ISSN: 2754-4990

Journal of Ophthalmology Research Reviews & Reports

SCIENTIFIC Research and Community

Research Article

Open Access

Rectifying Color Blindness by Graded Colored Filters

Mitsugu Shimmyo[1]* and Xiao Guan Chen[2]

[1]Associate Professor in Clinical Ophthalmology, Icahn School of Medicine of Mount Sinai Hospital, New York City, USA

[2]Professor of Mathematics, Jilin University, China

***Corresponding author**
Mitsugu Shimmyo, Associate Professor in Clinical Ophthalmology, Icahn School of Medicine of Mount Sinai Hospital USA.
E-mail: mshimmyo@aol.com

Received: May 03, 2022; **Accepted:** May 06, 2022; **Published:** May 11, 2022

Purpose
Significant number of men (5-8%) and a smaller number of women (about 0.4%) suffer from color perception deficiencies. This study introduces new computerized anomaloscope, new graded color filters to rectify color perception deficiencies based on quantitative analyses of the individual color sensitivity spectrum. This also reports mass survey of color blindness in 205,266 Chinese military candidates.

Introduction/Background
As a genetically determined disease, color perception deficiencies in humans had been considered incurable. Whether color blindness was rectifiable had been under discussion for some time, and research had been conducted in this field. As early as 1878, Delboeuf and Spring noted that red color optical filters may be used to help red- green color-blind persons (hereafter dyscromats) to discriminate between different color objects. Many types of optical filtering devices (hereafter filters) were designed to improve the dyscromats' ability to distinguish pseudo-isocolors. For example, King in 1991 introduced light control with color enhancement, with a pair of improved eyeglasses which can aid color discrimination for color blind viewers [1]. Levien in 1993 also devised eyeglasses with spectral color shift with color enhancing sunglasses which attenuate blue and red light [2]. The transmission curves therein are specifically designed to attenuate certain light but not to correct color blindness.

At present, researchers have uses various filters empirically to rectify color blindness. But there have not been devices accurate enough to test color blindness quantitatively. The light sensitivity of human eyes in visible light spectrum (380–720 nm) differs with wavelength (λ). The errors in recognition of color in color blind viewers may occur during certain physiologic process of color signals in retina. The external manifestation of color blindness is the change of sensitivity of eyes in visible light spectrum. Whereas filters in King and Levien could rectify color blindness in certain persons to a certain degree, theoretical basis is insufficient and the result unpredictable. The reason may be that these filters were not designed according to the spectral sensitivity curves of the abnormal color viewers. Moreover, it is not possible to rectify all the varieties of color blindness simply by using single or compound filter. One should devise filters to match the unique spectral sensitivity curves of each particular dyschromat.

In this study, using a newly devised computerized anomaloscope for quick and accurate quantitative analyses of color spectrum sensitivity and creation of new variety of mathematically designed color filters, we tested to see it is possible to rectify individual color sense abnormalities [3-5].

Materials and Method
Review of physiology
Normal humans have at least 3 types of cones in the retina with unique range and peak sensitivities [6,7].

Table 1: Cone cell types, ranges and peak sensitivities of human eye

Cone type	Name	λ range (nm)	Peak λ (nm)	Color
S	Blue(β)	400–500	420–440	Violet
M	Green(γ)	450–630	534–545	green
L	Red (ρ)	500–700	564–580	yellow

Table 2: Colors of the (visible) light spectrum

Color	λ range (nm)	Peak cone sensitivity
(Ultraviolet)	300 – 400	
Violet	400 – 450	S cone
Blue	450 – 490	
Green	490 – 560	M cone
Yellow	560 – 590	L cone
Orange	590 – 635	
Red	635 - 700	

The cones are conventionally labeled according to the wavelengths of the peaks of their spectral sensitivities: short (S), medium (M), and long (L) cone types, also sometimes referred to as blue,

green, and red cones. While the L cones are often referred to as the red receptors, micro-spectrophotometry has shown that their peak sensitivity is in the greenish-yellow region of the spectrum (564-580 nm). Similarly, the S and M cones do not directly correspond to blue and green, although they are often depicted as such (such as in the graph to the right). Peak sensitivity of S cone or blue cone lies in violet zone (420-440 nm). It is important to note that the RGB (Red Green Blue) color model is merely a convenient means for representing color, and is not directly based on the types of cones in the human eye [6,7].

