Receptoral Constraints on Colour Appearance

D.I.A. MacLeod

Psychology Department, University of California at San Diego, La Jolla, California 92093, USA

Goethe wrote: "All theory is gray," and his complaint is particularly applicable to studies of the earliest processes in color vision - the events at the receptor level. The phenomena of color vision that impress us most are seldom traceable to receptor behaviour. But still it is important to be clear about the ways receptors constrain sensation and the ways they don't. Most obviously, the receptors are the sole source of the information on which color vision depends. Radiation that fails to stimulate the receptors can't be seen; and stimuli that are equal to one another in their effects on the receptors have to appear identical if viewed under similar conditions. We do tend to forget this, because we generally know the stimuli only through our own visual reactions to them; but, when we neglect the physical diversity of color stimuli in this way we can be seriously misled. For instance, we may fail to appreciate the basic point that surfaces that are visually indistinguishable under one source of illumination will in general appear different from one another when the illuminant is changed. This type of deviation from perfect color constancy (which Dr. Land has also noted in his presentation) has a lot of aesthetic and commercial importance, and is an inevitable consequence of what Rushton (1971) called the Principle of Univariance, that the signal from a receptor depends only on its state of excitation, and not on the wavelengths of the exciting stimulus. Since any account of color appearance, under constant or varying conditions of illumination, must start by taking account of how much the receptors are excited by different wavelengths, I begin by very briefly reviewing our knowledge about that, then go on to a closely related issue: the physiological basis of trichromacy. Trichromacy, the fact that three independent adjustments are necessary and sufficient for matching any color, is thought to be a consequence of the existence of only 3 cone types whose excitations have to be equated for a visual match. Although there can be no argument with the proposition that 3 classes of cone exist in normal retinas, I am not satisfied with this account of trichromacy. I will show a clear counter-

example leading to the conclusion that trichromacy has its origin in a trivariance of neural organization. Finally, I describe briefly some experiments that bear on the contribution that receptor adaptation makes to color constancy.

First, then, as to the spectral characteristics of the receptors, it is enough for our present purposes to recognize that they are pretty much what Konig believed them to be nearly a hundred years ago. There are 3 cone types, with distinct but extensively overlapping spectral sensitivities. These broad spectra carry some penalty for color discrimination with natural broadband stimuli, but they are essential for retaining good visual sensitivity since if individual cones absorbed light in only a narrow spectral band, all of the incident quanta belonging to other spectral bands would inevitably be wasted.

Each cone generates a signal depending on its own excitation, and roughly speaking we can associate intensity with the sum of the three cone excitations, and chromaticity with their ratios. The receptoral basis of color vision is best appreciated graphically by using Cartesian coordinates to represent the excitations of each of the three cone types by a given stimulus as in Figure 1. The letters R, G and B are used here to index the axes corresponding to the 3 cone types; these of course stand for red, green and blue which are, roughly speaking, the parts of the spectrum that most selectively excite each cone type. To represent chromaticity independently of luminance, it's convenient to adopt a plane from this space as a 2 dimensional chromaticity diagram, for example, the unit plane R+G+B=1. This is the relatively clear Maxwell triangle, not the physiologically arbitrary CIE projection of it, which has held back insight into the visual process to such an extent that William Rushton was fond of saying that "it seems to have been designed to intimidate the young and bewilder the old." Still it is not quite the sort of diagram one would like to have for representing the relative cone excitations independently of luminance. There is evidence (Eisner and MacLeod, 1980) that the B cones make no detectable contribution to luminance as usually defined, so that in the 3D cone excitation space the constant luminance plane is not the oblique one, R+G+B=1 but the vertical one in Figure 1, R+G=1. If we adopt this plane as our chromaticity diagram (Luther, 1927), the excitations of all the cones for equiluminous stimuli can be read from linear orthogonal coordinates (MacLeod and Boynton, 1979). In Figure 2 the horizontal coordinate, r, shows R cone excitation, and if read from right to left, it shows G cone excitation. B cone excitation is the vertical axis, and the spectrum locus extends from violet at the top, down to green, through yellow to red on the right.

