

Quantification of color vision with cone contrast sensitivity

JEFF RABIN

College of Optometry, Pacific University, and U.S. Army Aeromedical Research Laboratory, Fort Rucker

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Abstract

Human color vision is based fundamentally on three separate cone photopigments. Hereditary color deficiency, which affects up to 10% of males, results from an absorption shift or lack of L or M cone photoreceptors. While hereditary S cone deficiency is rare, decreased S cone sensitivity occurs early in eye disease, underscoring the importance of quantifying S cone function. Our purpose is to describe a novel approach for quantifying human color vision based on the photopigments of normal color vision. Colored letters, visible to a single cone type, are presented in graded steps of cone contrast to determine the threshold for letter recognition. This approach quantifies normal color vision, indicates type and severity of hereditary deficiency, and reveals sensitivity decrements in various diseases.

Keywords: Contrast sensitivity, Cone contrast, Color vision, Visual acuity

Introduction

The ability to discriminate the various colors that surround us is conferred fundamentally by three separate retinal cone photopigments. Indeed, hereditary color deficiency, which affects 8–10% of males and 1 in 200 females, is due to a shift in peak absorption or lack of long-wavelength-sensitive (L) or middle-wavelength-sensitive (M) cone photopigment (Sakmar, 2003). Color-deficient individuals have difficulty judging differences in color, and confuse colors which appear quite distinct to color-normal individuals. Hereditary short-wavelength-sensitive (S) cone deficiency is rare (0.001–0.007%), but S cone sensitivity loss can occur as an early sign of ocular or systemic disease (Adams, 1982; Greenstein et al., 1989), making S cone clinical tests needed.

There are numerous color-vision tests, which detect the *presence* of color anomalies, but relatively few tests indicate *type* (L, M, or S cone) or *severity* of color deficiency, and few reveal S cone sensitivity loss. Despite the cone basis for both normal and abnormal color vision, human color-vision research is often directed at postreceptoral processing.

We describe further evaluation of a novel approach based on the photopigments of normal color vision (Rabin, 1996). Colored letters visible to a single cone type (L, M, or S) are presented in graded steps of cone contrast to determine the threshold for letter recognition (cone contrast sensitivity; cone CS). Our extended results, which include both hereditary and acquired color deficiency, indicate that cone CS provides a quantitative measure of normal color vision, reveals type and severity of hereditary color

deficiency, and discloses decrements in chromatic sensitivity early in ocular, systemic, and neurological disease.

Materials and methods

A high-resolution video card (24 bits per pixel; 16.7 million colors) was used in a standard computer to generate cone-specific colored letter charts on a color monitor. The red, green, and blue phosphors of the monitor were adjusted in relative intensity to stimulate single cone types. This was achieved through comprehensive measurement of the monitor luminance and CIE (x, y) chromaticity, and transforming these values to cone excitations (Cole & Hine, 1992) based on the psychophysically derived cone spectral sensitivities (Smith & Pokorny, 1975). Cone contrast was then computed from the amount of excitation in a colored letter relative to its gray background.

A separate series of letters was generated for each cone type (L, M, and S); with each series comprised of ten rows of letters (5 letters/row; 20/320 letter size, or 1.9 cycles/deg) which gradually decreased in visibility (contrast), by row, in 0.1 log unit steps. Threshold was determined for each cone series as the number of letters read correctly (0.02 log units per letter). Fig. 1 shows the systematic decrease in cone contrast with row number on L, M, and S cone tests, as well as cone excitations in CIE color space (inset). The S cone function is elevated relative to L and M because there are far fewer S cones, making it necessary to use higher contrast to achieve letter recognition. The horizontal line is the mean contrast to the two cone types not stimulated systematically on each series. These contrasts are below threshold for letter recognition, making each chart visible to a single cone type.

