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Vision Research 44 (2004) 2843–2855
Vision Research
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Modeling color percepts of dichromats

Thomas Wachtler [a,*], Ulrike Dohrmann [b], Rainer Hertel [a]

[a] Institut für Biologie III, Albert-Ludwigs-Universität, Schänzlestr. 1, 79104 Freiburg, Germany
[b] Institut für Humangenetik, Albert-Ludwigs-Universität, Breisacherstr. 33, 79110 Freiburg, Germany

Received 3 July 2003; received in revised form 24 March 2004

Abstract

Protanopes and deuteranopes, despite lacking a chromatic dimension at the receptor level, use the color terms "red" and "green", together with "blue" and "yellow", to describe their color percepts. Color vision models proposed so far fail to account for these findings in dichromats. We confirmed, by the method of hue scaling, the consistent use of these color terms, as well as their dependence on intensity, in subjects shown to have only a single X-chromosomal opsin gene each. We present a model for the processing of photoreceptor signals which, under physiologically plausible assumptions, achieves a trichromat-like representation of dichromatic receptor signals. Key feature of the dichromat model is the processing of the photoreceptor signals in parallel channels with different gains and nonlinearities. In this way, the two-dimensional receptor signals are represented on a manifold in a higher-dimensional space, supporting categorization for efficient image segmentation. Introducing a third cone opsin yields a model that explains normal, trichromat hue scaling.

Keywords: Dichromacy; Hue scaling; Color vision model

1. Introduction

Color vision in human trichromats is based on three types of retinal cone photoreceptors which contain three photopigments with different spectral sensitivities (Dartnall, Bowmaker, & Mollon, 1983). Two of these cone types constitute an older color system shared by most mammals (Mollon, 1989). The third cone pigment appeared relatively late in primate evolution, and is found almost exclusively in Old World primates. The molecular genetic basis of this trichromacy is well established, and the molecular evolution of normal and defective color vision has been analyzed extensively (Nathans, Thomas, & Hogness, 1986).

Perceptually, human color vision is organized in an opponent fashion, with pairs of mutually exclusive perceptual categories of "light"–"dark", "red"–"green", and "blue"–"yellow". Thus, color percepts can be represented in a three-dimensional space spanned by color axes corresponding to these opponent pairs.

It is often implied that the three-dimensional aspects of our perceptual color space result from the trichromatic receptoral substrate (e.g. Viénot, Brettel, Ott, Ben M'Barek, & Mollon, 1995, but see MacLeod, 1985; Shepard, 1992a). Dichromats lack one of the three cone photoreceptor types. Consequently, it is assumed that the dimensionality of their color percept is reduced and that, e.g., protanopes lack the "red"–"green" axis (see e.g. Brettel, Viénot, & Mollon, 1997; Sharpe, Stockman, Jägle, & Nathans, 1999; Viénot et al., 1995). However, many studies have shown that dichromats use these color terms, together with "blue" and "yellow", to describe their color percept (Boynton & Scheibner, 1967; Jameson & Hurvich, 1978; Kalmus, 1965, Table 5;

* Corresponding author. Address: Department of Physics/Neurophysics, Philipps-University, Renthof 7, 35039 Marburg, Germany. Tel.: +49 6421 282 6631; fax: +49 6421 282 7034.
E-mail address: thomas.wachtler@physik.uni-marburg.de (T. Wachtler).

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doi:10.1016/j.visres.2004.06.016

Scheibner & Boynton, 1968). This indicates that the number of perceptual color categories can be larger than expected from the spectral dimensionality of the receptoral substrate.

Born, Grützner, and Hemminger (1976) studied heterozygous females having protanope or deuteranope patches in their retinae. Their work documents the consistent use of all four color terms for stimuli presented in trichromatic patches as well as for stimuli in dichromatic patches of the photoreceptor mosaic.

That dichromat color percepts are not restricted to a subset of those of trichromats was extensively documented by Scheibner and Boynton (1968). All dichromats tested in this study (three protanopes and five deuteranopes) used "red" and "green"—in addition to "blue" and "yellow"—when tested with monochromatic lights of different wavelengths. The authors suggested that these percepts might be due to residual trichromacy. Molecular evidence for or against this proposal could not be obtained then, due to the lack of suitable methods to analyze the X-chromosomal opsins at the molecular level.

We determined the X-chromosomal opsin gene sequences of dichromats using the polymerase chain reaction (PCR) technique. In two dichromats who were found to have only a single X-chromosomal opsin gene each—either that for the middle-wavelength sensitive M cone or that for the long-wavelength sensitive L cone—we confirmed the results of Scheibner and Boynton (1968), using their method of hue scaling of monochromatic lights. We present a model of color processing, based on neurophysiologically plausible mechanisms, which explains the observations and accounts for the apparent discrepancies between cone input space and the structure of perceptual color space.

2. Methods

2.1. Subjects

Four protanopes and three deuteranopes, six males and one female, were examined. Preliminary screening was done using Ishihara plates and visual performance under long-wavelength (>710 nm) light. Two of the male subjects (AW and SH), as well as a normal trichromat control subject (RH) were tested with the Nagel anomaloscope, the Farnsworth–Munsell 100 hue test, and by characterizing the X-chromosomal opsin genes by molecular genetic analysis.

Subjects were students or colleagues, having some basic understanding of relevant concepts in color vision, such as hue and saturation. They were aware that their color vision was investigated, but were naive with respect to the purpose of the experiments. The tests were conducted in German, the native language of all subjects.

2.2. Polymerase chain reaction (PCR) and DNA sequencing

PCR. Genomic DNA was extracted from blood and used in three different PCR studies: (1) conventional PCR to determine whether exon 5 of either L or M opsin was deleted in the dichromats; (2) conventional PCR to generate amplicons for sequencing exon 5 for all three subjects; (3) quantitative PCR to measure gene dosage using the LightCycler instrument and the FastStart DNA Master SYBR Green I kit following the manufacturer's experimental protocol (Roche Diagnostics, Mannheim, Germany); data analysis was performed with the second derivative maximum method of the LightCycler software. At the end of the LightCycler runs, the PCR products were recovered and their lengths confirmed by agarose gel electrophoresis. The opsin amplicon values were normalized to those of glyceraldehyde-3-phosphate dehydrogenase (GAPDH).