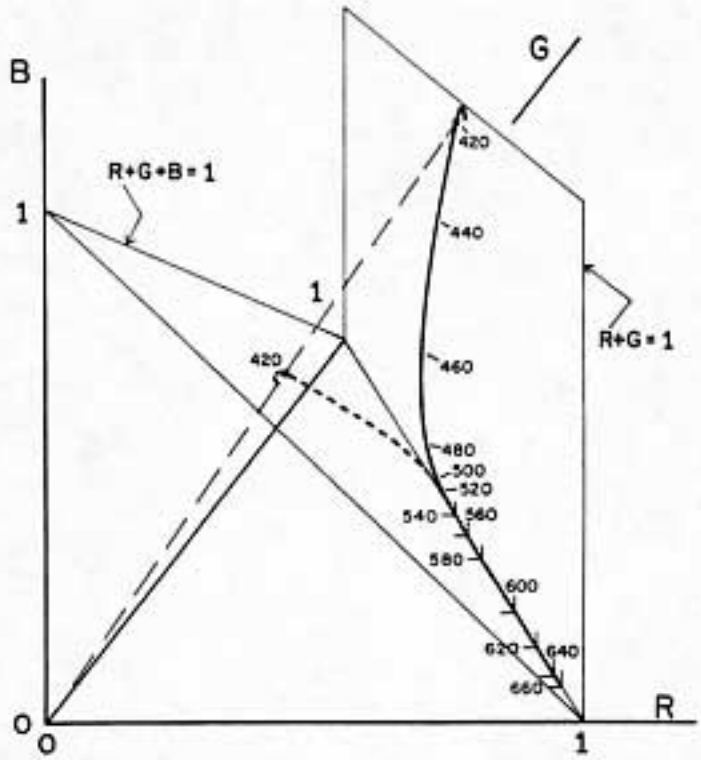

Fig. 1. Three dimensional cone excitation space, showing planes corresponding to triangular (R+G+B = 1) and rectangular (R+G=1) chromaticity diagrams.

In this cone excitation diagram, the spectrum locus never gets into a corner - there are no pure stimuli because of the spectral overlap of the receptor sensitivities. But if we were to normalize to white, the ratio of R cone to G cone excitation reaches a maximum of about 18 to 1 at 700 nm; the reciprocal ratio is greatest below 500 nm, but never gets higher than 2:1. That maximum is actually in the blue, around 465 nm. B cone excitation is rather slight for a typical white, and is more than 50 times greater in the violet. This very strong peak in the violet occurs because the spectral separation between the B cones and the other two is relatively large with maximum values of roughly 440, 540 and 560 nm. This arrangement gives the B cones a lot of leverage for color discrimination, but puts them at a disadvantage for catching quanta. Why is this better than equal spacing? It has been pointed out that chromatic aberration introduces a tradeoff between the demands of color discrimination (which is aided by spectral separation) and spatial resolution, which is hindered by it, most especially in the short wavelength spectral range. Bob Boynton (1980) has shown that spatial resolution is improved by the solution that the visual system seems to have adopted, of

having the R and G cone spectral sensitivities closely spaced at long wavelengths and entrusting the task of spatial resolution entirely to them. This dependence of spatial resolution on the R and G cones is assured by excluding the B cones from the luminance system, inasmuch as it is only luminance variations that are analyzed with high spatial precision by the visual system (Granger and Heurtley, 1973). A fly in the ointment here is that physiology has not exposed the hypothetical luminance system, devoid of any B cone input, which psychophysicists like to postulate; but psychophysics does require it somehow, somewhere, and we must just wait for physiologists to find it. An incidental advantage of this arrangement whereby B cones have no role in spatial resolution, is that it allows a great economy in their numbers. David Williams, Mary Hayhoe and I (1981) got psychophysical evidence for this by mapping B cone sensitivity against long wavelength backgrounds. This revealed quite large variations in sensitivity on a very small spatial scale, the variation amounting to as much as a factor of 4 in 6 minutes of arc. We presented evidence that the peaks of sensitivity are just the regions in the visual field where B cones exist, and the insensitive valleys are the large gaps between them, filled with R and G cones.

