Vision Research 48 (2008) 2586–2592

Contents lists available at ScienceDirect

Vision Research

journal homepage: www.elsevier.com/locate/visres

Rod contributions to color perception: Linear with rod contrast

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ARTICLE INFO

Article history:
Received 25 October 2007
Received in revised form 25 April 2008

Keywords:
Rods
Cones
Rod percept
Color vision
Rod contrast

ABSTRACT

At mesopic light levels, an incremental change in rod activation causes changes in color appearance. In this study, we investigated how rod mediated changes in color perception varied as a function of the magnitude of the rod contrast. Rod-mediated changes in color appearance were assessed by matching them with cone-mediated color changes. A two-channel four-primary colorimeter allowed independent control of the rods and each of the L-, M- and S-cone photoreceptor types. At all light levels, rod contributions to inferred PC, KC and MC pathway mediated vision were linearly related to the rod incremental contrast. This linear relationship could be described by a model based on primate ganglion cell responses with the assumption that rod signals were conveyed via rod–cone gap junctions at mesopic light levels.

1. Introduction

At mesopic light levels, both rods and cones contribute to vision. Anatomical and single-unit electrophysiological studies of mammalian retina have shown that rods and cones share the same neural pathways from ganglion cells to the brain (literature reviewed by Sun, Pokorny, & Smith, 2001b). Two primary pathways convey rod signals to the ganglion cells. One pathway is via ON rod bipolars, AII amacrine cells, and ON and OFF-cone bipolars. This is a high gain pathway hypothesized to mediate rod vision at low light levels. The second pathway transmits rod information via rod–cone gap junctions and ON- and OFF-cone bipolars, and is hypothesized to mediate rod vision at high scotopic and mesopic light levels (Daw, Jensen, & Bunken, 1990; Sharpe & Stockman, 1999). Physiological recordings at mesopic light levels reveal rod inputs to the magnocellular (MC), parvocellular (PC) and koniocellular (KC) pathways (Gouras & Link, 1966; Lee, Smith, Pokorny, & Kremers, 1997; Virsu & Lee, 1983; Virsu, Lee, & Creutzfeldt, 1987; Wiesel & Hubel, 1966). The sharing of post-receptoral pathways provides a potential neurophysiological basis for rod–cone interaction in detection, chromatic discrimination, color perception, temporal processing and spatial vision. Here we focus on rod–cone interaction in color perception.

At the mesopic light levels, increased rod activation enhances brightness (Benimoff, Schneider, & Hood, 1982; Ikeda & Shimozono, 1981; Sun, Pokorny, & Smith, 2001a) and decreases the saturation of spectral lights (Buck, Knight, Fowler, & Hunt, 1998; Lythgoe, 1931; Nerger, Volbrecht, & Haase, 2003; Stabell & Stabell, 1975). Literature reports of the effect of rod stimulation on hue are inconsistent (reviewed by Buck et al., 1998). Unique hue measurement studies found that unique blue, green and yellow generally shifted toward longer wavelengths when the dark adapted and light adapted data were compared. It was inferred that rods contributed to "short-wavelength red", "blue" and "green" percepts (Buck, Knight, & Bechtold, 2000). The data from hue-scaling studies suggest that rods contribute to a more bluish percept for monochromatic lights between 460 and 520 nm and a more greenish percept for monochromatic lights between 540 and 610 nm (Buck et al., 1998; Nerger et al., 2003).

Traditional methods such as unique hue measurement and hue-scaling have not yielded results that are easily explainable in terms of the underlying physiological mechanisms. Using a four-primary photostimulator that allows independent control of the stimulation of the 4-receptor types in the human eye (Pokorny, Smithson, & Quinlan, 2004; Sun et al., 2001a), we matched the color percepts associated with increased rod activation using cone stimuli (Cao, Pokorny, & Smith, 2005). When the rod signal increases, the percept appears bluish-green and brighter; when there is a decrease in the rod signal, the percept appears more reddish and dimmer (Cao, Zele, & Pokorny, 2008; Cao et al., 2005). We identified that the incremental rod contribution is analogous to an increment in M-cone excitation relative to L-cone excitation, and additionally, to an increment in S-cone excitation at light levels near cone threshold (1 or 2 Td).

The first purpose of this study is to extend our previous work, by investigating the rod contributions to color perception as a function of rod contrast. The rationale is that physiological recordings show a linear relationship between cell response and cone contrast in PC cells at all light levels, and a linear response for MC cells at light levels <30 Td (Purpura, Kaplan, & Shapley, 1988). At mesopic light levels, if rod signals enter the cone path-

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doi:10.1016/j.visres.2008.05.001

way via rod–cone gap junctions, then rod input should be analogous to cone input and we expect a linear relationship between the rod incremental contrast and the matched cone response mediated via PC and MC units.