The peak response of human color receptors varies, even among individuals with 'normal' color vision [8]. In non-human species this polymorphic variation is even greater, and it may well be adaptive [9].

The cone sensitivity curves of 3 cones and the total sensitivities all cones may be expressed mathematically below and shown in the graph in Figure 1.

Total normal sensitivity in black curve y (%):

$$y\,(\%) = f\,(\lambda) = R(\lambda) + G(\lambda) + B(\lambda) = e^{-\left(\frac{\lambda-\lambda Rmax}{\alpha R}\right)2} + e^{-\left(\frac{(\lambda-\lambda Gmax}{\alpha G}\right)2} + e^{-\left(\frac{\lambda-\lambda \mathrm{Bmax}}{\alpha B}\right)2}$$

L cone sensitivity in red curve: $R(\lambda) = e^{-\left(\frac{\lambda-\lambda Rmax}{\alpha R}\right)2}$

M cone sensitivity in green curve: $G(\lambda) = e^{-\left(\frac{(\lambda-\lambda Gmax}{\alpha G}\right)2}$

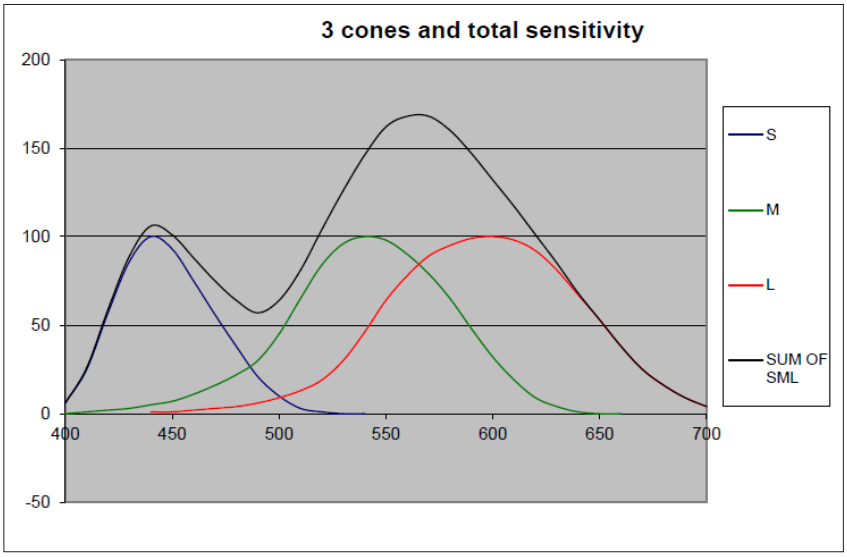
S cone sensitivity in blue: $B(\lambda) = e^{-\left(\frac{\lambda-\lambda \mathrm{Bmax}}{\alpha B}\right)2}$

Total normal sensitivity of 3 curves added:

$$R(\lambda) + G(\lambda) + B(\lambda) = e^{-\left(\frac{\lambda-\lambda Rmax}{\alpha R}\right)2} + e^{-\left(\frac{(\lambda-\lambda Gmax}{\alpha G}\right)2} + e^{-\left(\frac{\lambda-\lambda \mathrm{Bmax}}{\alpha B}\right)2}$$

Figure 1 shows sensitivity curves of L cone (red), M cone (green), S cone (blue) superimposed and total additive sensitivities of all 3 normal cones (black). Note the proximity of M and L cone sensitivities and merging of the range of these 2 cone sensitivities. The proximity of M and L cone sensitivities and merging of the range of these 2 cone sensitivities are noteworthy. Even in normal color vision individuals, the area and peak sensitivities vary. Highest sensitivities are seen in orange to green area in normal individuals. Presence of extra cone was suspected in normal color individuals, especially in some female (Tetrachromat) [6,7].

Figure 1: Sensitivity curves of L cone (red), M cone (green), S cone (blue) superimposed and total additive sensitivities of all 3 normal cones (black).