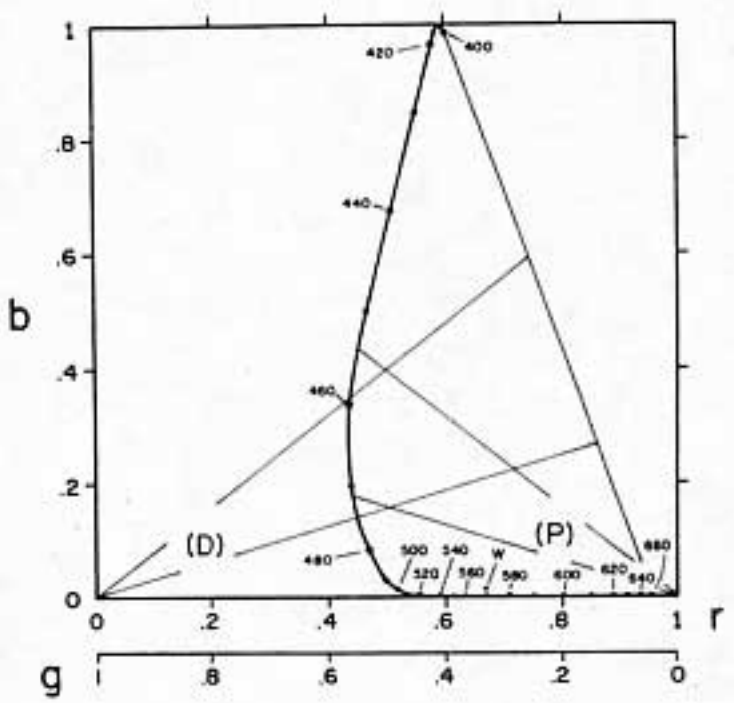

Fig. 2. Cone excitation chromaticity diagram (equivalent to the R and G =1 plane i Fig. 1). P and D indicate the confusion lines of protanopes and denteranopes, and the point W near the horizontal axis is the chromaticity of an (equal energy) white.

It is useful to consider further the fact that colors that have flattish spectra ("whites") do look subjectively neutral even though they provide only a tiny fraction of the B cone excitation available from spectral light. It is probably very useful for the visual system to have the balance between blueness and yellowness set up in this way, because physically pure blues and violets are rare in nature. Most natural stimuli are spectrally fairly flat, and a moderate skewing of energy toward long or short wavelengths is the most we are usually called on to respond to. So it is a good strategy to have the neutral point set to correspond to a fairly flat spectral reflectance, so that small deviatons from spectral flatness can be efficiently discriminated by allocating most of the response range of the color opponent cells to desaturated colors, even at the expense of discriminating power for saturated blues or yellows. Ed Pugh's contribution to this symposium gives clear evidence that nonlinearity of this kind is a prominent feature of color-opponent processing in the visual system.

One final point is made clear by this cone excitation diagram, a familiar one that was recognized by G. E. Muller in 1924. Redness and greenness are not simply correlated with excitation of the R and G cone types, that is with $\underline{r}$. In the diagram, greenish yellows have the same $\underline{r}$ coordinates as reddish violets, and if we go from blue to yellow, keeping subjective redness and greenness zero, the R and G cones must undergo a very substantial excitation modulation, about 20%. The B cones are therefore strongly implicated in the production of redness at short wavelengths, although the nonmonotonic variation of $\underline{r}$ with wavelength in the violet-green spectral range is also a factor.

TRICHROMATIC MATCHING AND COLOR APPEARANCE

Now let us concentrate on the issue of how the cone photoreceptors constrain our sensations of color. To begin with, of course, it is usually supposed, and for good reason, that the cones impose the visual constraints implicit in color matching. Cones seem to produce only one type of signal whatever the light that excites them, an idea that William Rushton (1971) called the Principle of Univariance. Two lights of different spectral composition can be matched for any cone receptor by intensity adjustments alone, so that the two stimuli are indistinguishably represented at the cone output. With 3 cone types, each containing a different pigment, a trichromatic match is possible such that the two matched stimuli are equivalent to one another in their action on each of the 3 cone types. In this situation, no information distinguishing between those stimuli leaves the cones, and the match established at the cone level must be a subjective match as well, whatever the neural processes that intervene between the receptors and sensation. Although I think this intuition about the receptoral basis of color matching is perhaps the most

fundamental and basic one in the whole field of color vision, I want to suggest it is at best a crude idealization and is, in some cases at least, fundamentally wrong.