Modern color vision models describe spectral information passing through two or more sequential processing stages. The first stage is spectral coding by L-, M- and S-cones, followed by a stage of excitatory and inhibitory recombination of cone excitations to produce postreceptoral spectral signals. Postreceptoral processing includes the putative red/green and blue/yellow spectrally opponent mechanisms, as well as an achromatic, black/white non-spectrally opponent mechanism. There are numerous zone theories, but most assume linearity between cone excitations and postreceptoral responses. Two frequently cited examples of linear zone models developed to model color perception are the two-stage Hurvich and Jameson model (Hurvich, 1981) and the multi-stage DeValois and DeValois model (DeValois & DeValois, 1993). The difference between the two models is that in the Hurvich & Jameson model, M-cones signal greenness and yellowness, while in the DeValois & DeValois model, M-cones contribute to greenness and blueness. To interpret rod contributions to unique hue measurements, Buck et al., 2000 attempted to incorporate rod excitation into the Hurvich–Jameson and DeValois–DeValois models and found that a linear combination of rod and cone activity was not sufficient to account for the unique hue shifts.

A physiologically plausible model based on the responses of primate ganglion cells has been successfully developed to describe chromatic discrimination data (Pokorny & Smith, 2004; Smith, Pokorny, & Sun, 2000) and to separate the effects of spatial and temporal chromatic contrast on discrimination (Cao, Zele, Smith, & Pokorny, 2008; Zele, Smith, & Pokorny, 2006). Here, this model is expanded to incorporate rod contributions at mesopic light levels, where rod signals are assumed to be transmitted via the rod–cone gap junction pathway (Kolb, Goede, Roberts, McDermott, & Gouras, 1997; Sharpe & Stockman, 1999). The second aim of the study is to extend the model to describe the effect of varying rod contrast.

2. Part I: Experiment and data

2.1. Methods

2.1.1. Apparatus

A 2-channel, 4-primary photostimulator allowed independent control of excitation of the rods and three cone types independently (Shapiro, Pokorny, & Smith, 1996). A complete description of the design of the photostimulator is given by Pokorny et al. (2004) and examples of its implementation can be found in Cao et al. (2008), Cao et al. (2005), Cao, Zele, and Pokorny (2006), and Cao, Zele, and Pokorny (2007). The dominant wavelengths of the four LED primaries were 459, 516, 561, and 658 nm. The radiances of the primaries were controlled by amplitude modulation of a 20 kHz carrier fed by an eight-channel analog output Dolby soundcard (M-Audio-Revolution 7.1 PCI) with a 24 bits digital-to-analogue converter (DAC) operating at a sampling rate of 192 kHz. The output of each DAC was demodulated (Puts, Pokorny, Quinlan, & Glennie, 2005) and sent to a voltage-to-frequency converter that provided 1 μs pulses at frequencies up to 250 kHz to control the LEDs (Swanson, Ueno, Smith, & Pokorny, 1987). The soundcard with demodulator has a precision of greater than 16 bits (Puts et al., 2005). All stimuli were generated using custom developed software running on a Macintosh G5 PowerPC computer.

2.1.2. Calibration procedures

The photostimulator was calibrated in two steps. The first pertained to the measurement of the spectral distribution and the linearization of physical light for each LED. The second involved observer calibrations to compensate for individual differences in pre-receptoral filtering and receptoral spectral sensitivities. Details of the calibration procedures have been described elsewhere (Cao, Zele, & Pokorny, 2007; Cao et al., 2005; Pokorny et al., 2004; Sun et al., 2001a).

2.1.3. Stimuli

The stimulus pattern consisted of a 2° circular central field within a 13° annular surround. A small, dimly-illuminated achromatic appearing fixation point placed the center of the stimulus at 7.5° in the temporal retina (left panel, Fig. 1). The center and surround had identical cone excitations, with the rod signal in the center being incremented in a 1 Hz temporal square-wave function (right panel, Fig. 1). Each cycle consisted of a 500 ms stimulus epoch with an incremental rod contrast in the central field (that differed in appearance from the surround) followed by a 500 ms matching epoch where the center and surround fields had the same rod and cone excitations and were uniform in appearance.

Data were collected for one cone chromaticity [L/(L + M) = 0.7, S/(L + M) = 0.2] at three light levels, 2, 10 and 100 photopic Td. This cone chromaticity, with a desaturated orange appearance, was chosen because it provided the largest photostimulator rod modulation gamut. Cao et al. (2005) demonstrated that the direction of the chromaticity shift accompanying increased rod excitation was independent of the starting chromaticity. The rod Troland (Shapiro et al., 1996) level of the surround and unmodulated center was set at a ratio of 0.6 to the cone Trolands.