Total normal sensitivity in black curve y (%):

$$y\,(\%) = f\,(\lambda) = R(\lambda) + G(\lambda) + B(\lambda) = e^{-\left(\frac{\lambda-\lambda Rmax}{\alpha R}\right)2} + e^{-\left(\frac{(\lambda-\lambda Gmax}{\alpha G}\right)2} + e^{-\left(\frac{(\lambda-\lambda Bmax}{\alpha B}\right)2}$$

where, L cone sensitivity in red curve: $R(\lambda) = e^{-\left(\frac{\lambda-\lambda Rmax}{\alpha R}\right)2}$

M cone sensitivity in green curve: $G(\lambda) = e^{-(\frac{(\lambda-\lambda Gmax}{\alpha G})2}$

S cone sensitivity in blue: $B(\lambda) = e^{-(\frac{(\lambda-\lambda Bmax}{\alpha B})2}$

Protanomaly (1% of males, 0.01% of females): L cone deficiency. Protanomalous individuals are less sensitive to red light than normal. This means that they are less able to discriminate colors, and they do not see mixed lights as having the same colors as normal observers. They also suffer from a darkening of the red end of the spectrum.

This causes reds to reduce in intensity to the point where they can be mistaken for black. It is interesting to note that the perception by dyscromats were described by rare chimeric individuals with normal perception in one eye and abnormal in the other.

Protanomaly is a fairly rare form of color blindness, making up about 1% of the male population. Protanomaly is carried on the X chromosome. (X- OPN1LW).

Figure 2 shows sensitivity curves of Protanomalies or L cone deficiencies. Normal total sensitivity (black), mild L cone defect, 75% L cone function (green), moderate L cone defect, 50% L cone function (red) and severe defect 25% L cone function (yellow) and theoretical Protan or 0% L cone function (blue). Note that some sensitivities are present across the spectrum of protanomalies, although in lesser sensitivity except in far long wave range. Theoretical protan who has no L cone activity cannot be corrected to see spectra above 600 nm.

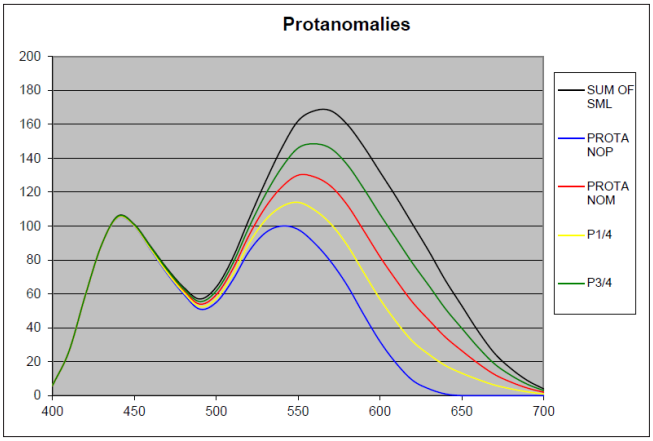


Figure 3 shows sensitivity curves of Deuteranomalies or M cone deficiencies. Normal total sensitivity (black), mild M cone defect, 75% M cone function (green), moderate M cone defect, 50% M cone function (red) and severe M cone defect 25% M cone function (yellow) and theoretical Deutan or 0% M cone function (blue). Note that some sensitivities are present across the spectrum of deuteranomalies, although in lesser sensitivity in middle wave range.

Deuteranomaly, M cone defect, is by far the most common type of color vision deficiency, mildly affecting red-green hue discrimination in 6% of males and 0.36% of females. It is hereditary and sex-linked; the gene is on X chromosome (X-OPN1MW).

Having a mutated form of the medium-wavelength (M cone) green pigment. The medium-wavelength pigment is shifted towards the red end of the spectrum resulting in a reduction in sensitivity to the green area of the spectrum. Unlike protanomaly the intensity of colors is unchanged.