Primers. The L opsin primers were derived from the sequence of the human cosmid XX-QC8B6 (GenBank accession number Z68193), which contains the complete L opsin gene sequence; the M opsin primers were derived from the human cosmid XX-CG1160 (GenBank accession number Z46936) which contains the 3′ end of the M opsin gene. Primer sequences present in both cosmids were designated L/M while primer sequences unique to either L or M opsin were designated L and M, respectively. The following list shows the forward (f) and reverse (r) primers used for the different PCR studies; the 5′ positions in cosmid XX-QC8B6 are given in parentheses: (a) specific amplification of M exon 5: Mex5f: gatggtcctggcattctgc (17669), L/Mex5r gggttgtagatagtggcac (17806), (b) specific amplification of L exon 5: Lex5f: gatctttgcgtactgcgtctgc (17669), L/Mex5r gggttgtagatagtggcac (17806), (c) primers for sequencing L or M exon 5 or L/M exon 5: L/Mex5fseq ggtggcaaagcagcagaaag (17594), L/Mex5r gggttgtagatagtggcac (17806), (d) quantitative PCR for exon 2: L/Mex2f ccttcgaaggcccgaattac (11957), L/Mex2r cacagggagacggtgtagcc (12248), (e) quantitative PCR for exon 3: L/Mex3f gatcacaggtctctgctctc (14240), L/Mex3r ctgctccaaccaaagatg (14407), (f) quantitative PCR for exon 5: L/Mex5fseq ggtggcaaagcagcagaaag (17594), L/Mex5r gggttgtagatagtggcac (17806), and (g) GAPDH primers were derived from GenBank accession number NM_002046: GAPDHf gtattgggcgcctggtcac, GAPDHr ccgttctcagccttgacggtg.

2.3. Hue scaling

Monochromatic stimuli (spectral width 20 nm) were produced with a diffraction grating monochromator (Bausch & Lomb, Rochester, USA) illuminating, via a light guide, a 2° field on a matte translucent screen. Using neutral density filters, stimuli of 920 td ("bright

stimulus'' condition) or 230 td (''dark stimulus'' condition) were produced. Stimulus luminance was constant within 15% for stimuli between 510 and 630 nm and, for technical reasons, was lower above and below this range.

Background intensity was adjusted by back-illuminating the screen surrounding the stimulus with a controllable fluorescent lamp. Two background conditions were used, a ''dark background'' of 10 td and a ''bright background'' of 920 td.

In each trial, the subject was asked to describe the appearance of the stimulus by giving the relative proportions of primary hues in the stimulus. Subjects were asked to use the four color terms ''blue'', ''yellow'', ''green'' and ''red'' if possible, but were in principle free to use additional terms in case they could not describe their percept with these terms. This situation never occurred. Stimulus wavelengths were chosen in 10 nm steps, in either ascending or descending order. Control trials where wavelengths were chosen randomly yielded identical results.

3. Results and model

3.1. Two subjects with a single X-chromosomal opsin gene

For the purposes of our study it was first necessary to establish the number of M and L opsin genes in our dichromat subjects. Several color vision tests, including the Farnsworth–Munsell 100 Hue test, had consistently and unambiguously established that subject AW was protanope and SH was deuteranope. These two dichromats were examined in more detail. PCR and sequence analysis were employed to determine their X-chromosomal opsin gene arrays. To summarize briefly, the analyses revealed unambiguously that each of these two subjects had only a single X-chromosomal opsin gene. Since the reliability of these results is essential, we describe analysis and results in greater detail in the remainder of this section.

In a first set of experiments we made use of the few nucleotide differences, most prominently seen in exon 5, that exist between M and L opsin (Sharpe et al., 1999). Using unique forward primers and a common reverse primer, several independent conventional PCR experiments were carried out with the genomic DNAs of SH and AW, and the DNA of a trichromat control, RH. These PCRs clearly showed that the DNA of the dichromat subjects could only be amplified with one type of primer pair each, whereas the DNA of the trichromat yielded two amplicons. The deuteranope SH did not show the M opsin amplicon, and the proteranope, as expected, missed the L opsin amplicon (Fig. 1a).

Subsequent sequence analysis showed that this was due to the deletion of at least most of exon 5. PCR products of exon 5 were generated but this time we used a common forward primer, 75 bp upstream of the specific forward primers. The trichromat's DNA produced a chimeric sequence, indicative of the presence of both opsin genes. The dichromats had only one type of sequence, namely the L-specific one in the deuteranope and the M-specific one in the protanope. Short, partial sequences, including codons 273–280, are given in Fig. 1b.

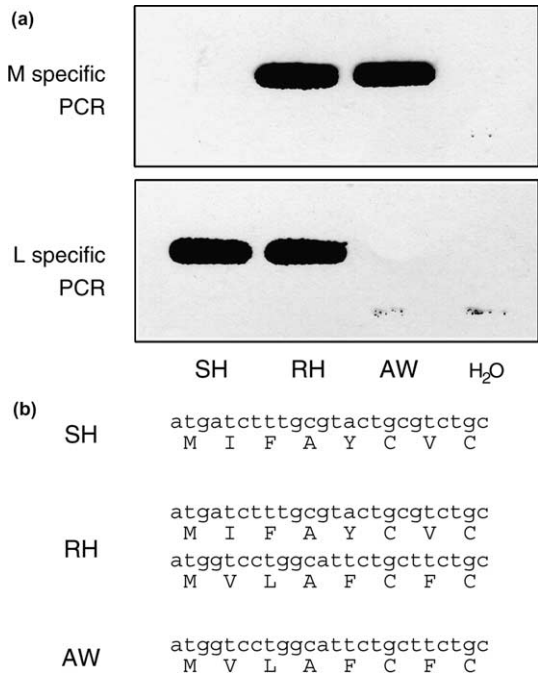

Fig. 1. PCR results. PCR analysis and sequencing of genomic DNA from one trichromat (RH) and two dichromat subjects (SH and AW). (a) PCR products (137 bp) using sequence specific forward primers and a conserved reverse primer for exon 5 of the gene for M opsin (upper panel) and of the gene for L opsin (lower panel). Control: no genomic DNA. (b) Partial sequences for exon 5, obtained from a separate PCR reaction (see text for details).