For each retinal illuminance level, four rod incremental contrasts were used. The Weber rod contrasts were 30%, 40%, 60% and 80%. For one observer (IS), matching rod percepts with rod contrasts $\geqslant$60% at 2 Td required large changes in cone signals that exceeded the photostimulator gamut, and rod contrasts of 20%, 30% and 40% were used. The rod modulation was highly conspicuous following dark adaptation, but invisible during the cone plateau (first 3–5 min) of dark adaptation following light adaptation to a 10,000 Td broadband light with a correlated color temperature of 5000 K, confirming rod isolation.

2.1.4. Procedure

Following 30 min of dark adaptation, matches were made for one light level with each rod contrast presented twice using a random presentation order. The change in color appearance of the center field due to the incremental rod signal was characterized by a temporal matching technique, in which the observer adjusted the cone signals [L/(L + M), S/(L + M) and (L + M)] of the center during the matching epoch to equate the rod percept seen during the stimulus epoch. The observer could toggle freely between the stim-

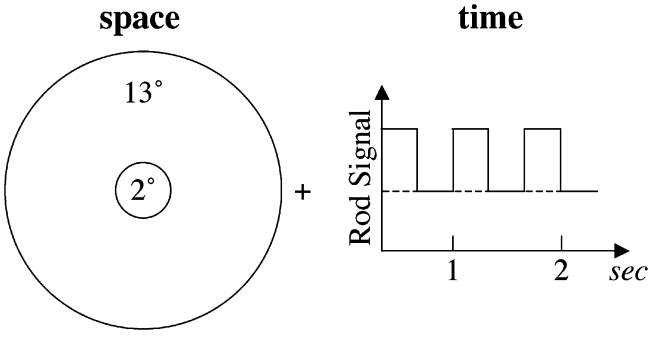

Fig. 1. The spatial structure and temporal profile of the stimulus. The center field was 2°, set within a 13° surround. The fixation point is indicated by the "+" sign and positioned the center field at 7.5° temporal eccentricity. The rod signal in the center was modulated in a 1 Hz square-wave.

ulus epoch, in which rod signal in the center was incremented in a 1 Hz square-wave, and the matching epoch, in which the cone signals in the center were incremented in a 1 Hz square-wave with the observer controlling the cone modulation depths. The 1 Hz square-wave presentations avoided the fading that is associated with steady viewing of peripherally located stimuli (Troxler, 1804). The surround was always unmodulated, with steady cone excitations during the stimulus epoch, and steady rod excitation during the matching epoch. Pressing buttons on a gamepad allowed adjustment of the cone signals during the matching epoch. The gamepad was programmed so that control was analogous to orthogonal directions in a MacLeod–Boynton type chromaticity diagram, with manipulations in L/(L + M) and S/(L + M), all at a constant retinal illuminance. Retinal illuminance could be independently adjusted. A confirmation button signaled a satisfactory match, and the next trial was then presented until the end of the session. Each session was repeated three times on different days. The mean and standard error over the three days for each rod contrast and light level were calculated.

2.1.5. Observers

Two observers, DC and IS, participated in the study. Both have normal color vision (assessed by the Neitz OT anomaloscope) and chromatic discrimination (as assessed by the Farnsworth–Munsell 100-hue test). DC, one of the authors, is an experienced psychophysical observer. IS was a paid undergraduate student who was not aware of the purpose or design of the experiment. All experimental procedures were approved by the Institutional Review Board at the University of Chicago.

2.2. Results

The change in L/(L + M), S/(L + M) and (L + M) required to match rod percepts with different rod contrasts at 2, 20 and 200 Td are shown in Fig. 2, with the data for DC on the top row and the data for IS on the bottom row. The dashed lines in Fig. 2 show the model predictions (see Section 3). A consistent pattern in the data was that a decrease in L/(L + M) and an increase in (L + M) were necessary to match rod percepts for the rod contrasts at all light levels. The change in S/(L + M) varied with light level. At 2 and 10 Td, an increase in S/(L+M) was required, although the change in S/(L + M) was very small at 10 Td. At 100 Td, a weak but systematic decrease in S/(L + M) was observed at high rod contrasts. With increasing light level, the magnitudes of the cone signals required to match the rod percepts decreased, as indicated by the decreasing slopes of the dashed lines in each panel of Fig. 2. Most importantly, the changes in L/(L + M), S/(L + M) and (L + M) were linearly related to rod contrast at each light level. A simple linear regression analysis indicated that a linear relationship was adequate to describe the change in L/(L + M), S/(L + M), and (L + M) with different rod contrasts ($R^2 > 0.85$ for DC or $R^2 > 0.92$ for IS).