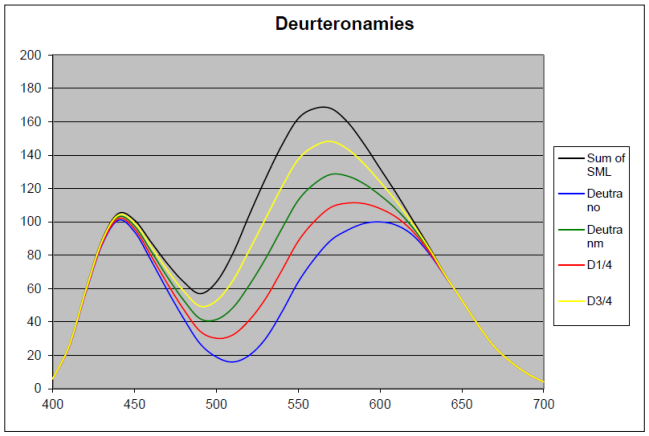
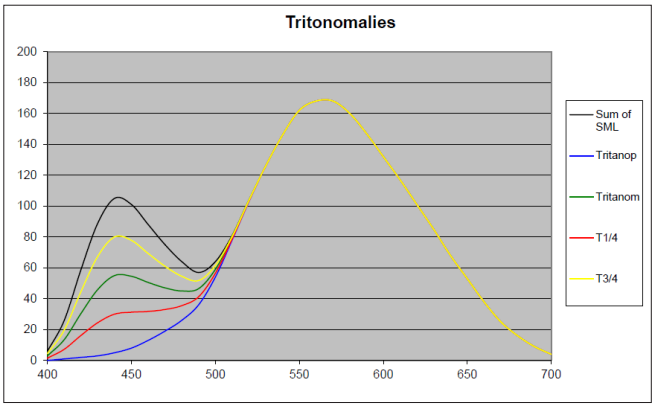


Figure 4 shows sensitivity curves of Tritanomalies, S cone deficiencies. Normal total sensitivity (black), mild S cone defect, 75% S cone function (green), moderate S cone defect, 50% S cone function (red) and severe S cone defect 25% S cone function (yellow) and theoretical Tritan or 0% S cone function (blue).

Tritanomaly is a rare, equally rare for males and females [0.01% for both], hereditary color vision deficiency affecting blue-yellow hue discrimination. Having a mutated form of S cone gene in chromosome 7, the short-wavelength pigment is shifted towards the green area of the spectrum. This is the rarest form of anomalous trichromacy. Note that some sensitivities are present across the spectrum of Tritanomalies, although in lesser sensitivity in short wave range. Even the theoretical Deutan with no M cone activity may be helped to see middle wave area due to overlapping of sensitivities of L and S cones covering this area of spectrum. Note that some sensitivities are present across the spectrum of Tritanomalies, although in lesser sensitivity in short wave range. Even the theoretical Deutan with no M cone activity may be helped to see middle wave area due to overlapping of sensitivities of L and S cones covering this area of spectrum.



Description of new diagnostic devise

A new computerized anomaloscope mixes spectral red and green lights in variable proportions, for comparison with a fixed spectral yellow.

The anomaloscope which was originally described in 1930's uses a reference color and testing color which mixes known quantities

of elementary colors. But this devise requires lengthy testing time and strict testing conditions. The result of the test does not provide sufficient information for designing rectifying filters.

Rational

A newly designed computerized anomaloscope assesses color perception deficiencies, developed and fitted with color corrective lenses, refined with computer technology. This idea of using color rectifying filter is based on the discovery that many dyschromatic persons possess normal functions of at least 2 cones and an attenuated function of the third to varying degrees. By suppressing the sensitivities of the spectrum of color perceived by two strong cones by filtering off the intensity of such spectra of color, the resultant mixture of color perceived by the individual will be balanced and approximate the mixture of the light perceived by a normal individual. By measuring the color sensitivity of the individual precisely by this device, spectral characteristic of the filters is determined to suppress wavelength of stronger spectra by the computers based on mathematical simulation to reverse the errors by the use of colored filters to reduce the intensity of certain light, a normalized mixture of light spectrum can be created. One drawback of such a filter is darkening of the images as certain spectra of light were filtered out. Further detail of the design of the device is described in the patent Granted [3-5].

Design of color rectifying filters

A red colored optical filter allows red light to pass through, but blocks other colors to pass through. Red-green color blindness was found to be helped in distinguishing missed colors. Yellow colored filter blocks blue light and blue filter blocks red light. Based on the color spectrum of 3 cones and the theoretical mixtures of the total sensitivities of the individuals with color anomalies with different degrees, 4 types of the optical filters with 8 different shades of density to block and allow certain spectra of light to balance the total spectrum were created. It was bases on the discovery that many dyschromatic individual retain broad spectrum of varying sensitivities. When sensitivities of the spectrum of highest sensitivities were attenuated by filters, the discrimination of the hues missed became possible. Mathematical models were developed to create mixture of colors for the optimum balance of sensitivities to distinguish hue were developed based on the following mathematical conditions [3-5].

