. It was soon replaced by what became known as the Young-Helmholtz theory, which today remains integral to the understanding of human perception. The ability to see color depends on the presence in the retina of cone cells, photoreceptors that contain pigments responding to different frequencies along the spectrum of visible light. The three types of cone cells can be described as red, green, and blue. These color descriptions are based on peak sensitivities that correspond to light of long, middle, and short wavelength, respectively. However, the sensitivities of the cones overlap significantly—red and green are particularly close, their peaks just 30 nanometers apart (a difference largely accounted for by a difference of 2 amino acids).[4] The range of spectral response for each photoreceptor is broad, although somewhat less so for the blue cone.

The science of seeing color

Photoreceptors do not pipeline encoded wavelength information directly to the visual cortex. Every photoreceptor acting alone is, in a sense, color blind, capable of signaling only the rate at which it absorbs photons. Any change in the output of an isolated cone that is induced by varying stimulus wavelength can also be induced by a suitable variation in intensity.[5] This is the principle of univariance, which implies that diverse wavelength-intensity combinations that produce an identical response from all three photoreceptor types will be perceived in the same way. Colored stimuli producing only a two-receptor match will be perceived by a person with normal vision to be of distinguishable hues.

These observations are summarized by indicating that normal human vision is trichromatic. Those afflicted by color blindness are almost always dichromatic, although as the number of functioning cone types is reduced, they will become monochromatic, or like Harbisson, achromatic. The dichromat relies on the contributions of only two photoreceptors—with perhaps an overlapping or weakly functioning third—and matches colors that most observers perceive to be different.

The principle of univariance is the basis of trichromacy, but it does not explain certain critical aspects of human vision: spectral colors as we perceive them form antagonistic pairs. For example, there is no color stimulus that is perceived as a combination of red and green hues. The same is true for blue and yellow. This tendency of spectral colors to arrange themselves along either a red-green or a blue-yellow axis is accounted for by the principle of opponency.[6] The physiologic basis of this principle resides in post-receptor processing. To single out one such process, midget ganglion cells behind the retina subtract the signals relayed by green cones from those of red cones before passing the encoded information to the lateral geniculate nucleus, a relay center in the thalamus. The brain simultaneously receives the information that a stimulus is red and that it is not green. This explains why a defect in the red photoreceptor—the most common variety of color blindness—is expressed as a failure to discriminate red and green.

The similar profiles of the red and green photoreceptors, their adjacent location on the X chromosome, and their nearly identical nucleotide sequences suggest that they arose from a phylogenically recent duplication of a single gene.[5] The duplication made possible the red-green discrimination required to forage small ripened berries amidst the abundant backdrop of leaves.

But there were also negative consequences. The proximity and similarity of the genes for the red and green photoreceptors predispose during meiosis to misalignments and crossovers of the maternal and paternal X chromosomes. These, in turn, produce hybrid photoreceptors that discriminate red and green stimuli poorly.

Because it is usually X-linked, color blindness is predominantly a disorder of males. The gene encoding the short wavelength (blue) photoreceptor, dwells remotely on autosomal chromosome 7 in, relatively speaking, a genomic safe space. It is of much earlier provenance than its longer wavelength cousins by a billion years, and only about 45 percent homologous to them.[7]

Blue-yellow color blindness does occur, but rarely. It can be inherited as an incomplete autosomal dominant, sometimes the result of poisoning or illness.[4]

Dalton was thought to have been a protanope, the word for those with a defect of the first red, long wavelength photoreceptor. This is now known to be in error. The evidence was hidden in plain sight for decades, in a glass jar where Dalton's eyes, removed postmortem at his request, lay entombed, awaiting the technical advances that might unravel their secret.

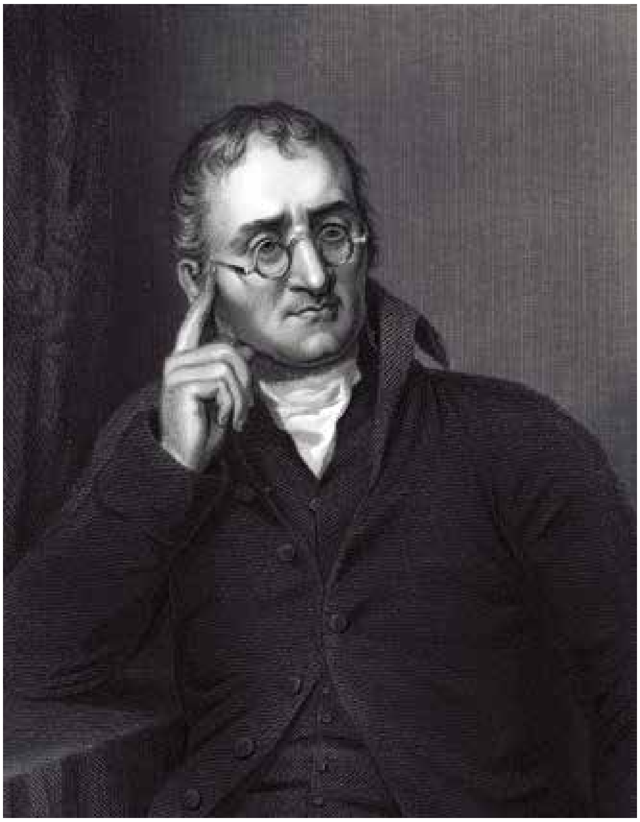
After successfully lobbying the curators of the Manchester Literary and Philosophical Society, in 1995 researchers were given a portion of one of Dalton's eyes for study.[2] At last, the surmising of the 19th century could be supplanted by the verdict of 21st century laboratory science.