3. Part II: model

3.1. Overview of the model

The model for L- and M-cone spectral processing is based on the spectral opponent PC pathway of primates (Derrington, Krauskopf, & Lennie, 1984; Lee, Pokorny, Smith, & Kremers, 1994; Lee, Pokorny, Smith, Martin, & Valberg, 1990) and has separate spatial and temporal components. The model can be used to derive threshold predictions for chromatic discrimination data (Pokorny & Smith, 2004; Smith et al., 2000) and the separate effects of spatial and temporal chromatic contrast on chromatic discrimination (Zele et al., 2006). The model includes a first stage gain control mechanism, and a second stage spectrally opponent signal, which is subject to subtractive feedback. The response to a chromatic contrast change from the adapting chromaticity follows a static saturation function that describes retinal ganglion PC cell responses to contrast changes from their adapted steady-state level. The KC spectral processing is modeled parallel to that for the PC spectral processing except that the opponency is generated between S-cone signals and the sum of L- and M-cone signals (Miyahara, Pokorny, & Smith, 1996). The MC processing is modeled by a weighted sum of the L- and M-cone responses. Here we extended the model to include rod processing, assuming that rod

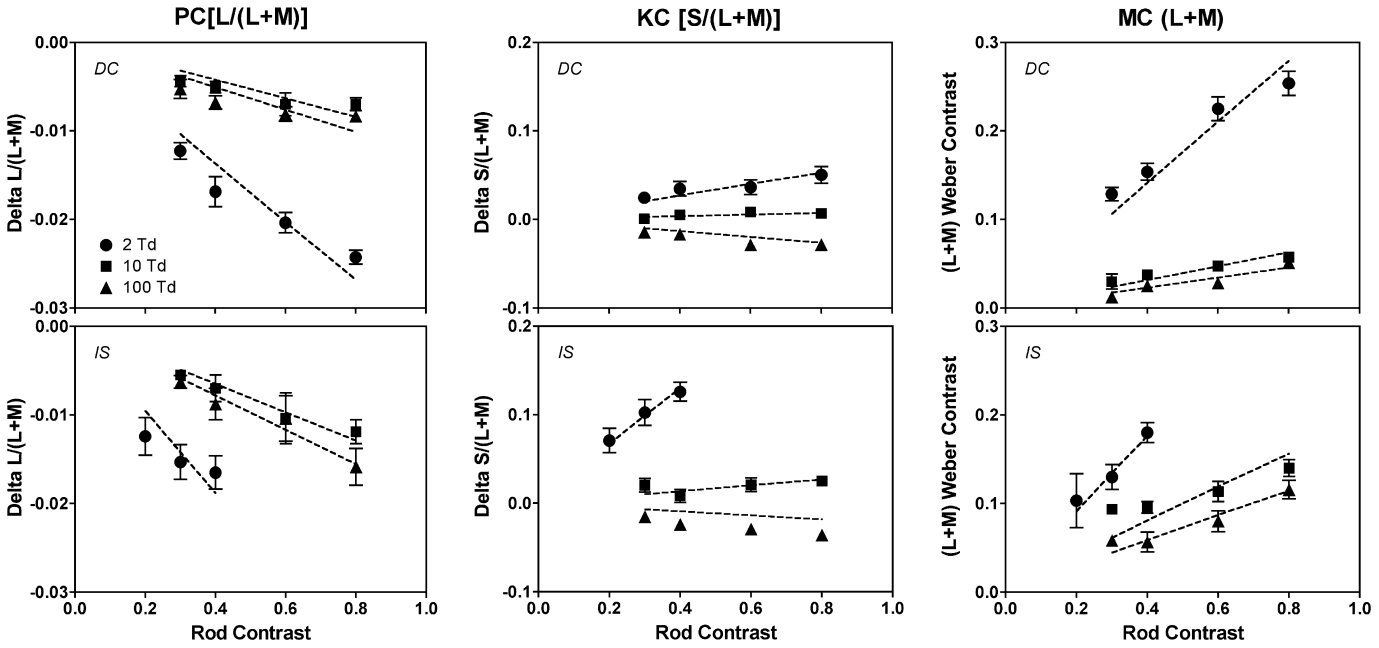

Fig. 2. The change in L/(L + M), S/(L + M) and (L + M) to match rod percepts with different rod contrasts at 2, 10 and 100 Td. The dashed linear are model fits. Upper: for observer DC. Lower: for observer IS. Note that with zero rod contrast, the model fits intersect at the origin.

inputs to ganglion cells were transmitted via rod–cone gap junctions. Details of the model are described next.