Next, it was important to show that the deletions were not limited to exon 5. Using quantitative PCR with conserved primers, we could establish that the deletions did not only concern exon 5 but also encompassed at least exons 2 and 3. In these exons, the relative doses measured (Table 1) were twice as high in the trichromat as compared to the dichromats. Together with the data shown in Fig. 1, these results are clear evidence that our

Table 1
Quantitative PCR of parts of the M and L genes

	Exon 2	Exon 3	Exon 5
RH	1.1 ± 0.02	0.93 ± 0.02	1.1 ± 0.07
SH	0.48 ± 0.01	0.65 ± 0.02	0.47 ± 0.01
AW	0.54 ± 0.03	0.59 ± 0.07	0.46 ± 0.14

Normalized amounts of DNA (mean and standard error) from exons 2, 3, and 5 for the three subjects. $N = 4$ for exon 2, $N = 2$ for exons 3 and 5 (see text for details).

dichromat subjects possess only one functional X-chromosomal opsin gene each, i.e. the exons relevant for the photoreceptor function (Nathans et al., 1986) were found as single copies. The data presented do not exclude the possibility that other parts of a second gene, e.g. exon 6, were still present in the dichromats.

3.2. Hue scaling of dichromats

Several features characteristic for hue scaling results of dichromats, as reported in previous studies (Boynton & Scheibner, 1967), were found consistently in our experiments testing four protanopes and three deuteranopes. As in trichromats, "blue" was reported by all subjects in stimuli below 500 nm. Around 500 nm, there was a peak for "green". In comparison to results of trichromats, this "green" peak was narrower in most dichromats, where no "green" was reported for stimuli above 530 nm. The mean and standard deviation for the peak wavelength for "green" was 504 ± 5 nm, the average maximum value was 0.89 ± 0.08, and the width of the peak was 26.4 ± 12 nm.

In the wavelength region above 530 nm, stimuli were typically described as mixtures of "yellow" and "red", with a clear tendency to increasing proportions of "red" at longer wavelengths. A contribution of "red" was also reported for short-wavelength stimuli below 450 nm by all subjects. At 440 nm, the proportion of "red" was on average 0.11 ± 0.07.

Interindividually, there were considerable quantitative differences in the relative contributions of primary hues assigned by different subjects for a given wavelength (Scheibner & Boynton, 1968). But qualitatively, the hue scaling functions were similar. It should be noted that, in very rare instances, some dichromat subjects reported "red" or "green" ratings that did not match their other results. We observed this in two subjects, where, in one test run each, substantial percentages of either "green" or "red" were reported in a seemingly random fashion in a narrow spectral region around 510–530 nm. A similar case had been observed by Scheibner and Boynton (1968). One of these subjects, again in one instance only, reported "green" at 440 nm.

In the following, we focus on the results of the two male subjects whose lack of L and M cone opsin genes, respectively, had been confirmed by our molecular genetic analysis. The hue scaling results of these subjects are shown in Fig. 2. The data illustrate the specific features of dichromat hue scaling, including the strong dependence on stimulus intensity. This dependence is particularly conspicuous in the long-wavelength region. Here, the relative proportions of "red" and "yellow" consistently shift with intensity, favoring "red" at lower, "yellow" at higher intensities (see also Paramei, Bimler, & Cavonius, 1998; Scheibner & Boynton, 1968). A similar effect could be achieved by varying the intensity of the background on which the stimuli are presented (data not shown), where increasing the background luminance had an effect corresponding to decreasing stimulus intensity. Such behavior had been reported earlier (Scheibner & Boynton, 1968). Thus, while discrimination of long-wavelength stimuli by dichromats is based exclusively on intensity, a mismatch in intensity may be perceived as a color difference. In our data, this is also reflected in the variation of hue proportions with wavelength in the long-wavelength region (Fig. 2).

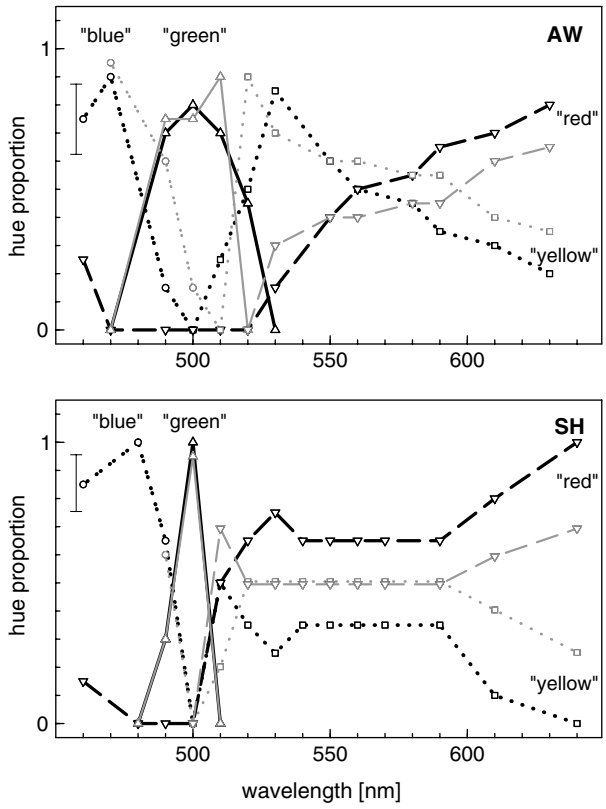

Fig. 2. Hue scaling results of dichromats. Proportions of "red" (dashed lines), "green" (solid), "yellow" (dotted), and "blue" (double-dotted) as functions of wavelength for the protanope (top) and the deuteranope subject (bottom). Results for low and high luminance stimuli are plotted with black and gray lines, respectively. Data points are means of two experiments. Error bars at left denote the maximal deviations observed across all data points; for most data points, deviations were considerably smaller. Besides "blue" and "yellow", both dichromats use "green" and "red" for stimuli at different wavelengths.

There are three lines of evidence that color percepts of dichromats are comparable to percepts in trichromats. First, in the spectral region around 420–450 nm, subjects report a contribution of "red". When asked about the relation of the percepts described by "red" in different stimuli, the subjects explicitly stated that the "red" seen in mixture with "blue" (i.e., in short-wavelength stimuli) had the same perceptual quality as the "red" in mixtures with "yellow" (long-wavelength stimuli).

Secondly, in heterozygote retinal mosaics, a small spot of 642 nm light is perceived as "red" (Born et al., 1976, see below) even when presented in a protanopic area of the retina. The heterozygous women certainly know what "red" is like, from experiences in the trichromatic areas of their mosaics. Thirdly, the use of "red" and "green" by our subjects was consistent within and across experimental runs, which were separated by several weeks. Furthermore, the "red"–"yellow" intensity dependence at long wavelengths had the same qualitative features as in trichromatic subjects.