DNA isolated from the retina of Dalton's left eye was shown to contain the gene corresponding to the long-wavelength (red), but not the middle-wavelength (green) photoreceptor. Dalton was a deuteranope. His diagnosis had been firmly and correctly made, even if delayed by 223 years.

Daltonism

The ease with which "daltonism," as it came to be known, escaped notice for so many centuries is remarkable, and instructive. Dalton's many-faceted career, which included chemistry, meteorology, and the grammatical use of participles, provides dubiety as to whether color blindness need be a disadvantage in any domain. There is a great deal of discontent with the way color blindness has been categorized, and stigmatized.

Elizabeth Green Musselman argues that Dalton's color blindness was taken by his liberal contemporaries—the inheritors of the Age of Enlightenment—as a metaphor for parochialism and deference to discredited classical teaching.[3] To his peers, Dalton's scientific achievements proved



John Dalton, English chemist, c1860.
Photo by Oxford Science Archive/Print Collector/Getty Images

that his origin in the provinces (he received his earliest education in the village of Pardshaw Hall, County Cumberland) need not be an obstacle to acceptance of scientific universalism, and therefore, that color blindness need not be an obstacle either.

Oliver Sacks (AΩA, Albert Einstein College of Medicine of Yeshiva University, 1970, Faculty), the British neurologist, portrayed by Robin Williams in the movie *Awakenings*, wrote a book describing an island in Micronesia with an unusually large population of achromatopes.[8] In his description of the color blind, he describes their superior sense of "tonalities, shapes, and textures," and their preternatural ability to live in a world of "heightened reality."[8]

These accounts of color blindness are informative, but the political or sentimental motives overarching them cast a long shadow. What kind of professional success might be enjoyed by those whose sense of color is profoundly altered?

Charles Meryon

The French artist Charles Meryon entered life inauspiciously, facing poverty and abandonment.[9] He was born in 1821 to a Parisian ballet dancer and an English physician who returned to his wife in London months before. As a young man, Meryon's ambition was to be a naval officer.

Officer candidates in that era were not subjected to vision tests. Thus, Meryon had a clear path to the naval academy, which he entered in 1837. Upon completing his training, he embarked on several worldwide voyages, discovering during his travels that he had a talent for sketching the landscapes and the indigenous people he encountered. At age 25, Meryon left the navy to dedicate his life to art.[9]

After serving an artistic apprenticeship rendering landscapes strictly in sepia tones, Meryon took up watercolor. Within months, he was writing to his father confessing a "color defect" that drove him to give up painting. He turned to etchings depicting the medieval neighborhoods soon to be demolished by Napoleon III.

Only one of Meryon's oil paintings survives, hanging in the Louvre. "The Ghost Ship" is recognizably the work of a red-green dichromat. Both red and green tints are eschewed, since they were indistinguishable to the artist. In their place is a streaky surfeit of whipped up blues and yellows.

"The Ghost Ship" gives a vivid sense of the world as Meryon must have seen it, less softly hued, more phantasmal, more imbued with turbid and bilious grays. In its idiosyncratic fashion, Meryon's rendering of the blackish sea with its bleached crests of spume, its pallid ship seems to disappear into an expanse of ochre sky—a fitting tribute to his vision of the world.

In 2009, the article "Colour blindness does not preclude

Charles Meryon's, "The Ghost Ship," 1857. Photo: Gérard Blot fame as an artist: celebrated Australian artist Clifton Pugh was a protanope," makes the case that color blindness among male artists is as common as it is in the general population, affecting roughly nine percent of both groups.[10] It also notes that color blindness has been detected in works of art where it demonstrably did not exist. For example, Eugene Carriere, the French artist who came under suspicion for his striking monochromatic style, submitted to a formal examination which found conclusively that his color vision was normal.[10]

Discerning the colors in works of art

If critics cannot discern between the artwork of a dichromat and the work of a stylist adopting a comparable technique, that may be because dichromacy has its own aesthetic legitimacy.

The centerpiece of the article is a case study that supports the authors' claim that color blindness does not "preclude fame as an artist." The Australian artist Clifton Pugh won the Archibald Prize for portraiture on three occasions, and placed 24 paintings in the Australian National Gallery. Pugh underwent formal testing of his color vision once, in 1942, when he reportedly applied to the navy and was rejected. He died in 1990 after waging a lifelong struggle with color; however, a few of his relatives were examined. An older brother and the brother's grandson were shown to be protanopes, suffering defects of the red photoreceptor. The conclusion that Pugh was a protanope, while not airtight, is convincing.