3.2. Description of ganglion cell response model

The model assumes an early cone-specific multiplicative adaptation process (Lee et al., 1990; Smith, Lee, Pokorny, Martin, & Valberg, 1992; Swanson et al., 1987), followed by a spectral opponency between L- and M-cones for the PC pathway or between S and (L + M) cones for the KC pathway. The cone responses to a light of specific L-, M-, S-cone Trolands are given by:

$$R_L = L/l_{max} \quad (1)$$

$$R_M = M/m_{max} \quad (2)$$

$$R_S = S/s_{max} \quad (3)$$

where L, M and S are cone Trolands, and l_{max}, m_{max} and s_{max} are maximal sensitivities of the Smith and Pokorny (1975) cone fundamentals. The cone responses are subject to multiplicative sensitivity regulation (gain control):

$$G(L_A) = 1/(1 + k_1 L_A/l_{max})^{k_2} \quad (4)$$

$$G(M_A) = 1/(1 + k_1 M_A/m_{max})^{k_2} \quad (5)$$

$$G(S_A) = 1/(1 + k_1 S_A/s_{max})^{k_2} \quad (6)$$

where L_A, M_A and S_A are adapting cone Trolands and k_1 and k_2 are constants. The value of k_1 is about 0.33 Td and the value of k_2 is about 0.5 (Miyahara, Smith, & Pokorny, 1993).

The cone spectral opponent term can be derived for each of the subtypes of PC pathway cell, (+L/−M), (+M/−L), (−L/+M), and (−M/+L). For an (+M/−L) cell for example, the spectral term at the test chromaticity is given by:

$$OPP_{(+M/-L)} = [M_T/m_{max}G(M_A) - k_3 L_T/l_{max}G(L_A)] \quad (7)$$

where L_T and M_T represent cone Trolands at the test chromaticity, and k_3 represents the surround strength of spectral opponency. In retinal ganglion cells, the surround strength of PC pathway cells is in the range of 0.7–1.0 (Smith et al., 1992) and 0.8 is used for the PC modeling. For the equiluminant chromatic stimuli, the (+L/−M) and (−M/+L) give redundant information, responding positively to "redward" changes from their adaptation point; similarly the (+M/−L) and (−L/+M) give redundant information, responding positively to "greenward" changes from their adaptation point (Lee et al., 1994). To model the PC spectral response, only a pair of cells of opposite chromatic signatures is required; for example, (+L/−M) for a "redward" response and (+M/−L) for a "greenward" response.

For the +S/−(L + M) cell in the KC pathway, the opponent term is given by:

$$OPP_{+S/-(L+M)} = S_T/s_{max}G(S_A) - k_3[pL_T/l_{max}G(L_A) + (1-p)M_T/m_{max}G(M_A)] \quad (8)$$

where k_3 is the surround strength of the opponent signal, and p refers to the relative weight of L-cones in the MC pathway for a Judd observer, and has a value of 0.6189.

For both the PC and KC pathways, the spectral opponent signals are subject to a subtractive feedback, the strength of which is determined by the opponent signal at the adapting chromaticity:

$$OPP_C = OPP_T - k_4 OPP_A \quad (9)$$

where OPP_C is the spectral opponent signal to a chromaticity change, C, from a fixed adapting chromaticity, A, to a test chromaticity, T. OPP_T is the spectral opponent term at the test chromaticity, OPP_A represents the spectral opponent term at adapting chromaticity, and k_4 represents the subtractive feedback strength. The response of a spectral opponent cell to a chromaticity change, C, from a fixed adapting chromaticity, A, is:

$$R_{OPP} = K \cdot OPP_C/(OPP_C + SAT) \quad (10)$$

where OPP_C is a spectral opponent term in Eq. (9), K is a scaling factor of the response, and SAT is the static saturation. In a single cell, K can be considered the criterion response divided by the maximal response rate R_{max}. The application of a physiological model to psychophysical discrimination data however, involves higher order processes that combine inputs from arrays of retinal cells (Smith & Pokorny, 2003; Zele et al., 2006) and knowledge of the maximal response rate, R_{max}, as well criterion response and semi-saturation value of the single cell is lost.

Finally, the luminance mechanism in the MC pathway, LUM, is modeled by the sum of L- and M-cone responses following the gain (Miyahara et al., 1996):

$$LUM = K[pL_T/l_{max}G(L_A) + (1-p)M_T/m_{max}G(M_A)] \quad (11)$$

where K is a scaling constant and p has the same value as in Eq. (8).