3.3. A new model of color processing in dichromats

3.3.1. "Classical" models fail to explain hue scaling results of dichromats

Protanopes and deuteranopes claim and name a color percept "green" at wavelengths around 510 nm, and "red" at both ends of the visible spectrum. However, published color vision models (e.g. De Valois & De Valois, 1993; Guth, Massof, & Benzschawel, 1980; Hassenstein, 1968; Hurvich, 1981; Ingling, Barley, & Ghani, 1996; Werner & Wooten, 1979) fail to predict these aspects. They describe hue naming and scaling in trichromats quite adequately, but when one of the longer-wavelength receptors is omitted, only "blue"–"yellow", but not "green"–"red" can be derived from receptor stimulation. Most studies do not even address the problem of dichromats. Hassenstein (1968) explicitly considers the dichromat cases. However, his model does not predict the hue scaling results of dichromats as reported by Boynton and Scheibner (1967), and his phenotype/genotype assignments have not been confirmed by recent molecular analysis (Nathans, 1999). Guth et al. (1980) derived predictions for dichromat wavelength discrimination from their model, but did not consider the issue of hue scaling. Cicerone, Nagy, and Nerger (1987) proposed a partial model to explain "red" and "green" percepts of protanopes in the short-wavelength region. But this model fails to predict other features of dichromat hue scaling as described here and by Boynton and Scheibner (1967).

All previous models have the following general structure to describe the signals in the "red" vs "green", *rg*, and "blue" vs "yellow", *by*, opponent channels:

$$by = k_1 S - k_2 M - k_3 L, \tag{1}$$

$$rg = k_4 S - k_5 M + k_6 L, \tag{2}$$

where S, M and L (in *italics*) represent the excitations of S, M, and L cones, respectively; k_i are positive coefficients. Hassenstein (1968) and Guth et al. (1980) omitted the term $-k_2 M$ in the "blue"–"yellow" channel; De Valois and De Valois (1993) assume that this term has a positive sign. Certain discrepancies between the models' predictions and trichromatic experimental data had been noted, but the proposed nonlinear modifications concerned the "blue"–"yellow" channel (Larimer, Krantz, & Cicerone, 1975; Werner & Wooten, 1979) and their consequences for color appearance had not been considered.

Simulations of dichromatic vision (Brettel et al., 1997; Sharpe et al., 1999; Viénot et al., 1995) are typically based on a linear model. They are very useful in illustrating for trichromats the color discrimination abilities of dichromats. But from the experimental results, it has to be concluded that they do not convey the richness of color experience that dichromats enjoy and express.

3.3.2. Nonlinear processing as basis for dichromat color categories

We propose a new model for the clearly established capacity of dichromats to meaningfully use "red" and "green". The structure of our model is motivated by the observation that, while the dimensionality of the receptoral color space of dichromats is reduced, their reports reflect all perceptual aspects of trichromats. This suggests that the structure of perceptual color space is not in one-to-one correspondence with the dimensionality of the receptoral inputs, as pointed out by MacLeod (1985).

How could color categories similar to those of trichromats be derived from two receptor types? Linear combination of the two cone signals, as assumed in earlier models (see above) does not lead to sufficiently different spectral characteristics, regardless of the choice of cone weights. One reason for this constraint is that the long-wavelength tail of the S cone spectral sensitivity is virtually zero at the long-wavelength flank of the M cone, and likewise for the short-wavelength tail of the M cone at the short-wavelength flank of the S cone. Thus, varying cone weights in linear combinations can only affect the region between the peaks of the spectral sensitivities, but will have no substantial effect outside this region.

A plausible way to obtain a spectral response curve that is qualitatively different from that of the existing cones is to take into account nonlinearities in sensory processing. Even with relatively small deviations from linearity, by adequate combination of the signals, it is possible to achieve an effective spectral response curve that differs from the original spectral sensitivities as much as the third cone opsin in trichromacy does.

In the case of protanopes, we consider M cone signals, M, passed through a compressive nonlinearity φ,

$$\widehat{M} = \varphi(M). \tag{3}$$

The resulting spectral response curve will be broader than the original M cone response curve, yielding relatively higher responses at the tails, where response levels are low. Opponent processing leads to inhibition by S cones,

$$\widetilde{Q} = \lfloor \widehat{M} - \alpha S \rfloor_+, \tag{4}$$

where α is the scaling of S relative to $\widehat{M}$, and $\lfloor \ldots \rfloor_+$ denotes half-wave rectification. Thus, the short-wavelength tail of the $\widehat{M}$ response curve will be reduced, achieving a net effective spectral response $\widetilde{Q}$ that is shifted to longer wavelengths with respect to the original M curve (Fig. 4a). For deuteranopes, an analogous response curve can be obtained from L cones.

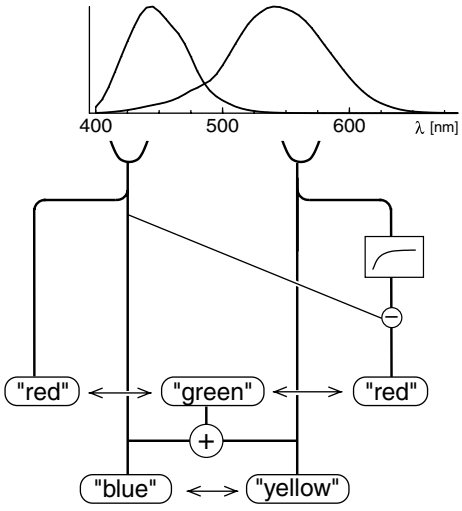

Fig. 3. Dichromat model. Simplified schematic of the proposed model for color vision of dichromats. Here the case of the protanope, with human S and M cone spectral sensitivities (top) is shown. A single input has been drawn for each of the parallel pathways originating in a cone type; whether an individual cone feeds into both pathways or only one of them is not specified in the model. The general architecture is similar to those of earlier models, except for the additional channel that combines nonlinearly transformed receptoral signals, thus achieving a representation of the visible spectrum by "blue", "yellow", "red", and "green". Half-wave rectification stages are omitted for simplicity.

With these considerations we do not mean to propose that the visual system of dichromats explicitly constructs a third type of signals. Rather, we present this as an intuitive mechanism for illustrative purposes. For the real visual system, it is conceivable that different degrees of nonlinearity across neurons yield a spread of effective spectral response curves. These could then be segregated during development by Hebbian-type mechanisms (Boycott & Wässle, 1999; Nathans, 1999), and thus be classified into different subpopulations.