Trichromatic Additivity and Homogeneity of the Cone Sensitivities

A most important aspect of trichromatic matches that supports Young's hypothesis that they are determined at the cones is the additivity of lights of different spectral composition. According to Grasmann's Third Law (and a related principle known as von Kries's Persistence Law) two matching lights of different spectral composition continue to match whatever additional light is exposed along with them. (Of course, the added light has to be the same on the two halves of the matching field.) This is a striking fact because as von Kries knew the actual apparent color of both the matched fields can be quite drastically changed by uniform adapting lights like this. The interpretation is that the receptor signals from the three cone types are selectively attenuated by variously colored background lights, but the equality of excitation between the 2 halves of the matching field continues to hold for each cone type, so the match still holds. If the matched fields were differently exciting the cones, and only became equivalent at some later neural stage, this addivitity or persistence of matches during adaptation would not generally be expected. Grassmann's Law stating that it does is therefore a cornerstone of the trichromatic theory.

Now we know that there are 3 cone types, but still I think this account of trichromacy is inadequate. One problem is posed by inevitable variations in spectral sensitivity between cones of the same class. Between individuals, we have evidence, from a factor analysis of color matching data (MacLeod and Webster, 1983) that cones of the same class vary in their wavelengths of peak absorption with a standard deviation very close to 1 nm. This is not much but it is enough to ensure that in general when a trichromatic match is being made, cones with this degree of variability will collectively distinguish between the matched lights - for instance the long wave displaced cones will be more excited by the half of the matching field that has red in it. The difference between the two matched fields as viewed by a single cone can exceed 10%, which is a difference that is in general easily detectable. So why do we accept trichromatic matches? One answer could be that cones of the same class in a single individual's retina are less variable than I have assumed. But microspectrophotometry does not support this, suggesting instead standard deviation substantially greater than 1 nm (Dartnall, Bowmaker and Mollon, 1983). Some psychophysical evidence about variability among the cones of an individual observer is available in the additivity of trichromatic matches. Nagy, Heyneman, Eisner and I (1981) failed to find any reliable deviations from additivity in normal subjects when suitably chosen colored backgrounds were

added to the matching fields, an observation difficult to reconcile with the microspectrophotometric evidence since our calculations indicated that variability on the scale suggested by MSP would lead to easily measured failures of additivity, although variability with a standard deviation less than 1.5 nm would not. Despite these uncertainties the evidence for variability among cones is enough to raise serious doubt as to whether trichromatic matches are determined at the receptor level: the cones probably are distinguishing reliably between the matched fields.

Additivity Failure in Heterozggotes: Neural Trivariance

Fortunately, however, we have found perfectly clear violations of Grassmann's Laws in a particular, not uncommon group of subjects (Nagy, MacLeod, Heyneman and Eisner, 1981). For these subjects we can therefore say for certain that trichromatic matches are not established at the cones. These subjects are women who carry genes for anomalies of color vision. People with anomalous color vision make abnormal trichromatic matches because one of their pigments, either the R or the G pigment, has been replaced by an abnormal one. Lights that match for the normal pigments will generally be a mismatch for the anomalous pigment, and this pigment will dictate some different match. These cone pigments are produced by genes on the X chromosome, and the heterozggous carriers who do not manifest the anomaly presumably have one X chromosome making the normal pair of pigments and another where a mutated gene makes an anomalous pigment instead of one of the normal ones. Moreover, we know (from evidence recently reviewed by Luzzatto and Gartler, 1983) that in each cell of a mammalian carrier's body one or another of her two X chromosomes is dominant; the result is a mosaic of cells with characteristics given by her maternal or paternal X chromosome. For the carriers of anomalous vision the retina should have a mosaic of 4 cone types each with its own pigment. If these women use the information from their cones they must reject most trichromatic matches and only be satisfied with 4 variable, tetrachromatic matches. We find, however, that these carriers are always able to make a trichromatic match that satisfies them. However, we have discovered that for at least some of these women Grassmann's Laws do not hold. If we ask for a trichromatic match, and then add the same uniform background light to both sides of the matching field, the two sides of the field often become clearly distinguishable and the match has to be changed to a different setting (Fig. 3). The interpretation here is that in these carriers' retinas signals from normal cones and their anomalous replacements travel the same postreceptoral pathway and so become averaged, and that colored backgrounds selectively attenuate the signals from the normal or anomalous cones, whichever type is more sensitive to the background. The changes in the match follow the pattern to be expected on this hypothesis.