3.3. Model extension with rod input

The model described above is expanded to incorporate rod contributions at mesopic light levels, where rod signals are assumed to be transmitted by the rod–cone gap junction pathway (Kolb et al., 1997; Sharpe & Stockman, 1999). Therefore, with rod input (V′), Eqs. (1)–(3) become:

$$R_{L+V'} = (L + k_5 V')/l_{max} \quad (12)$$

$$R_{M+V'} = (M + k_5 V')/m_{max} \quad (13)$$

$$R_{S+V'} = (S + k_6 V')/s_{max} \quad (14)$$

where k_5 and k_6 are the rod input strength into the L/M-cones and S-cones, respectively.

For a given cone chromaticity, the perceived greenness/redness is determined by the difference in responses from the (+M/−L) and (+L/−M) units, and the difference is normalized such that an equal energy spectrum (EES) light, which appears white in the absence of rod input, has a zero difference. The greenness/redness at each light level, G/R, is determined by:

$$G/R = R_{+M/-L} - \frac{R_{+M/-L_{white}}}{R_{+L/-M_{white}}} R_{+L/-M} = R_{+M/-L} - r_w R_{+L/-M} \quad (15)$$

where $R_{+L/-M}$ and $R_{+M/-L}$ are responses for the (+L/−M) and (+M/−L) units, while $R_{+L/-Mwhite}$ and $R_{+M/-Lwhite}$ are responses for the cell units to an EES light without rod input, which appears white. Note that the normalization is not critical in terms of the quality of the fits: with different normalizations, the fitted parameter k_5 will be different whereas k_5 will not change with retinal illuminance for the PC modeling. With k_3 = 0.8, the calculated ratio of $R_{+M/-L_{white}}/R_{+L/-M_{white}}(r_w)$ is 0.139, 0.265, and 0.354 at 2, 10, and 100 Td, respectively. If the normalized difference in Eq. (15) is positive, then the appearance will be greenish, compared to white. If the normalized difference is negative, then the appearance will be reddish. When the rod signal is incremented from V' by a Weber contrast c, with constant cone excitations, the rod contributions to greenness/redness, $RC_{G/R}$, can be modeled as:

$$RC_{G/R} = G/R_{V'(1+c)} - G/R_{V'} \quad (16)$$

where $R/GV'(1 + c)$ and $R/G(V')$ are the greenness/redness with rod input of $V'(1 + c)$ and V', respectively.

Typically, the static term in Eq. (10) has a value of 10. For the light level used in the experiment (2, 10 and 100 Td), the term *OPPc* has a maximum value of 0.3, which is substantially less than the value of SAT. Therefore, Eq. (10) can be approximated by a linear function:

$$R_{OPP} \approx K \times OPP_C/SAT = K' OPP_C \quad (17)$$

where K' is equal to K/SAT . By some algebra, it can be shown that $\mathrm{RC}_{R/G}$ is approximately a linear function of rod contrast, c:

$$\begin{aligned}\mathrm{RC}_{G/R} \approx K'[(1+r_w k_3)G(\mathrm{M_A}+k_5V')/m_{\max} \\ -(k_3+r_w)G(\mathrm{L_A}+k_5V')/l_{\max}]k_5V'c\end{aligned} \tag{18}$$

In other words, the model predicts an approximately linear relation between rod contributions to the greenness/redness and rod contrast as observed in the data (dashed lines in Fig. 2).

For KC modeling, we assume that the perceived blueness/yellowness is determined by the response of S/(L+M) unit alone. The rod contributions to blueness/yellowness, $\mathrm{RC}_{B/Y}$, can be modeled as the difference in B/Y values with rod signals of V' and $V'(1+c)$:

$$\mathrm{RC}_{B/Y} = R_{\mathrm{OPP}_{\mathrm{C}\cdot V'(1+c)}} - R_{\mathrm{OPP}_{\mathrm{C}\cdot V'}} \tag{19}$$

where $R_{\mathrm{OPP}_{\mathrm{C}\cdot V'(1+c)}}$ and $R_{\mathrm{OPP}_{\mathrm{C}\cdot V'}}$ are response of S/(L + M) with rod signal V' and $V'(1+c)$, respectively. Similar to rod contributions to greenness/redness, it can be shown that given the spectral opponency is much smaller than the saturation term SAT, the rod contributions to the blueness/yellowness is approximately a linear function of rod contrast, c,:

$$\begin{aligned}\mathrm{RC}_{B/Y} \approx K'\{k_6G(\mathrm{S_A}+k_6V')/s_{\max} \\ -k_3[pk_5G(\mathrm{L_A}+k_5V')/l_{\max}+(1-p)k_5G(\mathrm{M_A}+k_5V')/m_{\max}\}V'c\end{aligned} \tag{20}$$

Finally, the rod contributions to the MC pathway is a also linear function of rod contrast:

$$\begin{aligned}\mathrm{RC}_{\mathrm{L+M}} &= \mathrm{LUM}_{V'(1+c)} - \mathrm{LUM}_{V'} \\ &= K[pG(\mathrm{L_A}+k_5V')/l_{\max}+(1-p)G(\mathrm{M_A}+k_5V')/m_{\max}]k_5V'c\end{aligned} \tag{21}$$

Eqs. (18)–(21) demonstrate that for the light levels we used in the experiment, the model predicts the rod contributions to the PC, KC and MC pathways each to be a linear function of rod contrast.