For achromatopsia type A, B, or C, the correction curves should meet the following equation:

$$F(\lambda) = Ae^{-(\frac{\lambda-600}{\alpha})2} + Be^{-(\frac{\lambda-535}{\beta})2} + Ce^{-(\frac{\lambda-440}{\gamma})2},$$

Wherein the values of A, B or C is 1/63, 2/63, 3/63 …….,63/63, and A + B + C = 1, λ being wavelength in nm, α, β, γ being weighted indexes. Detailed mathematical rationales and derivations are previously published [3]. By suppressing the sensitivities of the spectrum of color perceived by two strong cones by filtering off the intensity of such spectra of color, the resultant mixture of color perceived by the individual will be balanced and approximate the mixture of the light perceived by normal individuals.

Severe color defect requires denser filter and this makes the lenses very dark and cosmetically not appropriate to the observer's view. To counter such a shortcoming of darkness, further new idea was developed to use the filter in one eye and keep unfiltered clear lens in the other eye to maintain total brightness. However, this causes a cosmetic problem as the individual will be wearing spectacles with one dark lens and a clear lens in the other eye. To counter such a problem, another idea was developed to modify the front surface of the spectacles by mirror finish with vacuum chrome plating evaporation process so that both right and left sides of the spectacles will appear identical to the outside observers. These lenses were tested in randomly selected 300 achromatic individuals and performances of these lenses were evaluated with 24page color vision testing book. The design of the rectifying filter and the rationale is described in mathematical terms in the patent granted [3-5].

Preliminary Human Study

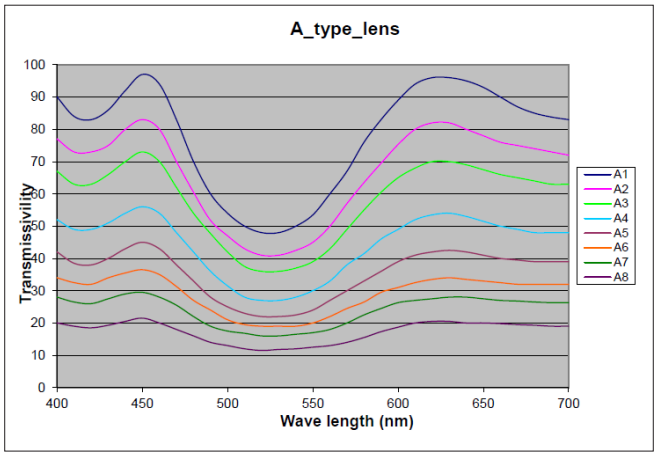
As a preliminary step, in December, 1991, arbitrarily selected 300 dyschromatic individuals were tested and fitted with new color corrective lenses. Four types of spectral specificity type lenses used and color wave length penetration rate of lenses in percentage. 300 patients tested were corrected by these 4 types of lenses with spectral penetration rate defined above. 85% of the subjects were corrected with Type A and Type B lenses.

Table 3: Type Transmissibility of A lenses for Deuteranomaly correction

Transmissibility at wavelength in percentage

Lens grade	A1	A2	A3	A4	A5	A6	A7	A8
λ = 440	80	75	70	65	60	55	50	40
λ = 535	40	35	30	25	20	15	9	4
λ = 600	95	90	85	80	75	70	65	60

Figure 5: Transmissibility of Type A lenses

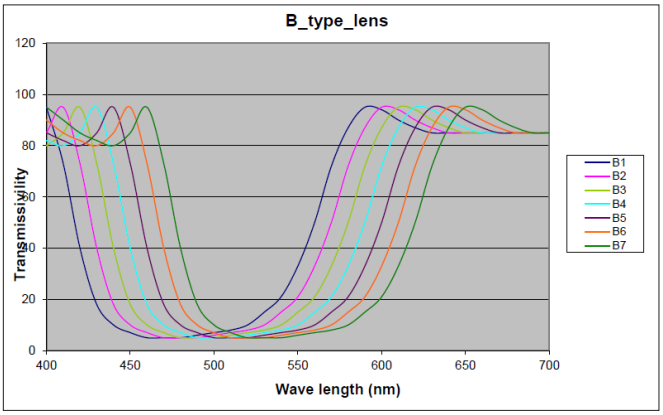


This type is used to correct deuteranomalies by suppressing strong sensitivity spectra in mid length waves, allowing long wave area to maintain the strength.

