Journal of the Optical Society of America A
OPTICS, IMAGE SCIENCE, AND VISION

# Gaining the system: limits to compensating color deficiencies through post-receptoral gain changes

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Received 2 November 2022; revised 13 December 2022; accepted 14 December 2022; posted 14 December 2022; published 12 January 2023

**Color percepts of anomalous trichromats are often more similar to normal trichromats than predicted from their receptor spectral sensitivities, suggesting that post-receptoral mechanisms can compensate for chromatic losses. The basis for these adjustments and the extent to which they could discount the deficiency are poorly understood. We modeled the patterns of compensation that might result from increasing the gains in post-receptoral neurons to offset their weakened inputs. Individual neurons and the population responses jointly encode luminance and chromatic signals. As a result, they cannot independently adjust for a change in the chromatic inputs, predicting only partial recovery of the chromatic responses and increased responses to achromatic contrast. These analyses constrain the potential sites and mechanisms of compensation for a color loss and characterize the utility and limits of neural gain changes for calibrating color vision.** 

https://doi.org/10.1364/JOSAA.480035

## 1. INTRODUCTION

Typical human color vision is trichromatic and depends on comparing signals in the short (S), medium (M), and long (L) wavelength cones. Congenital X-linked color deficiencies affect up to 8% of males and result from the loss (dichromacy) or alteration (anomalous trichromacy) in the spectral sensitivity of the M or L cones [1]. This can result in two cones with spectral peaks close to the normal M cones (protanomalous) or to the normal L cones (deuteranomalous). The difference in the peaks of the normal and anomalous pigment can range from roughly 2 to 12 nm, compared with a separation of greater than 20 nm between normal L and M cones [2]. This results in a reduction of the chromatic signal provided by the difference in the L and M cone responses. Specifically, sensitivity to the L versus M signal should be reduced in proportion to the reduced L versus M separation [3]. If this were the only difference between normal and anomalous trichromats, then their experience of color should reflect this simple reduction or loss, which is the basis for simulations of the color appearance of images by anomalous or dichromatic observers (along with assumptions about the hues corresponding to the intact and altered chromatic dimensions) [4–6].

However, several studies have explored the possibility that the color experiences of anomalous trichromats may be more similar to color-normal individuals than their altered cone sensitivities predict. Increased sensitivity to the L versus M dimension has been found with a wide variety of paradigms, including perceptual salience [7], color similarity judgments [3], hue scaling [8], hue loci [9], contrast matching [10], color contrast scaling [11] and discrimination [12], and color-contingent adaptation [13]. Amplified signals, relative to those predicted by anomalous pigments, have also been suggested by cortical responses to color measured by fMRI [14] or evoked potentials [15]. These findings also parallel reports of only a weak association between the degree of cone spectral shift and loss in discrimination (as determined by the mean and range, respectively, of the Rayleigh matches used for diagnosing color deficiencies [2]). While there are substantial individual differences in these tasks, these results suggest that at least some anomalous observers exhibit post-receptoral compensation for the weaker color signal provided by their cones [2,16].

The nature of this compensation is poorly understood, but one simple and plausible mechanism would be gain changes in the responses of post-receptoral neurons [17,18]. For example, if the input to an opponent L versus M neuron is reduced, the cell could increase its sensitivity to restore the normal response magnitude. If this gain change occurred before the sites introducing noise, then this could effectively undo the weakened input signal, so that color discrimination and perception could be nearly normal despite the large differences in the cones. Yet, empirically, the compensation when it is observed is typically only partial, which raises the question of what factors might limit the ability of the visual system to adjust for a color deficiency.