3.4. Model fitting

The model is used to fit the data from the experiment, with k_5 (rod input strength to the L/M-cones), k_6 (rod input strength to the S-cones), and K for the MC pathway (Eq. (21)) as the free parameters at each light level. The values of remaining parameters (See Table 1) were set in accord with physiological data (e.g. Smith et al., 1992) and results from psychophysical chromatic discrimination studies of the PC- and KC-pathways. A least squares procedure was used to search the value of the free parameters to minimize the sum of squared difference between the predicted values and data. At each light level, a common value of k_5 was searched for all of the matching L/(L + M), S/(L + M) and (L + M) data. A single k_6 value was searched for the S/(L + M) matching data, and a single K value for the (L + M) matching data. At 100 Td, the value of k_6 was set to zero because a decrease in S/(L + M) was required to make the match, and a negative k_6 value of is physiologically implausible. The model fits are shown in Fig. 2 as the dashed lines in each panel. Note that the model implies that with zero rod contrast, the rod contributions are zero and the model fit will intersect with the origin. Overall, with a limited number of parameters for the rod input strength to the PC, KC and MC pathways, the models described the matching data well. Fig. 3 shows the fitted values of k_5 and k_6 at different light levels for two observers. Overall, the input strength to the L/M-cones or to the S-cones was strongest at 2 Td and decreased greatly at 10 and 100 Td.

Table 1
The notation and the values of the parameters for the model

Parameters	Notation	PC pathway	KC pathway	MC pathway
Cone parameters	$l_{\max}$	0.63721	0.63721	0.63721
	$m_{\max}$	0.39242	0.39242	0.39242
	$S_{\max}$		1.6064	
	P		0.6189	0.6189
Early adaptation parameters	k_1	0.33	0.33	
	k_2	0.5	0.5	
Opponent parameters	k_3	0.8	0.6	
	k_4	0.95	0.8	
Strength of rod input[a]	k_5	Free	Free	Free
	k_6		Free	
Parameters used to fit chromatic discrimination	SAT	10	10	
Scaling parameter	K	45	45	Free

[a] The value of k_s is commonly searched based on the data from the PC, KC and MC pathway.

4. Discussion

We used a temporal matching technique to estimate the appearance of a rod incremental stimulus by altering L/(L + M), S/(L + M) and (L + M) cone excitations during a matching interval. At mesopic light levels, rod signals access ON- and OFF-cone bipolar and ganglion cells via gap junctions between the cone pedicles and rod spherules (Sharpe & Stockman, 1999). We propose that rod inputs to the cone pathways via gap junctions should be analogous to direct cone inputs to cone bipolar cells and associated pathways. The cone post-receptoral pathways have no information about which photoreceptor class initiated the signal. The rod percepts therefore have an appearance equivalent to a specific level of PC, KC and MC pathway excitation. The results of the cone matches to the rod percepts demonstrate that rod contributions to color perception involve differential weightings between the MC, PC and KC pathways as a function of the rod incremental contrast and illumination level. In particular, for each illumination level, the measured rod contributions to color perception were linearly related to rod contrast, with the strength of rod input weakening with an increase in retinal illuminance levels in a non-linear fashion. Consistent with the physiological data (Purpura et al., 1988), we observed a linear relationship between the rod incremental contrast and rod input to the MC pathway up to 100 Td. The linear relationship is related to the rod contrasts being effectively equivalent to low cone contrasts, thereby falling on the linear proportion of the contrast–response function. Note Buck et al. (2000) examined whether rod and cone signals combined linearly in the context of the linear zone models; here we examined the linearity in the relationship between rod contrasts and rod percepts.

To incorporate rod input into the retinal ganglion cell based model, we assume rod signals are transmitted via rod–cone gap junctions. Therefore, rod contrast is converted into an equivalent contrast after the rod signal is transmitted via the gap junctions, and a linear relationship between the rod contrast and rod contributions to the PC, KC and MC pathways can be expected. The model not only describes the relationship between rod contrast and cone contrast, but also describes how the rod percept varies with different cone chromaticities and retinal illumination levels (Cao et al., 2005). Overall, the proposed model can be used to describe color perception at mesopic light levels and photopic light levels.