Allowing themselves a bit of free rein, the authors used Photoshop to digitally alter several of Pugh's original paintings. They reproduced the effect of red-green color blindness. The intent was to nullify the color choices Pugh appeared to have made visually, but as a protanope, must have made using non-visual cues. The altered paintings were shown to a group of known protanopes, most of whom could not tell the altered pieces from the original artwork.

Somehow, Pugh was able to circumnavigate, disguise, and/or exploit his abnormal color vision while turning out a succession of masterpieces.

The disadvantages, and advantages, of color blindness

It no longer is sufficient to claim that color blindness is a handicap that might, under favorable conditions, be compensated. Rather, it seems possible that color blindness, in the proper domain, might bestow some advantage. For instance, in a camouflaged setting the color blind possess the gift of enhanced sight. Camouflage relies on the fact that certain stimuli, metamers, are visually indistinguishable despite being composed of light of different wavelengths.[11] Faint objects that fall on the blue-yellow axis can be disguised by placing them against a dappled background of variegated reds and greens. U.S. army combat uniforms were designed on this principle.[12]

A small blue foreground object placed against such a background is swallowed up. To the color blind observer, however, a dappled red-green background appears a uniform gray, and a daub of blue placed against it stands out like a pearl on velvet.

This was used by the Japanese ophthalmologist Shinobu Ishihara who hand painted the color plates used for testing color blindness. These plates rely on the failure of the color blind to discriminate red and green, but a few of them take advantage of the camouflage principle.[13] In 1917, given the task of devising a screening test for Japanese military recruits, Ishihara developed plates to identify camouflage-immune combatants.

Color blind mothers

Some mothers of sons with anomalous trichromacy, the most common form of color blindness, are mildly color blind. The Lyon hypothesis, named for Mary Lyon (AΩA, Emory University, 1965), states that the retinal cells of such mothers contain two X chromosomes, only one of which bears the gene for the anomalous red or green photoreceptor received by her color blind son. It is an anomalous photoreceptor because its spectral sensitivity is shifted. The Lyon hypothesis suggests that one of the X chromosomes is inactivated in a strictly random process. It proposes that the maternal retina contains a mosaic of normal and abnormal cones, potentially giving rise to a *form fruste* of color blindness.

However, another outcome is possible. The process gives rise in the affected mother to four types of cones, three of the usual kind, and one with anomalous properties. But, four distinct functioning cones might provide an entree to color discriminations of a superhuman kind. An analogous process of upward chromatic mobility is known to occur in New World monkeys. Heterozygous females become trichromatic by virtue of possessing an abnormal gene, as well as two normal ones, while the males of the species remain strictly dichromatic.[5]

There are not many tetrachromatic women, because humans will be permanently a trichromat if they are wired for only three photoreceptors, no matter that they have four actual photoreceptors. This is for the same reason that you will be viewing black-and-white television if you own a 1950 Motorola, even if you are watching this year's Tournament of Roses Parade. Tetrachromacy may begin in the retina, but it must be preserved in the post-retinal ganglion cells, as well in as their immediate connections, all the way to the visual cortex. Perhaps this is why the true human tetrachromat has proven so frustratingly elusive. Looking back, trichromacy may be viewed not as the terminus of a journey that the color blind never complete, but as a stopover en route to something better.

Hearing colors

Harbisson has declared his mission as persuading the chromatic world that something better has arrived. A microchip under his skin translates electromagnetic waves into vibrations that travel by bone conduction to his ear. In this way, he can "see" not only the entire visual spectrum, but spectral signals extending into the infrared and ultraviolet. He hears ultraviolet as an F minor chord.

Pointing his antenna at a deeply saturated color has the effect of turning up its volume. Harbisson is also Wi-Fi ready, which means that when his cranium is aligned with an orbiting satellite, he can receive phone calls from cyberspace without using a phone.

Harbisson's claim that he is the first officially recognized cyborg is based on the fact that his antenna is clearly visible in his official passport photo, where it arcs from the back of his skull like an inverted fish hook.

With the aid of his enhanced "vision," Harbisson has produced works that dwarf the accomplishments of many normal-sighted artists. This is exemplified in his sound portraits,[16] where Harbisson first approaches his subject and pauses. With his antenna he scans the face he wishes to paint, generating tones that can be transcribed and performed on an electronic keyboard.

The artistic process also works in reverse. A speech by Martin Luther King, Jr., can be transferred to canvas by imagining the colors that would have produced, for Harbisson, the unmistakable sonorous tones of Dr. King's voice. This process of translation is illustrated in a TED talk Harbisson gave in 2012.[16] King's "I Have A Dream," speech looks to be the product of the avant-garde school of geometric abstractionism.

Not only is there the possibility that there are tetrachromats walking among us—stubbornly sought prodigies eluding the most determined scientific search—our minds must be open to something still more astonishing: galleries of the future may showcase work by perception-bending cyborgs (Harbisson is their primordium) bringing to life new worlds of infinite color dimensions. Such galleries will hum with melodious vibrations of colors.

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