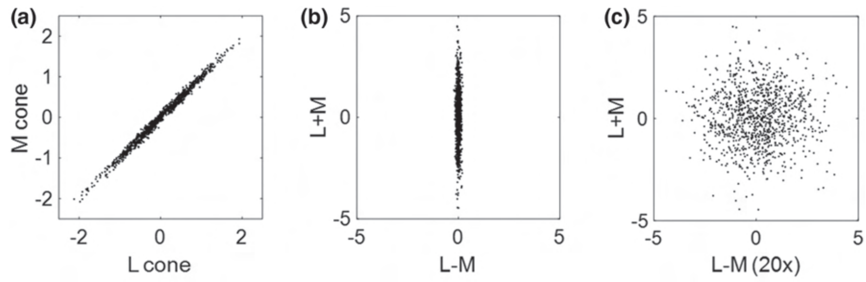


**Fig. 1.** For natural color distributions, the L and M cone signals are highly correlated ($r > 0.995$) leading to a roughly twentyfold difference in the contrast range along the achromatic axis (L + M) vs. the chromatic axis (L − M). (a) Hypothetical L and M cone responses showing the predicted level of correlation. (b) Chromatic (L − M) and luminance (L + M) responses showing the difference in the root mean square (rms) contrast ranges between these two types of signals. (c) Gain on the L − M responses independently increased twentyfold in order to match the range of responses for luminance and chromatic signals.

In this study, we explored limits to the impact of gain changes imposed by properties of the visual coding of color. If the visual system represented the L versus M dimension of color independently of other dimensions (such as the achromatic dimension), then the L versus M gain could be adjusted independently. However, as we consider below, the signals are instead multiplexed both within individual neurons and across the population, and this multiplexing can arise both before and after the potential site(s) of the gain changes. Our aim was to model how this nonindependence impacts the extent to which the system can compensate for a color deficiency by simple gain changes in the population.

## 2. NEURAL GAIN AND CODING EFFICIENCY IN COLOR VISION

Setting the neural gain is relevant not only for compensating color deficiencies but more generally for calibrating all color vision. A central tenet of vision science is that visual coding is matched to the statistical structure of the visual environment [19]. Along with principles for how to optimize this match, such as information theory, this predicts that many properties of vision can be inferred from properties of the stimulus [20,21]. In the case of color vision, this match has been explored in a wide variety of ways, including how color information is sampled and represented [22–30]. Because of the overlapping spectral sensitivities of the normal L and M cones, their signals are highly correlated [Fig. 1(a)], especially for the broad and gradually varying color signals characteristic of natural illuminant and reflectance spectra. For measurements of natural scenes, the correlations between the L and M responses have been found to exceed 0.995 [26,31,32]. This corresponds to a roughly twentyfold difference in the root mean square (rms) contrast of signals along the achromatic axis (L + M) versus the chromatic axis (L − M) [Fig. 1(b)]. To optimally encode the signals, the gain for luminance and chromatic contrast should be set separately so that, for each dimension, the responses evenly sample the output levels for the full dynamic range of the neuron [32]. In effect, this "spheres" the responses so that the distribution of output levels is equivalent for the different dimensions [Fig. 1(c)]. This predicts that the neural gain for chromatic contrast should be much higher than for luminance contrast. Consistent with this, sensitivity to chromatic contrast is many times higher than for luminance contrast, when the dimensions are compared on the basis of cone contrasts [33].

In the case of anomalous trichromacy, the range of luminance variation remains comparable (though not identical) to normal trichromacy, while the range of chromatic variation is reduced. For example, for a pair of pigments separated by 6 nm, the rms contrast of the chromatic signals should be further reduced from 5% to close to 1% of the luminance variation. Yet, in principle, the same scaling adjustments could again be applied to match the responses for luminance and chromatic contrast, though the required gain changes in this case would be higher. However, several factors limit the extent to which the response range could be fully restored. In this study, we focus specifically on factors related to how chromatic signals are encoded at different stages of the visual system and how well simple gain changes could equate their responses for different input ranges.