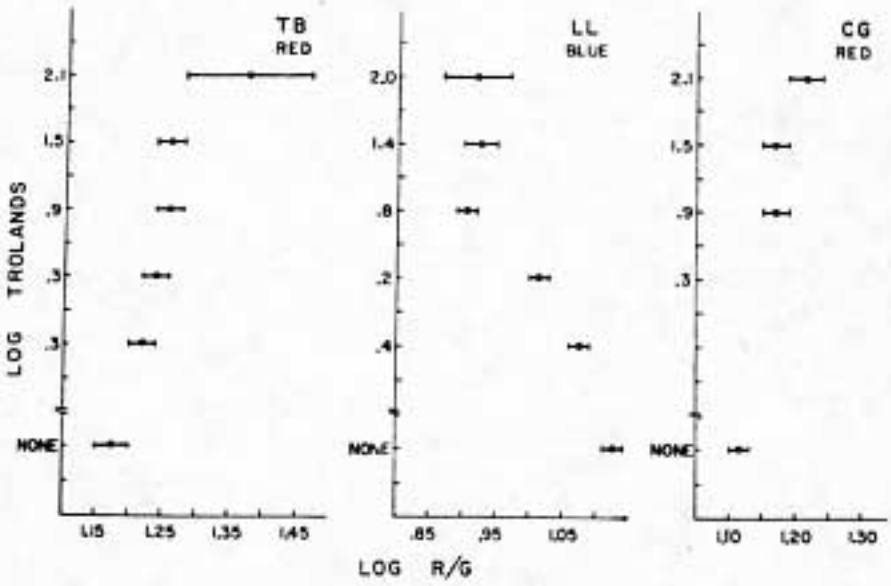

Fig. 3. Failure of Grassmann's Third Law (of additivity): the ratio of red (660 nm) to green (546 nm) primaries required in a mixture to match a 588 nm yellow (horizontal axis) is altered by red (670 nm) or blue (455 nm) backgrounds of various intensities in three women who were carriers of an abnormality of color vision.

The trichromatic vision of these women is therefore not the result of their having only 3 cone pigments but must be due to a trivariance of postreceptoral neural organization. Such a neural trivariance must be characteristic of normal visual systems too since neither the availability of an additional receptor type (the rods), nor variation in spectral sensitivity among cones of the same class, seems able to disrupt the trichromacy of steady-state normal visual matching. Although this neural trivariance must be recognized, a precise statement of what is implied by the concept involves difficult logical problems. To begin with, although it would be sufficient for neural trivariance if the receptors fell into only 3 classes on the basis of their central connections (as I have assumed here in the case of the anomalous carriers), that condition is not necessary and is doubtless not satisfied in the case of rod intrusion. It is more accurate to require that at some postreceptoral stage the various neural responses are dependent on only three variables which in turn are derived from a generally larger number of receptor signal variables. The simplest way for this situation to arise would be to have the three variables represented by univariant signals at some stage of processing. But even the most orderly physiological results

(Derrington, Lennie and Krauskopf, 1983) do not suggest an organization as tidy as this would require. Instead, postreceptoral variability from cell to cell is greater than at the receptor level. So long as this variability is present, it seems to me that we still do not have a satisfactory physiological basis for trichromacy; but we can say that under some conditions at least trichromacy rests on a neural (and not a receptoral) trivariance.