Table 4: Transmissibility of Type B lenses for Protanomaly correction. Transmissivity in percentage at wavelength

Lens grade	B1	B2	B3	B4	B5	B6	B7	B8
λ = 440	40	35	30	25	20	15	10	3
λ = 535	5	5	5	5	5	5	5	3
λ = 600	60	65	70	75	80	85	90	95

Figure 6: Transmissibility of Type B lens



This type is used to rectify protanomalous by suppressing middle wave spectra and allowing long and short-wave area to maintain sensitivities by progressively shifting the area of peak sensitivity.

Table 5 Transmissibility of Type C lenses.

Type C lenses for AR anomaly type: transmissivity at wave length in percentage. These were designed to help mild deuteranomalous but were found to be less effective than A type in the preliminary trials. As the use this type of lenses are limited, the graph is not shown.

Table 5: Transmissibility of Type C lenses for mild Deuteranomaly correction

Lens grade	C1	C2	C3	C4	C5	C6	C7	C8
λ = 440	85	80	75	70	65	60	55	50
λ = 535	70	67	64	61	58	56	53	50
λ = 600	90	85	80	75	70	65	60	53

Table 6 Transmissibility of Type D lenses for AB anomaly type.

Type D lens for AB anomaly type: transmissivity at wave length in percentage. These were designed to help tritanomalies, but the number of tritanomalies were found to be very small among the population studied. As the use this type is limited, the table of transmissivity in percentage in 3 colors are shown in a table, but the graph is not shown.

Table 6: Transmissibility of Type D lenses for Tritanomaly correction

Lens grade	D1	D2	D3	D4	D5	D6	D7	D8
λ = 440	32	27	22	17	12	7	2	0
λ = 535	68	64	60	56	52	48	44	40
λ = 600	90	85	80	76	72	68	64	60

Final large-scale study

Later between January and May 2005, out of 205,266 candidates for military services were tested for color perception in Shenyang district, in the Peoples republic of China, and 10,266 individuals were identified and further classified for type and degree of color function deficiencies. 205,266 candidates for military services were first tested using screening color plates similar to Ishihara plates. Those who failed the screening tests were further tested with the new computerized anomaloscope and color sensitivity spectrum were determined, and color rectifying filters were selected. Being under the supervision of the Chinese military, Investigational Review Board approval was not obtained. The study involved testing color vision and no invasive therapeutic contacts with the persons tested were made. There was no violation of tenets of Helsinki.

Result

Table 2 Number of dichromats and color defect types among 205,266 Chinese military service candidates. Shenyang, Jan to May, 2005.

Table 7: Classification of dyschromatopsias

Type of dyscromatopsia	Number	Percentage	deficient	strong	weak
Protanomalies	3,512	34.20%	L cone	G	BR
Deuteranomalies	6,570	64.01%	M cone	G/Y	BRB
Tritanomalies	180	1.75%	S cone	R	BGB
Protan	2	0.02%	No L cone	B	BRG
Deutan	0	0%	No M cone	Y	BG
Tritan	0	0%	No S cone	GR	BB
Monochromacy	2	0.02%	all	none	
Total number	10,266	100%			

As in the table 34.20% had Protanomalies of different degrees. Theoretically pure Protan with no L cone activity was very rare 0.02%. 64.01% had Deuteranomalies of varying degrees with no Deutan with theoretically zero M cone activity. There was 1.75% of Tritanomalies of varying degrees and no Tritan with theoretically zero S cone activity. Although the incidence of Protan and Deutan are cited in literature to be 1 % each, our large scale anomaloscopic analyses show very small incidence of theoretical zero activity of each cone cells [6-9].