## 3. POST-RECEPTORAL COLOR CODING

As a roadmap for the following analyses, Fig. 2 shows a schematic illustration of different models of post-receptoral color coding. In the upper panel, color information is carried in three independent "cardinal mechanisms" that respond to variations in luminance (L + M) or to chromatic signals corresponding to the opposing signals in the L versus M cones (L − M) or S vs. both L and M cones [S − (L + M)] [34]. Many aspects of early color coding can be accounted for by an organization of color information in terms of these dimensions, which also describe how the cone signals are organized within the primary pathways and cell types in the retina and geniculate [35,36] (though notably these dimensions do not provide an explanation for the structure of color appearance [37–40]). As we illustrate below, if these channels were independent, then within each a decrease in input could be largely, but not completely, offset by an increase in gain. However, physiological and psychophysical studies have demonstrated that the signals along these dimensions are instead comingled, e.g., so that the representation includes mechanisms tuned to different combinations of signals along the cardinal axes. This is illustrated by the bottom panel, in which the three dimensions are instead spanned by multiple mechanisms. In the case of the L − M and S − (L + M) chromatic signals, this multiple-channel organization is thought to arise in visual cortex by combining

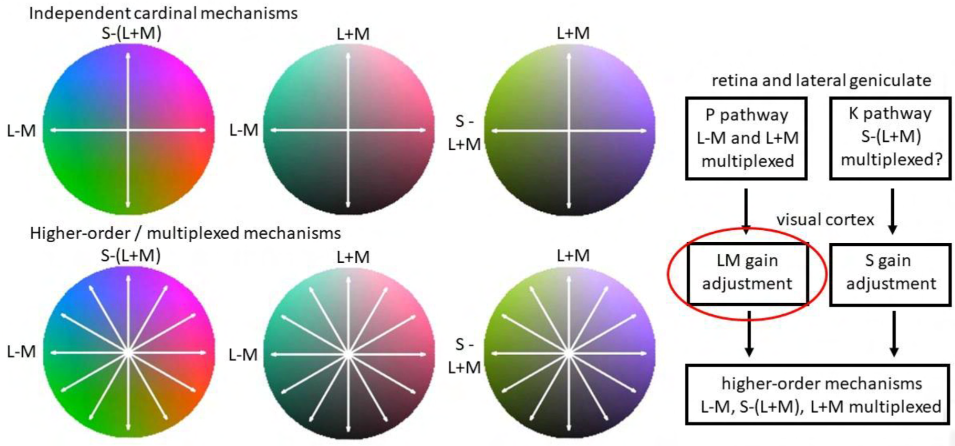


**Fig. 2.** Post-receptoral representation of color in normal trichromats. Top panels: Primary cell types and pathways of the retina and lateral geniculate carry information about color in terms of three cardinal dimensions corresponding to opposing signals in the $L-M$ cones or $S-(L+M)$ cones or to nonopponent luminance signals given by the summed inputs of the $L+M$ cones. Lower panels: However, color mechanisms also respond to many different combinations of the cardinal axes, so that within each plane there are mechanisms tuned to different directions. Right panel: Schematic of the color representation at different stages. In the retina and geniculate, $L-M$ and $S-(L+M)$ are segregated into the P and K pathways, but cells within each pathway respond to luminance and chromatic contrast. In the cortex, the cardinal mechanisms are also combined to form higher-order mechanisms that are tuned to intermediate directions in color space. We focus on gain changes in the cortex before the $L-M$ and $S-(L+M)$ signals are combined; however, this does not preclude gain changes in higher-order mechanisms. The red circle represents the potential site of the gain adjustment compensating for anomalous trichromacy.

the initial segregated cardinal chromatic mechanisms to form later "higher-order" color mechanisms, each tuned to a different hue angle [41,42]. This representation may code for directions in color space in ways that are analogous to the population codes for other stimulus features such as spatial orientation [43–45]. Yet for other signals, such as for luminance and chromatic dimensions, the multiplexing is also present early in the retina because of how the neurons' receptive fields combine the cone signals. In particular, at least at these initial stages, the same neurons respond to both luminance and chromatic contrast from the L and M cones [46,47], though this "double-duty" receptive field organization is less evident in the koniocellular pathway of the lateral geniculate nucleus that carries the signals from S cones [48]. In the following, we consider different stages of post-receptoral color coding to examine the implications of the multiplexed representations for adjusting to a color deficiency. As we illustrate, the impact of these adjustments depends in part on whether the gain changes occur before or after the signals are multiplexed.