Using this model, we analyzed the strength of rod input to different cone pathways. Overall, rod input to the L/M and S-cone pathways decreased with increasing retinal illumination. The results of the modeling analysis suggest that rod inputs to S-cones are stronger than L/M-cones at low mesopic light levels. Intuitively, rod inputs to the center of the S/(L + M) cell receptive field

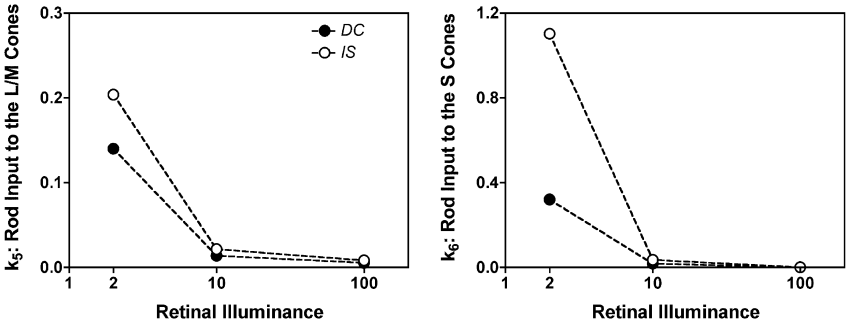

Fig. 3. The fitted strength of rod input to the L/M-cones and S-cones for observers DC (left) and IS (right).

would be expected to be stronger to balance the combined rod input to the L- and M-cones in the surround. In fact, our data indicated positive rod input to the S-cones at 2 and 10 Td, but negative rod input at 100 Td (Fig. 2). The model outputs suggest that rod input to the S-cones outweighs input to the summed L- and M-cone response at 2 and 10 Td. At 100 Td however, L- and M-cone outputs may simply surpass the output from the S-cones, perhaps due to S-cone saturation (Mollon, Astell, & Cavonius, 1992), thus leading to a reduced S/(L + M) excitation in the matched rod percepts. Physiological recordings have revealed no rod input to the bistratified ganglion cells (Lee et al., 1997). This finding, however, does not rule out the possibility that rod input to the bistratified ganglion cells is very weak and it requires a very large modulation contrast to be seen. Rod input to the koniocellular layer in the LGN was reported with an increment threshold procedure (Virsu & Lee, 1983; Virsu et al., 1987), which can achieve high modulation contrast.

To fit the model, we assumed that the strength of rod input to the L/M-cones (k_5) was equal at the photoreceptor level for all the PC-, KC- and MC- pathways. Therefore any measured differences in rod contributions to the post-receptoral pathways need be related to the differences in the post-receptoral anatomical wiring and pathway contrast gain. The scaling factors K in Eqs. (10) and (11) are related to the contrast gain of the pathways. However, the K values for the MC-, PC- and KC pathways cannot be compared because the metrics differ. The calculated K reflect differences either between the matching and starting L/(L + M), S/(L + M) chromaticities or the Weber contrast between the matching and starting (L + M). The values in $\Delta L/(L+M)$, $\Delta S/(L+M)$, and (L + M) contrast were of a very different numerical order (see Fig. 2), leading to disparate K values for different pathways.

One fundamental assumption of the model may necessitate scrutiny. It is assumed that at high scotopic and mesopic light levels the rod signals are transmitted to the cone pathways via rod–cone gap junctions. The rod to rod-bipolar pathway saturates with stimuli evoking more than about ~1 isomerization per rod per integration period, but saturation is insignificant under dark-adapted conditions because the probability of multiple photon absorptions in a single rod is small (e.g. Berntson, Smith, & Taylor, 2004; Robson & Frishman, 1995). In macaque retina, rod–cone electrical coupling extends to the upper range of scotopic vision (Hornstein, Verweij, Li, & Schnapf, 2005) suggesting there are scotopic light levels where both rod pathways are operational. If so, Eqs. (12)–(14) would need to be extended to account for rod signals that are not combined at the photoreceptor level. Nevertheless, the model predication about the linear rod contributions to color perception is still viable as long as the signals from both rod pathways are combined linearly. Indeed, the data show that rod contributions to inferred PC, KC and MC pathway mediated vision are linearly related to the rod incremental contrast, a relationship adequately described by a model based on primate ganglion cell responses with the assumption that rod signals are conveyed via rod–cone gap junctions at mesopic light levels.

Acknowledgments

Supported by National Eye Institute Grant EY00901-35, Australian Research Council Discovery Project DP0773544 and a Research to Prevent Blindness Challenge Grant. We thank Jules Quinlan for technical assistance with the optical system and Linda Glennie for technical assistance in computer programming.

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