Discussion

Perception of color, namely hue discrimination involves complex mechanisms, from photochemical reaction in retinal cone cells, electric signal transmission and modulation in the retinal ganglion cells, where input from 3 types of photoreceptors are integrated, then electrical impulse transmission through neurofibers to lateral geniculate body, where input from ganglion cells are further merged, and messages end at the occipital lobes, where final cognitive decision to discriminate color, shape and meaning of figures are made. When testing hue discrimination, one need to take into consideration many distractive conditions, such as surrounding lighting condition, order of testing, Purkinje effect, complimentary color effects, etc. Even emotional state and life time experience can influence color perception. Never the less, the most significant differences in hue discrimination between normal and color blind lie at the level of genetic composition of cone cells in the retina.

The peak response of human color receptors varies, even among individuals with 'normal' color vision [8]. In non-human species this polymorphic variation is even greater, and it may well be adaptive [8,9].

As a conventional terminology, as an individual possesses 3 functioning cones, one is deemed trichromatic if all 3 cones are fully functional. If the sensitivity of one of the cones are reduced, one is anomalously trichromatic. When one of the cones is totally absent, one is dichromatic

We observed Protonamalies, Deuteranomalies and Tritanomalies of varying degrees [7]. The proportions of these corresponds to the distributions in literature. One variation is that rarity of Protan, Deutan and Tritan with theoretically no L, M or S cone activities. This may be due to a new find by quantitative analyses or possibly due to integration and modulation of input from individual cones at the level of ganglion cells, lateral geniculate body or visual cortex.

Occupations

Color blindness may make it difficult or impossible for a person to engage in certain occupations. Persons with color blindness may be legally or practically barred from occupations in which color perception is an essential part of the job (e.g., mixing paint colors), or in which color perception is important for safety (e.g., operating vehicles in response to color-coded signals). This occupational safety principle originates from the Lagerlunda train crash of 1875 in Sweden.

Driving motor vehicles

Some countries (for example, Bulgaria, Romania and Turkey) have refused to grant individuals with color blindness driving licenses. In Romania, there is an ongoing campaign to remove the legal restrictions that prohibit colorblind citizens from getting drivers' licenses. The usual justification for such restrictions is that drivers of motor vehicles must be able to recognize color-coded signals, such as traffic lights or warning lights.

Genetics

L cone gene: **OPN1LW** lies in X chromosome and is highly polymorphic (a recent study by Verrelli and Tishkoff found 85 variants in a sample of 236 men) [9]. Anomalies in this gene causes Protanomalies with the incidence of 6% in males and 0.36% in females in Caucasians. In their study, they suspect up to twenty percent of women may have an extra type of color receptor [11].

M cone gene: **OPN1MW** lies in X chromosome, variations are rare. Anomaly of this gene causes Deuteranomalies [10].

The opsins (photopigments) present in the L and M cones are encoded on the X chromosome; defective encoding of these leads to the two most common forms of color blindness. The OPN1LW gene, which codes for the opsin present in the L cones, is highly polymorphic (a recent study by Verrelli and Tishkoff found 85 variants in a sample of 236 men [9]. Some women may have an extra type of color receptor because they have different alleles for the gene for the L opsin on each X chromosome [10,11] .X chromosome inactivation means that only one opsin is expressed in each cone cell, and some women may therefore show a degree of tetrachromatic color vision [12]. Variations in OPN1MW, which codes the opsin expressed in M cones, appear to be rare, and the observed variants have no effect on spectral sensitivity.

Inheritance patterns of protanomalies and deuteranomalies follow classical mendelian sex linked recessive form. Theoretically a female can inherit a defective L cone from one parent and a defective M cone from another parent. Theoretically, half of the female products of a colorblind father and carrier mother become color blind. When a female child inherits OPN1LW and OPN1MW genes, the result becomes complex and unpredictable. As normal allele can suppress the abnormal allele, one may turn out to be normal. I have personally encountered such an example. As a personal communication, very complex color sensitivity patterns in a female colorblind subject have been observed.

Tritanomaly is rare, equally rare for males and females (0.01% for both male and female), hereditary color vision deficiency affecting blue-yellow hue discrimination. S cone gene is on autosomal chromosome 7. The OMIM gene code for this mutation is 304000.

Jeremy H. Nathans (with the Howard Hughes Medical Institute) proved that the gene coding for the blue receptor lies on chromosome 7, which is shared equally by males and females. Therefore, it is not sex-linked. This gene does not have any neighbor whose DNA sequence is similar. Blue color blindness is caused by a simple mutation in this gene [11].