The proposed third channel does not carry an independent color signal, and the color space remains two-dimensional. However, due to the nonlinear processing, subregions in this two-dimensional space can be defined that correspond to color categories similar to those of trichromats.

Several candidate nonlinearities to achieve such a pseudo-trichromatic representation can be found along the visual pathways. Variations in receptor pigment optical density have been suggested to subserve anomalous trichromacy in subjects with multiple copies of pigment genes with the same peak absorption wavelength (Neitz, Neitz, He, & Shevell, 1999). However, our main dichromat subjects had only a single pigment gene copy. Further nonlinear mechanisms are the contrast gain control in the parvocellular system (Kaplan & Shapley, 1986), or nonlinearities in On- and Off-pathways (Valberg, Lange-Malecki, & Seim, 1991). Finally, it

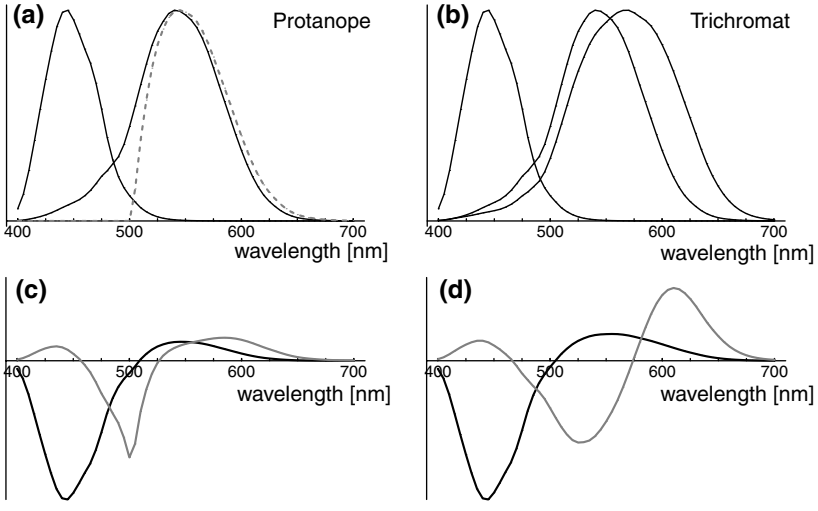

Fig. 4. Intermediate stages of the model. Spectral response functions at different stages of the dichromat model for the protanope case (left column). For comparison, corresponding curves for the trichromat case are shown (right column). (a) Spectral response functions of S and M cones (black solid lines) and the nonlinear third channel $\widetilde{Q}$ (Eq. (4)) of the dichromat model (gray dashed line). (b) Spectral response functions of S, M, and L cones of trichromats. (c) Opponent functions for protanopes obtained by linear combination of the curves in (a) (Eqs. (5) and (6)). Black: yellow–blue; Gray: red–green. (d) Opponent functions for trichromats. Rectification and scaling of the opponent functions (Eqs. (7) and (8)) yields the hue scaling curves (Fig. 5).

may even be conceivable that in dichromats, signals from the magnocellular pathway are exploited for color vision, in which case the nonlinearities would be fairly strong.

3.3.3. The model accounts for hue scaling results of dichromats

To derive hue scaling data from the pseudo-trichromatic signals, we use the type of color vision model that has proven successful to describe normal trichromatic vision (Eqs. (1) and (2)). Opponent signals corresponding to "blue" vs "yellow", *by*, and "red" vs "green", *rg*, are obtained by linear combinations of the pseudo-trichromatic signals (S, M, $\widetilde{Q}$),

$$by = \sigma_{by} S - (\mu_{by} M + \lambda_{by} \widetilde{Q}), \tag{5}$$

$$rg = \sigma_{rg} S - \mu_{rg} M + \lambda_{rg} \widetilde{Q}. \tag{6}$$

The hue valences, b, y, r, g, are represented by the positive or negative parts of the opponent signals, respectively,

$$b = \lfloor by \rfloor_+, \quad y = \lfloor -by \rfloor_+, \quad r = \lfloor rg \rfloor_+, \quad g = \lfloor -rg \rfloor_+. \tag{7}$$

Hue proportions r, g, b, y, as measured by hue scaling, correspond to the normalized signals,

$$\mathrm{r} = \frac{r}{r+g+b+y}, \quad \mathrm{g} = \frac{g}{r+g+b+y},$$
$$\mathrm{b} = \frac{b}{r+g+b+y}, \quad \mathrm{y} = \frac{y}{r+g+b+y}. \tag{8}$$

The structure of this model is equivalent to those of trichromat vision models of previous studies (e.g. De Valois & De Valois, 1993; Guth et al., 1980; Hassenstein, 1968; Hurvich, 1981; Ingling et al., 1996; Werner & Wooten, 1979), but is applied to the pseudo-trichromatic data derived from signals of S and M cones only. Fig. 3 illustrates the model architecture.

For the nonlinearity φ, we chose a simple power function

$$\varphi(s) = s^{\gamma} \tag{9}$$

with the parameter γ determining the degree of nonlinearity ($\gamma = 1$: linearity). As cone spectral sensitivities we used the estimates of Stockman and Sharpe (2000). Settings for the model parameters to qualitatively reproduce the experimental data are given in Table 2. To simulate the bright stimulus condition, the nonlinearity parameter γ was reduced by 10% and the gain in the *rg* channel was adjusted by a factor of 0.6 (Hurvich & Jameson, 1955; Judd, 1948). The plots in Fig. 5a and b show the model results for protanopes and deuteranopes, respectively. Our model accounts for all characteristic features of the dichromat hue scaling results.

Table 2
Parameter values used for the hue scaling simulations in Fig. 5

	γ	α	σ_{rg}	μ_{rg}	λ_{rg}	σ_{by}	μ_{by}	λ_{by}
P	1.15	5	1	8	8	5	0.35	0.35
D	1.3	5	1	7	10	5	0.8	0.8

P: protanope; D: deuteranope.