Each series was administered to 30 subjects with normal color vision, 28 subjects with hereditary color deficiency, and to various patients with ocular, systemic, and neurological disease. In accord

Address correspondence and reprint requests to: Jeff Rabin, Pacific University College of Optometry, 2043 College Way, Forest Grove, OR 97116, USA. E-mail: rabin@pacificu.edu

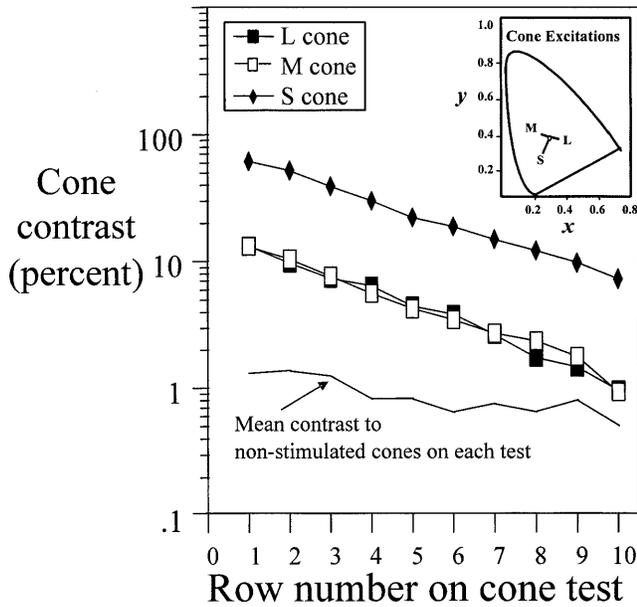


Fig. 1. The decrease in cone contrast with row number on the L, M, and S cone letter series. The inset shows cone excitations for each series in CIE color space.

with the Declaration of Helsinki, all subjects gave their informed consent after protocol approval by the Institutional Review Board.

Results

Figure 2 shows results for cone CS in individuals with normal color vision, and in those with hereditary color deficiency. The boxes represent mean values and the error bars show 99% confidence intervals for color normal subjects (± 3 SDs from the mean). For comparison, individual results are shown for L cone (protan) and M cone (deutan) color-deficient subjects (filled and unfilled

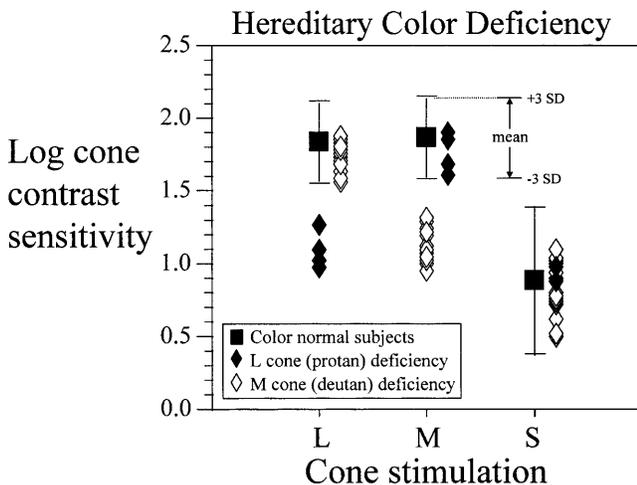


Fig. 2. Log cone CS is plotted against L, M, and S cone stimulation. Mean and 99% confidence interval (± 3 SDs) is shown for normal observers, with individual results shown for color-deficient subjects.

diamonds). L cone deficient subjects show significantly decreased L cone CS, but normal M and S cone CS, making the specific diagnosis of protan deficiency unequivocal. M cone (deutan) deficient subjects show the reverse effect, with significantly reduced performance limited to the M cone test. The *type* of deficiency indicated by cone CS was in complete agreement with Nagel I anomaloscope findings. In all subjects with decreased M cone CS, the midpoint of the anomaloscopic matching range was shifted toward the green primary (consistent with deutan deficiency), and all subjects with decreased L cone CS showed a midpoint shift toward the red primary (consistent with protan deficiency; mean shift for all anomalous trichromats was 11 SDs from midpoint for color normals). The *severity* of hereditary color deficiency indicated by cone CS was correlated with performance on several tests of color vision, spanning a range of tasks. Log cone CS was inversely correlated with log anomaloscope matching range ($r = 0.7, P < 0.001$), FM 100 hue total error score ($r = 0.65, P < 0.001$), D-15 error score, both normal ($r = 0.37, P < 0.01$) and defaturated ($r = 0.36, P < 0.05$), and with the number of errors on Farnsworth Lantern ($r = 0.65, P < 0.001$) and PIP testing ($r = 0.71, P < 0.001$).