From Matching to Appearance

These reservations notwithstanding, it is agreed that for most observers under purely photopic conditions trichromatic matches are at least approximately determined by receptor constraints and will be practically unaffected by the vagaries of postreceptoral processing. But what about the appearance of the matched lights? To what extent do the receptors put constraints on color appearance? The short answer is, I think, not at all. If we think about this in physiological terms, we must realize that a visual neuron connected to 2 different cones with different pigments is logically free to exhibit any spectral sensitivity whatsoever (so long as the ratio of the quantum catches of the 2 cones is monotonic with wavelength). If 3 such neurons encode 3 dimensions of apparent color, we can say nothing at all a priori about how the firing rates or color sensations will depend on the cone quantum catches.

Even for a uniform field of light in the dark, color does not seem to be a simple fixed function of receptor excitations. One example: Rushton and Baker (1964) found that people vary substantially in their relative sensitivity to red and green and that this variation is correlated with their endowment of R and G cones as measured by retinal densitometry. Yet all such people see yellow at about the same point in the spectrum, with a far greater degree of consensus than Rushton and Baker's receptor measurements would lead one to expect (Rubin, 1961), suggesting that individual differences in neural organization induced genetically or (more likely) by experience compensate for the differences at the receptor level. Another example of this is the sensations of the unilaterally color blind. If color blindness in these cases is due simply to a change in the R or G visual pigments, it can be shown that the gamut of sensations they experience must be restricted to a vertical line in the ($\underline{r}$, $\underline{b}$) chromaticity diagram, running for instance from yellow to violet. This is not observed (MacLeod and Lennie, 1976), so an abnormality in neural organization must be modifying the appearance of colors, in addition to the influence of the pigment switch.

The Contribution of Receptors to Color Constancy

Color constancy is generally viewed as the prime example of a failure of color experience to correlate with receptor signals. I

do not wish to argue that postreceptoral processes have no role in color constancy but I do think it is instructive to consider what is probably the simplest possible model for constancy, a model where constancy is essentially the work of the cones themselves.

William Rushton, during his last years, was very interested in the relationship between visual adaptation and color appearance. He had a model for adaptation in the rod adaptation pool and in the cone receptors, which actually implies color constancy, as well as the fading of stabilized retinal images which is what he was most interested in. In Rushton's adaptation model (never fully published, but see Rushton, 1972), a cone under an illumination of time average intensity $\underline{I}$ adopts a steady state sensitivity inversely proportional to $\underline{I}$ so that the output (intensity X sensitivity) is independent of $\underline{I}$. This, of course, immediately accounts for the fading of retinally stable images. When images are not stabilized each cone type will see temporal transients equal to the difference of excitation across the unstabilized contour. The signal produced will be proportional to the edge contrast as seen by that cone type. This is exactly what we need as the first step in implementing a Land type model (Land, this symposium) for color constancy.

Rushton (1972) got evidence that something like this does go on in cone vision in an experiment where he allowed a stabilized image of a bipartite field, with 2 half circles of different intensity $\underline{I}_1$ & $\underline{J}_1$ to fade into a uniform circle or disappear totally. Then he abruptly changed the intensities in the two halves of the field independently. In general, the field would now look non-uniform, but it could be kept uniform if the temporal intensity ratio (or temporal contrast, if you like) was made the same on the 2 sides of the field.

This experiment shows that adaptation to stable images introduces a reciprocal modulation of visual sensitivity, but it says nothing about the location where that change occurs. In fact, a series of many neural stages could be involved. I did some experiments to investigate this point by seeing whether the fading of stabilized images might occur independently in the rod and cone systems. If it does this would suggest that the processes concerned operate before the retinal ganglion cell level, where rod and cone signals come together. Like Rushton I used the Yarbus technique for image stabilization, with a suction cap contact lens stuck to the eye. Apparatus details aren't important except that the equipment provided two superimposed stabilized or unstabilized beams, one of short wavelength light for rods and the other of long wavelength for the cones. The essence of the results is most easily seen by considering one particular case: a bipartite field seen by rods, with the two halves different in intensity in a ratio of 4 to 1, was allowed to fade, and on it was superimposed a physically uniform flashed or unstabilized test

field seen by cones. The key observation is simply that now with the transient test stimulus going through a different set of receptors than the stabilized background, the test stimulus that was physically uniform did appear subjectively completely uniform. The need for proportioning the intensities on the 2 halves to make the contrast the same, as found by Rushton, is no longer present. The processes that make the responses contrast-dependent occur separately in the rod and cone systems. I should note that this experiment leaves open the possibility that some processes relevant to fading occur after the ganglion cell, as physiological data tend to suggest; the implication is only that when stabilized images are involved such processes are more or less linear. Nor does the experiment locate the process as early as the rods and cones themselves necessarily: it could equally well be in the bipolar cells.