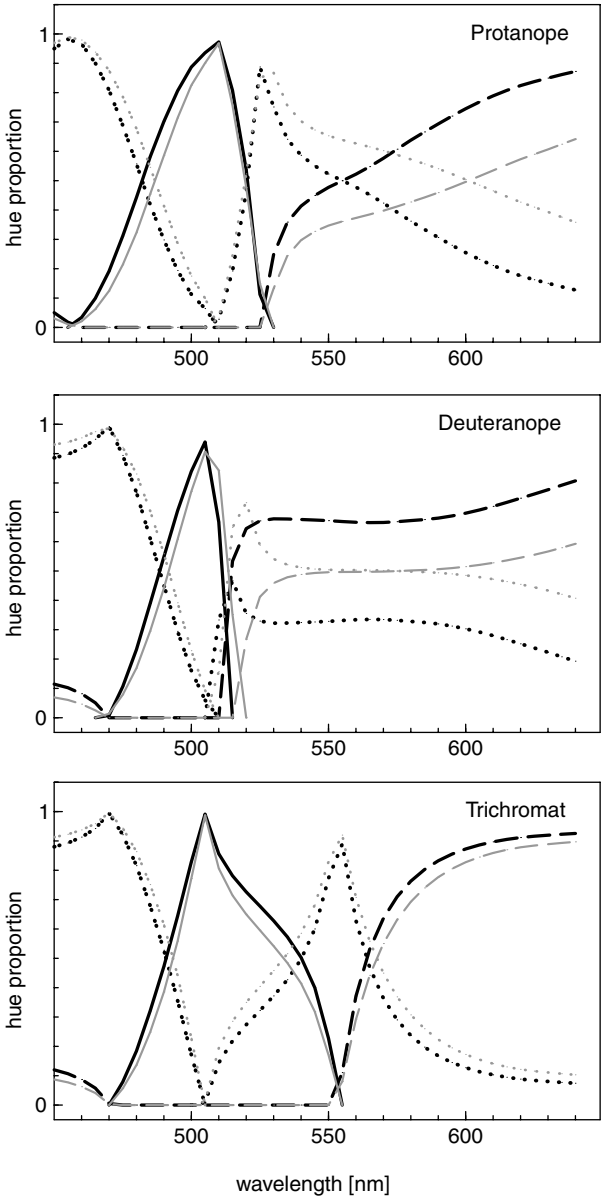

Fig. 5. Hue scaling model predictions. Top: protanopes; middle: deuteranopes; bottom: trichromats. Gray values and thickness of lines denote hues and luminance as in Fig. 2. The model accounts for all qualitative features specific for hue scaling of dichromats, including the relatively strong intensity dependence. The extended model (see text and Fig. 6) reproduces hue scaling results of trichromats (bottom).

3.3.4. Aspects of dichromat color vision accounted for by specific features of the model

Key feature of our model is a multi-stage architecture (De Valois & De Valois, 1993) with nonlinearities,

including half-wave rectification, at several stages. A consequence of this structure is that the net contributions of S and M cones to a hue percept are not fixed, as in linear models, but can be stronger or weaker, and positive or negative, depending on the contribution of the respective other cone. In the following, we consider various observed features of dichromat hue scaling, and identify the corresponding specific elements in our proposed model of sensory processing (Fig. 3). As above, we discuss the case of protanopes with S and M cones explicitly. The deuteranope case is analogous and obtained by using L cones instead of M cones.

The appearance of "red" and "green" besides "blue" and "yellow" from dichromatic input is a result of the assumption that the signals from M cones are branching into two separate paths with different transducer functions (see boxes in Fig. 3). In one channel, light absorbed is more or less linearly transmitted towards the "blue"/"yellow"-decision. The second path leads towards "red" through a steeper, but saturating function. This results in a competition between "red" and "yellow", predicting for longer wavelengths that at low light intensity "red" will appear; with increasing intensity, the second channel will saturate and the first one will take over towards "yellow".

Intensity dependence of perceived hue is also known from trichromatic vision. The so-called Bezold–Brücke effect has been explained in the context of color vision models by proposing a difference in gain control between the *by* and the *rg* opponent channels (Hurvich & Jameson, 1955; Judd, 1948). In our model, the long-wavelength path in addition exhibits gain control. Both effects combine, yielding the stronger intensity dependence in dichromats.

Another striking feature of dichromat hue scaling is the consistent appearance of a sharp peak of "green" around 510 nm. This spectral region corresponds to excitation of both S and M cones. The model accounts for both the spectral position and the more limited spectral range of "green" in dichromats (see below). That "green" originates from both M and S responses would be consistent with the following observations of Hemminger and Georgi (1982). In hue scaling data of deuteranomalous subjects, the short-wavelength slope of "green" was not shifted towards longer wavelength as compared to the data of normals. Such a shift would be expected if only M signaled "green". The slope towards "red", around 600 nm, however, was found to be shifted in deuteranomalous vision.

"Red" and "green" both arise from S and M cone signals. However, unlike "green", which appears when both S and M cone responses are present, "red" appears if either of them alone is present. Our model assumes cross-inhibition between the signals from M and S cones, but there is no internal inhibition within S or M cone signals. Thus, the short as well as the long wavelength ends maintain their contribution to "red", the overlap zone will be "green".

Finally, our results and the model are in line with the findings of Jameson, Highnote, and Wasserman (2001) who studied color categories in heterozygous females. Subjects were asked to divide the visible spectrum by color appearance. The four protanopes divided the spectrum into 5.3 segments on average, compared to 7.3 for normal trichromats. The five or more bands seen by the dichromats would be unlikely with only "blue"–"gray"–"yellow" as often assumed (e.g. Viénot et al., 1995); from our model, however, "violet", "blue", "green", "yellow", "orange", and "red" might be expected, a number of colors that would be in accordance with the experimental results.

3.4. Trichromat model

Our model implicitly assumes a mechanism of neural plasticity which, during development, wires together neurons carrying similar signals and separates neurons with more dissimilar signals, thus segregating the cone signals according to the degree of nonlinearity into different pathways. Under this assumption, a single further step is sufficient to achieve trichromacy, namely a gene duplication with differentiation producing separate M and L cone pigments. It is well established that one photoreceptor cell, in an all-or-none control of gene expression, receives exclusively one type of opsin, never a mix (Wang et al., 1999). Different receptor cells—M and L—are probably defined by their pigments only (Smallwood, Wang, & Nathans, 2002). Nathans (1999) and Smallwood et al. (2002) provide evidence for a random filling mechanism, without an M cell/L cell pre-determination before opsin synthesis.