For the present analysis, we assumed that the gain adjustment occurs in early visual cortex, consistent with neuroimaging evidence that primary visual cortex is the earliest potential locus for a gain compensation in anomalous trichromacy [14]. This places the multiplexing of chromatic and achromatic signals before the gain, and the elaboration of higher-order chromatic mechanisms after the compensatory gain. Adaptation at later stages, including within the population of higher-order mechanisms, is also well established [43] but is less relevant to compensation for a color loss if that compensation also occurs at earlier stages. To explore the impact the different stages have on compensation, the analyses first consider the limits imposed by the joint coding of color and luminance in the retina and geniculate and then how the $L-M$ and $S-(L+M)$ signals are combined in the cortex.

## 4. CHROMATIC AND ACHROMATIC CODING IN INDIVIDUAL NEURONS

Chromatic signals from the L and M cones are primarily carried within the midget bipolar and ganglion cells in the retina, which in turn project to the parvocellular (P) layers of the lateral geniculate [49]. Luminance signals are also carried through a further pathway corresponding to the magnocellular (M) layers, which contain cells combining L and M cones in a nonopponent manner and which are the likely basis for luminance sensitivity, as measured by conventional criteria such as flicker photometry [50]. In contrast, the achromatic signals in P cells may instead underlie the achromatic lightness dimension of color appearance [51].

The cells of the P pathway represent the first post-receptoral stages at which compensation for a color deficiency could occur. However, P cells exhibit little short-term adaptation to contrast [52] (though adaptation is suggested by some fMRI measurements [53]). Moreover, an analysis by Lutze, Pokorny, and Smith [54] has also argued against adaptation in the long-term. The authors examined whether the contrast response functions (CRFs) of P cells were adapted to a lifelong change in the strength of chromatic inputs, by comparing response functions in normal and color deficient observers. The majority of P cells (Type I) have spatial and chromatic opponency, with cone opponency arising because different cone classes feed the center and surrounding regions of the receptive field [46,47,55] (though the extent to which the inputs are restricted to specific cone classes or sampled randomly remains debated [56]). As a result, the cells respond to chromatic and achromatic contrast.