This receptor sensitivity modification is an outrageously simple model for image fading and color constancy and several objections could be raised against it.

First, adaptation may seem too quick to underly the fading of stabilized images. One could however defend the model by arguing that the adaptive state though rapidly modifiable crawls rather slowly toward its final asymptotic level, taking several seconds to achieve a reciprocity sufficiently perfect to prevent any visibility of stabilized image.

Land and Daw (1962) have shown that there can be approximate constancy in a flash. But again, most of the adaptation process is certainly very quick at ordinary light levels. And even if we need to invoke different processes to explain constancy in a flash, it is still possible that constancy in situations closer to the steady state is due largely to adaptation.

Adaptation is not all in the cones, as witness, for example, Dr. Pugh's contribution to this symposium. There are also color appearance phenomena, for instance the observation of supersaturated yellow after adaptation of the B cones which don't themselves respond to yellow - which call for explanation in terms of postreceptoral processes. This has to be acknowledged but I think we can still defend the Rushton model as a useful approximation. We don't yet know, very well, the relative importance of the postreceptoral factors under natural conditions. It's worth noting, I think that a point in favor of the "early fading" model is that when sensations are produced by direct electrical stimulation of the cortex, there is, according to Brindley (1973) no fading. Surprisingly, the cortex seems to lack the equipment for that job.

The resurrection of lightness and color from local contrast at nearby and remote contours in the field (Land, this symposium; Arend, Buhler and Lockhead, 1971) has to be imcorporated into a

full constancy model, but the sensitivity regulation model is intended to describe only the first stage, the derivation of local signals suitable for later integration.

There are deviations from constancy: Helson (1938) found that in a room papered with neutral grays, whites and blacks the whites take on the illuminant color and the blacks take on the complementary color. This can very naturally be explained on the receptor model if we recognize that the simple intensity scaling model for adaptation is quantitatively inexact. There is independent evidence that the transient responses in the light adapted eye are more positively accelerated functions of stimulus intensity (if we represent them by power functions, they have a higher exponent) than in dark adaptation; and it has been shown by Nayatani, Takahama and Sobagaki (1983) that this will account well for the Helson deviations from constancy.

Finally, a more fundamental objection to this model, and to many others, is that it tries to explain constancy by making the signals produced under different illuminations the same. By doing that it does too much. We recognize illuminant quality as well as surface color, as pointed out most thoughtfully and convincingly by Heggelund (1974). We must assume that enough of a signal survives the adaptation process to provide cues to illuminant quality (and also, by the way to allow control of pupil size, which is known to be indifferent to image stabilization). Here again, it's not really clear whether the model is completely misguided or whether it can serve as a useful approximation.

In the light of all these objections the sensitivity regulation model has to be viewed as at best a rather crude and approximate account of the first stage in color constancy. It does have the merit of indicating a possibly important connection between constancy and the fading of stabilized images. It also is useful in illustrating the potential and limitations of what might be called "stupid" processes in color constancy - processes, in this case that produce signals simply related to local intensity differences and the time average of local intensity.

If we give up the idea that color constancy is achieved by the receptors, there is a rather wide range of alternative possibilities to explore. Going to the opposite extreme, the logically admissible arbitrariness in the relation between receptor signals and sensation is enough to allow perception to proceed like medical diagnosis: the signals from the cones, instead of being simply registered or subjected to simple more or less linear transformation might be invested with significance only as symptoms - symptoms of the presence of a certain kind of object surface. The information used could be quite varied including such things as awareness of the distribution of light sources, observation of the intensity profiles of shadows to exploit

secondary reflection cues, remembered information from previous fixations, and so on. But a great problem with "smart" systems like this is that they require a lot of education to form the proper associations between receptoral symptoms and their objective causes - perhaps as many hours of education as a medical doctor gets. And we know from cases where people born with cataracts begin to see after cataract removal late in life (van Senden, 1960) that color naming can be very good immediately without refined visual experience to allow such learning. In view of this the potential of "stupid" systems— if not necessarily quite as stupid as the sensitivity modification system I've described—may still deserve to be explored.