Fig. 3 shows decrements in cone CS observed in patients ($n = 26$) with various ocular conditions and diseases. Boxes and error bars represent the mean and 99% confidence interval (± 3 SDs) for normal observers, and individual results are shown for patients with anomalous conditions spanning all levels of the visual system. Retinal conditions include age-related and traumatic macular disease, diabetic retinopathy, and central serous retinopathy; optic nerve conditions include glaucoma, nerve hypoplasia, and neuropathy; and visual pathway disorders include retrobulbar neuritis, multiple sclerosis, and amblyopia. Regardless of the site of disease, cone CS is decreased relative to normal values (mean decrease across all subjects and cone tests = 3.87 SDs below the normal mean). The reduction in cone CS was observed despite normal findings on pseudoisochromatic plate testing, and with minimal decrease in vision (mean visual acuity = 20/25), exemplifying the sensitivity of this approach. While more extensive color-vision testing would be valuable diagnostically and for comparison to cone CS, this was not feasible due to time and equip-

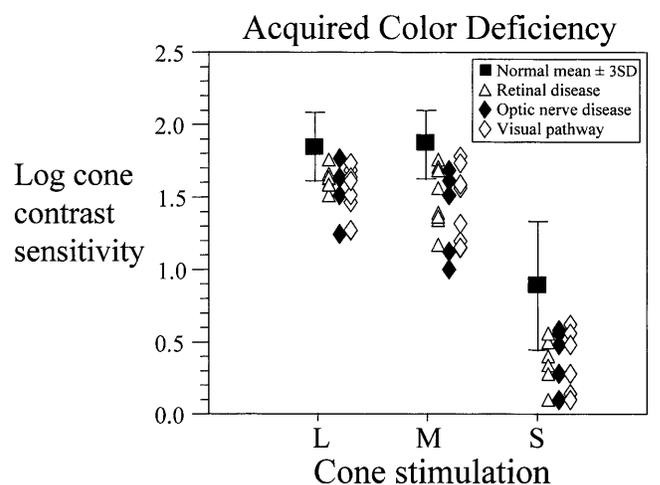


Fig. 3. Log cone CS is plotted against cone stimulation for various ocular diseases which span levels of the visual system. See text for further details.

ment constraints of the clinical setting. Two-way repeated-measures ANOVA of Z-scores (SDs below normal mean) revealed no difference in cone CS across disease categories (retina, optic nerve and pathway; $F = 0.65$, $P > 0.5$), but a significant difference between cone tests (L, M, and S; $F = 13.04$, $P < 0.01$), with M cone CS showing greater sensitivity (average CS decrease across all patients = 4.97 SDs below normal mean) as compared to L and M cone CS (3.37 and 3.26 SDs, respectively). It is conceivable the relative enhancement of M cone CS for detection of acquired color deficiency may relate to (1) lower variability of M cone CS (SD = 0.08) as compared to S cone CS (SD = 0.15); and (2) lesser redundancy in the M cone retinal array compared to the L cone array (since L cones typically outnumber M cones; Carroll et al., 2003), making early or subtle insults to vision more detectable with M cone CS. Further research is needed to explore this possibility.

Discussion

Cone CS provides a quantitative index of normal color vision, indicates type and severity of hereditary color deficiency, and reveals decrements in chromatic sensitivity which can occur as an early sign of ocular, systemic, and neurological disease. Both software-based and hard-copy versions of cone CS, suitable for adult, pediatric, and cross-cultural applications, currently are under development.

The present approach of using cone-isolating stimuli is similar to the original Hardy, Rand, and Rittler (H-R-R) pseudoisochromatic plate test (now in reproduction), and is quite comparable to an effective computer-based color-vision test (Mollon & Reffin, 1989; Regan et al., 1994). Whereas the H-R-R and computer-based test use luminance and spatial noise to minimize detection by nonchromatic mechanisms, cone CS utilizes systematic steps in cone contrast, coupled with clinically expedient *by-letter* scoring (Bailey et al., 1991) to quantify color vision.

Human color-vision research often emphasizes the application of isoluminant stimuli to evaluate luminance and chromatic post-

receptoral processing. Cone CS represents a more fundamental approach of stimulating at the receptoral level. Cone CS is comparable to classical studies of selective chromatic adaptation, in which a colored stimulus is superimposed on an intense background, typically of the opposite color, to isolate chromatic mechanisms. However, in the cone CS approach, a constant, achromatic background is used for all stimuli, thereby insuring a constant and normal state of visual adaptation.

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