Molecular genetics places the X-chromosomal opsin gene duplication between New World monkeys and Old World primates, 30–40 million years ago (Nathans, 1999; Smallwood et al., 2002). Which of the modern X-chromosomal opsin genes is more closely related to the ancestral gene in dichromat monkeys is a matter of debate. Boissinot et al. (1998) favor M, while Nei, Zhang, and Yokoyama (1997) prefer L. We develop our argument starting with M; however the model and the proposed evolutionary scheme are equally valid if L was the ancestral gene.

Introducing a new opsin gene with different spectral sensitivity in our model amounts to duplicating the pathways from M cones (see Fig. 6). Since signals from different cone types will tend to differ, the proposed developmental learning mechanism would segregate the signals according to cone type. With such a mechanism, the cone type sensitive at the long-wavelength end of the visible spectrum (L) would most likely become associated with the higher-gain pathway associated with "red". In this spectral region, L cone signals may occur

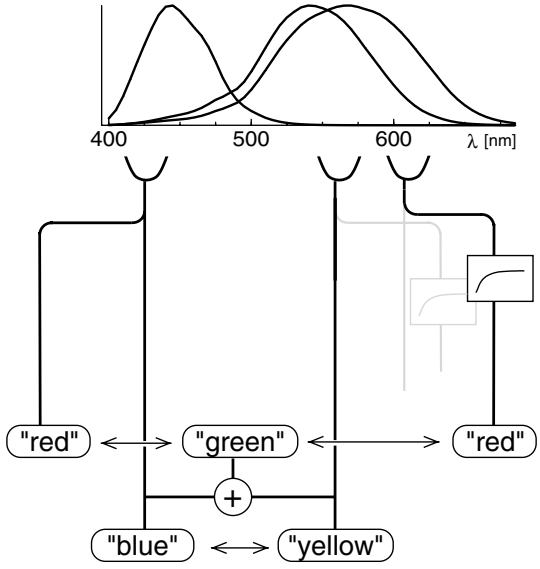

Fig. 6. Trichromat model. The model for trichromatic vision is derived from the dichromat model by assuming the occurrence of a third cone opsin. Thus, the channels from the longer-wavelength receptor are duplicated with the respective spectral sensitivity. Cone-type specific weakening of connections (gray lines) by proposed mechanisms of neural plasticity could lead to segregation of cone signals into pathways towards "red" from one receptor and towards "yellow" from the other.

without any substantial M or S cone signals. In these cases, however, even the L cone signal will be low, due to the low spectral sensitivity, and thus only the higher-gain pathway will be active together with the L cones. Thus, the L cone signals will supersede the signals in the nonlinear pathway $\widetilde{Q}$ (see above).

By introducing a third cone type in our dichromat model, a model for trichromatic color vision is obtained which correctly predicts the hue naming and scaling of normal subjects, as can be seen by comparing Fig. 5 (bottom) with the data of Boynton and Scheibner (1967) and Hemminger and Georgi (1982).

4. Discussion

Hue scaling results of protanopes and deuteranopes, qualitatively similar to those shown here (Fig. 2), have previously been reported by Boynton and Scheibner (1967) and Scheibner and Boynton (1968). All subjects of their study used four color terms. Furthermore, an intensity dependence of "red" vs "yellow", to an extent comparable to that of our study, was clearly documented. Thus, our results confirm and extend their measurements by the use of molecular techniques to demonstrate that our subjects had only one M or L cone opsin, respectively (Fig. 1; Table 1).

Scheibner and Boynton (1968) explained their data by assuming the existence of a third opsin. In the study by Sharpe et al. (1998), a number of dichromats, defined by Raleigh matches, were tested for X-chromosomal opsin genes: 13 of 34 protanopes had only one M opsin, and 28 of 57 deuteranopes had only one L opsin; the other dichromats carried more complex arrangements. Furthermore, Ueyama et al. (2003) report that, of 102 deuteranopes, 76 had an array consisting of a single L opsin gene. From these results, it would be highly unlikely ($p < 0.0015$) if not at least one of the subjects in the Scheiber and Boynton studies had been a carrier of a single X-chromosomal opsin gene.

Our confidence that dichromats report percepts corresponding to those of normal trichromats is based on the consistent hue scaling with "red" in the "blue" at very short wavelengths where M and L should hardly contribute. "Green" as the remaining primary color percept was used by the dichromats in a plausible manner. Furthermore, as mentioned before, heterozygous females report "red" with stimulation of their dichromat mosaic areas (Born et al., 1976).

In two of our subjects, we found occasional variations in the naming of "red" and "green" at short wavelengths. This indicates that these color categories in dichromats are not as robust as in trichromats. Our model is not incompatible with the occurrence of such variability under certain conditions. Both cone types are assumed to support, via different channels, both "red" and "green" percepts (Eqs. (1)–(8), Fig. 3). Depending on the balance of signal strengths in the different channels and the magnitude of noise, variability in the final output may occur under certain conditions. To investigate this issue systematically would require appropriate modeling of noise, which was not considered in the present study.

Could the observed results reflect an acquired cognitive strategy of dichromats to deal with the trichromatic color language? Our two main subjects and further dichromats we interviewed informally, as well as dichromats reported in the literature, claim that "red" and "green" constitute unique percepts, qualitatively different from "blue" and "yellow". Both dichromats and trichromats have to learn color naming of their percepts. We propose that dichromats possess percepts corresponding to those of trichromats and that the color names learned constitute not a perfect, but an *acceptable* match of their percepts with those of trichromats.

Smith and Pokorny (1977) found that dichromats exhibit trichromacy with stimulus sizes above 4°. We, as well as Scheibner and Boynton (1968), used smaller test fields, and therefore large-field trichromacy can be ruled out as an explanation of our results.

The possibility of a contribution of rods to the color percepts of dichromats in our experiments, although unlikely, cannot be excluded. Published evidence is contradictory, Montag and Boynton (1987) and Nagy and Boynton (1979) argue in favor of rod involvement,

Scheibner and Boynton (1968) and Crognale, Teller, Yamaguchi, Motulsky, and Deeb (1999) against it. Our model, however, predicts the data extremely well even without the assumption of rod interference.

Neitz et al. (1999) found evidence for a kind of trichromacy in dichromats caused by optical density differences in cones with opsins of the same type from different X-chromosomal genes. Our main subjects had only one X-chromosomal opsin gene. Therefore, we can exclude this specific mechanism in these subjects. Nevertheless, differences in the spectral responses of cones with identical opsins might exist for other reasons and could contribute to the proposed mechanisms.