References

Arend, L. E., Buehler, J. N. and Lockhead, G. R. (1971). Difference information in brightness perception. Perception and Psychophysics, 9, 367-370.

Boynton, R. M. (1980). Design for an Eye. In Neural Mechanisms and Behavior. (ed. D. McFadden). pp. 38-70. New York: Springer-Verlag.

Brindley, G. S. (1973). Sensory effects of electrical stimulation of the visual and paravisual cortex in man in Jung. (ed.). Visual Centers. In the Brain, Handbook of Sensory Physiology, 3, 583-594. Berlin: Springer-Verlag.

Dartnall, H. J. A., Bowmaker, J. K. and Mollon, J. D. (1983). Microspectrophotometry of Human Photoreceptors. In Colour Vision. (ed. J. D. Mollon and L. T. Sharpe). pp. 68-80. London: Academic Press.

Derrington, A., Lennie, P. and Krauskopf, J. (1983). Chromatic Response Properties of Parvocellular Neurons. In the Macaque LGN in Colour Vision. (eds. J. D. Mollon and L. T. Sharpe). pp. 245-251. London: Academic Press.

Eisner, A. and MacLeod, D. I. A. (1980). Blue-sensitive cones do not contribute to luminance, J. Opt. Soc. Am., 70, 121-122.

Granger, E. M. and Heurtley, J. C. (1973). Visual Chromaticity-modulation transfer function. J. Opt. Soc. Am., 63, 1173.

P. Heggelund (1974). Achromatic Color Vision - 1: Perceptive variables of achromatic colors. Vision Research, 14, 1071-1079.

Helson, H. (1938). Fundamental problems in color vision I. The principle governing changes in non-selective samples n chromatic illumination. J. Exp. Psychol., 23, 439-476.

Land, E. H. and Daw, N. W. (1962). Colors seen in a flash of light. Proc. Nat. Acad. Sci., 48, 1000-1008.

Luther, R. (1927). Aus dem Gebiet der Farbreizmetrik. Zeitschr. f. techn. Physik., 8, 540-558.

Luzzatto, L. and Gartler, S. (1983). Switching off blocks of genes. Nature, 301, 375-376.

MacLeod, D. I. A. and Boynton, R. M. (1979). Chromaticity diagram showing cone excitation by timuli of equal luminance. J. Opt. Soc. Am., 69, 1183-1186.

MacLeod, D. I. A. and Webster, M. A. (1983). Factors influencing the color matches of normal observers. In Colour Vision. (eds. J. D. Mollon and L. T. Sharpe). pp. 81-92. London: Academic Press.

Muller, G. E. (1924). Darstellung und Erklarung der verschiedenen Typen der Farbenblindheit. Vandenhoeck-Ruprecht, Gottingen.

Nagy, A. L., MacLeod, D. I. A., Heyneman, N. E. & Eisner, A. (1981). Four cone pigments in women heterozygous for color deficiency. J. Opt. Soc. Am., 71, 719-722.

Nayatani, Y., Takahama, K. & Sobagaki, H. (1981). Formulation of a nonlinear model of chromatic adaptation. Color Res. and Appl., 6, 161-171.

Rubin, M. L. (1961). Spectral hue loci of normal and anomalous trichromats. Am. J. Ophthalmol., 52, 166-172.

Rushton, W. A. H. (1971). Color vision: An approach through the cone pigments. Invest. Ophthal., 10, 311-322.

Rushton, W. A. H. (1972). Light and dark adaptation. Invest. Ophthal., 11, 503-517.

Rushton, W. A. H. & Baker, H. D. (1964). Red/green sensitivity in normal vision. Vis. Res., 4, 75-85.

Von Senden, M. (1960). Space and Sight. London: Methuen.

Williams, D. R., MacLeod, D. I. A. & Hayhoe, M. M. (1981). Punctate sensitivity of the blue-sensitive mechanism. Vis. Res., 21, 1357-1375.