Having a mutated form of the (S-cone) blue pigment. The short-wavelength pigment is shifted towards the green area of the spectrum. This is the rarest form of anomalous trichromacy. Comparing the sensitivity curves of normal, anomalous trichromats, and dichromats, there is still wide range of sensitivities across the spectrum.

Computerized color spectrum perception analyses of patients and customization of color spectrum correcting lenses enabled 300 patients using 4 types of corrective lenses and 32 combinations of specified color penetration rates. These were performed in December, 1991, for US patent application No 5,369,453. These new lenses were found to be very effective in color perception deficiencies. However, monochromacy which is extremely rare products of very close consanguine unions such as siblings, cannot be corrected with the filters described here as there is no room in the spectrum to modulate sensitivities in these eyes.

Old world apes are mostly trichromatic, but new world apes are mostly dichromatic. Therefore, new world apes are used to study color blindness and its treatment. A recent attempt to modify dichromacy to trichromacy has been reported.

In September 2009, the journal Nature reported that researchers at the University of Washington and University of Florida were able to give trichromatic vision to squirrel monkeys, which normally have only dichromatic vision, using gene therapy [13].

The strength of this study includes innovation of new diagnostic technique, innovation of rectifying optical filters, technical innovation to manufacturing process of vacuum coating the lens surface with a mixture of 3 dyes matching patient's spectral characteristics. Another strength of this study is the sheer size of the studying over 200,000 persons.

The weakness of this study is that female subjects were not included, as this study was performed on the military service candidates who were only males. Qualitative diagnosis and fitting the rectifying lenses were performed, but post diagnostic evaluation of the degree of color discrimination performance in real life or degree of satisfaction were not included [14].

Conclusion

As a genetically determined disease, color blindness in humans remains incurable at gene level, short of gene modulation in the future. Computerized color spectrum perception analyses of patients and customization of color spectrum correcting lenses described here enabled color sense anomaly persons to recognize colors as tested by pseudochromatic plates. Their perceptions of color were modified to approximate that of normal trichromatic persons. These lenses are produced in China and available over 10 years in China and Japan with many satisfied individuals [3].

Financial Disclosures
Mitsugu Shimmy, MD: No financial interest Xiao Guan Chen, PhD: patent holder
No part of this manuscript was published except for the patent document.

References

1. King WJ (1991) Light control with color enhancement. U.S. Patent No. 5,054,902, Oct 8, 1991. (Application no 4,300,819)
2. Levien RL (1993) Eyeglasses with spectral color shift. U.S. Patent No 5,218,386 (5,149,183).
3. Chen X, Lu Z (1994) Method and eyeglasses for rectifying color blindness. U.S. Patent No 5,369,453.
4. Chen X, Lu Z (1988) A topological model of color perception simulation on the echanism of color blindness adjustment. Proceedings of the international conference of omathematics.
5. Chen X, Lu Z (1998) Engineering by computer diagnosis and adjustment of color perception abnormality. World congress on medical physics biomechanics. USA.
6. Wyszecki Günther, Stiles WS (1982) Color Science: Concepts and Methods, Quantitative Data and Formulae (2nd ed.). New York: Wiley Series in Pure and Applied Optics.
7. Hunt RWG (2004) The Reproduction of Colour (6th ed) Chichester UK: Wiley– IS&T Series in Imaging Science and Technology 11-12.
8. Neitz J, Jacobs GH (1986) Polymorphism of the long wavelength cone in normal human color vision. Nature 323: 623-625.
9. Neitz J, Jacobs GH (1990) Polymorphism in normal human color vision and its mechanism. Vision Res 30: 621-636.
10. Verrelli BC, Tishkoff SA (2004) "Signatures of selection and gene conversion associated with human color vision variation". Am. J. Hum. Genet 75: 363-375.
11. http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pmcentrez&artid=1182016.
12. Jeremy H Nathans (2010) (with the Howard Hughes Medical Institute "Seeing, Hearing and Smelling the World, a Report for the Howard Hughes Medical Institute". Howard Hughes Medical Institute. http://www.hhmi.org/senses/b130.html.
13. Roth Mark (2006) "Some women may see 100 million colors, thanks to their genes" Post-Gazette.com
14. Dolgin E (2009) "Colour blindness corrected by gene therapy". Nature News. doi:10.1038/news.2009.921.