Cicerone et al. (1987) reported that protanopes but not deuteranopes made "red" vs "green" judgments in the short-wavelength part of the spectrum. They argue from the plausible assumption that S cones signal towards "red", and that in protanopes this "red" from S should not be eliminated by the mutation. Cicerone et al. (1987) also report—and our findings agree with their observation—that protanopes use "green" around the neutral point. However, their report of deuteranopes not using "green" is in disagreement with results by others. The data of Boynton and Scheibner (1967), Scheibner and Boynton (1968), and our data consistently show that protanopes and deuteranopes both report "green" in the wavelength region around 500 nm. Secondly, the model of Cicerone et al. (1987) does not explain hue naming of protanopes or deuteranopes at wavelengths above 550 nm. Thirdly, the assumption of a difference of M and L cones *before* filling with M or L opsin seems implausible in view of recent evidence from molecular genetics (Nathans, 1999; Smallwood et al., 2002).

The main assumption of our scheme is parallel processing from the cones via two different channels, one with higher gain, saturating earlier, the other one with lower gain and more linear response. Further, our model implies that S and M cones signal towards "green", and assumes inhibition between the short and the long-wavelength path towards "red".

In our model, receptors are assumed to respond in a linear way to light absorption. Experimental evidence indicates that a logarithmic-like response characteristic may be more adequate (Chaparro, Stromeyer, Chen, & Kronauer, 1995). For our model, there would be no qualitative difference between these cases. The results do not depend on the assumption of linearity vs nonlinearity, but rather on differences in nonlinearities.

A multi-stage computation was discussed in detail by De Valois and colleagues (De Valois & De Valois, 1993; De Valois, De Valois, Switkes, & Mahon, 1997). In this work, the multi-stage processing was considered to obtain estimates for the relative contributions of the cone types. The model was linear, and therefore was equivalent to a one-stage model. Our model contains several stages with nonlinearities, but is otherwise similar in structure to the model by De Valois and De Valois (1993). While this may suggest correspondences to certain stages in the visual system, we do not make strong assumptions about the loci of the different mechanisms. It would not be implausible to identify the first opponent stage of our model with processing in the retina or lateral geniculate nucleus. The splitting into parallel channels may occur at peripheral stages, or at early cortical stages, where intermediate representations exist (De Valois, Cottaris, Elfar, Mahon, & Wilson, 2000; Wachtler, Sejnowski, & Albright, 2003), which are not considered explicitly in our model. The percepts have to be assumed to arise at higher cortical stages (Bartels & Zeki, 2000; Rüttiger et al., 1999).

In a number of papers, Valberg and colleagues (Lee, Valberg, Tigwell, & Tryti, 1987; Valberg, Lee, & Tryti, 1987; Valberg, Seim, Lee, & Tryti, 1986) investigated responses of parvocellular neurons in the lateral geniculate nucleus. They found that differences in nonlinearities were responsible for the specific response properties of On- and Off-center opponent cells. In particular, they were able to account for specific aspects of trichromatic vision, such as the Bezold–Brücke effect (Valberg et al., 1991). These findings can be taken as strong support for the main assumption underlying our model, since the existence of parallel processing pathways with different degrees of nonlinearity was clearly demonstrated. Furthermore, these studies emphasize the dissociation between early neural opponent responses and perceptual opponency (Valberg, 2001).

We did not attempt to quantitatively fit the model parameters to the data. Reasonable choices of values already yield remarkably good qualitative fits, and due to the number of parameters and the multi-stage architecture, numeric fitting procedures did not converge robustly. Quantitative modeling would be more promising in the context of systematic mapping of the color space of dichromats, an approach to be pursued in future work.

The dichromat model required the assumption of nonlinearities; these are highly conspicuous in dichromats, but exist also in trichromatic vision. The "red"–"yellow" and "red"–"blue" intensity dependence in dichromats is reminiscent of the Bezold–Brücke effect described for trichromats (Boynton & Gordon, 1965). Recently, Kremers, Stepien, Scholl, and Saito (2003) reported significant residual sensitivity in dichromats for stimuli designed to isolate their respective missing cone type when subjects were adapted to red light. This may indicate that the technique of cone isolation fails for certain adaptation conditions, which might reflect early nonlinear interactions in color processing.

Our model relies further on the assumption of developmental plasticity, strengthening or weakening

connections between neurons according to the statistics of their signals. Such epigenetic focussing has been suggested in several instances, e.g., for orientation selectivity of cortical neurons (Blakemore & Cooper, 1970; Blakemore & van Sluyters, 1975). Evidence for developmental plasticity is found in primate and human color vision (Brenner, Schelvis, & Nuboer, 1985; Brenner, Cornelissen, & Nuboer, 1990; Crognale, 2002; Teller, 1997), and has been specifically proposed for the segregation of cone signals in trichromats (Boycott & Wässle, 1999), as well as in heterozygous female dichromat monkeys (Mollon, 1989) and humans (Jordan & Mollon, 1993). Presumably, such mechanisms were already present in our dichromatic simian ancestors. New world monkeys have been shown to make use of trichromacy acquired by pigment gene polymorphism (Tovée, Bowmaker, & Mollon, 1992). These abilities do not seem to be associated with anatomical differences (Solomon, 2002), which speaks in favor of changes at the synaptic level. The statistics of color signals in the natural environment may support learning of consistent color categories (Shepard, 1992b; Yendrikhovskij, 2001) even in dichromats. With appearance of the third cone type, these mechanisms could segregate M and L cone signals and thus yield color-selective processing for trichromatic vision (Doi, Inui, Lee, Wachtler, & Sejnowski, 2003).

Obviously, pseudo-trichromatic processing as described by our model would not yield any benefits in terms of color discrimination, as necessary for finding fruit in foliage (Osorio & Vorobyev, 1996). However, it constitutes a mechanism to establish a relatively large number of categories within a lower-dimensional signal space (Lehky & Sejnowski, 1999). Such categorization, if in reasonable accordance with surface categories in the environment, would be beneficial in terms of perceptual scene segmentation and object recognition.

Acknowledgments

We thank Ad Aertsen, José Campos-Ortega, Stefan Heyl, Wolfgang Michalke, Stefan Rotter, Gerd Stefan, Gerd Scherer, Dieter Schmidt, and Klaus Vogt, for providing means, equipment, advice and criticism. We are grateful to Tobias Erb and Ramona Pop for technical assistance, and to the anonymous reviewers for valuable comments and suggestions. We thank our dichromat subjects for their invaluable contribution, their patience, and their sense of humor.

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