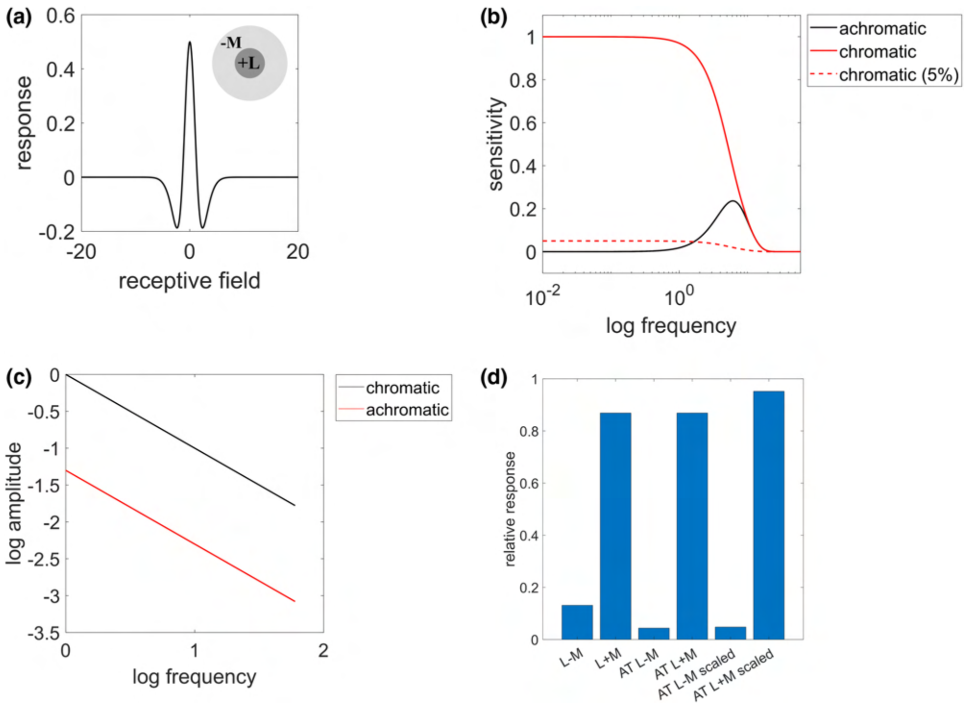


**Fig. 3.** Chromatic and achromatic coding in parvocellular neurons. (a) Receptive field of an idealized P cell with a + L center and −M surround. (b) Response of the cell shows bandpass tuning for achromatic contrast and lowpass tuning for chromatic contrast. (c) 1/f amplitude spectrum for luminance and chromatic contrasts for natural scenes, weighted by the cone contrasts. (d) Response of the cell to the achromatic and chromatic spectrum (left pair of bars), the color loss in an anomalous trichromat (middle pair), and the responses after compensating for the reduced chromatic input (right pair).

Lutze *et al*. showed that the achromatic CRF was similar for normal and color deficient observers despite the differences in the chromatic signals [54,57] and also noted that the CRF is similar between dichromatic and trichromatic members of new world primate species [58,59]. Thus, P cells are an unlikely site for the compensation.

However, even if they could adapt, the joint responses to luminance and chromatic contrast would likely limit the extent to which the cells could correct for a specific loss in chromatic contrast. To illustrate this, we modeled the net modulation of an idealized P cell with a + L center and −M surround [Fig. 3(a)], with the strength of the +L and −M weighted equally for a neutral (e.g., flat) spectrum. For simplicity, we also assumed a linear CRF. Our analysis is therefore only qualitative and not meant to simulate the specific neural response. As many studies have documented, because of the coupling of spatial and chromatic opponency and latency differences between the center and surround, the cells respond to both chromatic and achromatic contrast but with different spatiotemporal sensitivities [46]. For achromatic contrast, the responses of the cells show bandpass tuning in both space and time; for chromatic signals, the responses are lowpass [Fig. 3(b)]. This results in stronger responses for chromatic contrast than achromatic contrast at low frequencies, while the achromatic responses are stronger at higher frequencies. To model these responses, we assumed that spatial contrast for luminance and color falls with increasing spatial frequency as 1/f, which is the characteristic amplitude spectrum of natural images [60,61]. However, as noted in Fig. 1(a), for the normal trichromat, the chromatic cone contrast is only 5% of the achromatic variation [Fig. 3(c)]. Based on this, the response of the modeled P cell to the 1/f amplitude spectrum of the chromatic inputs is roughly 13% of the response to the achromatic spectrum [Fig. 3(d)].

For an anomalous observer with an L to M peak separation of 9 nm, the chromatic input to the cell should again be reduced to 33% of the normal trichromat. However, if it could vary at all, the gain of the cell should be set by the total modulation, to both the luminance and achromatic contrast. Since the overall reduction is about 8.75% (the change in total chromatic and achromatic input), the gain should only adjust by a factor of 1.1%, which results in weak compensation for the chromatic losses as well as a weak increased gain for the luminance signals. In other words, a three-fold gain is required to offset the weakened chromatic signals, but a much smaller adjustment is required to maintain responses to the joint chromatic and achromatic inputs [Fig. 3(d)]. For a dichromat, the predicted change in the neural gain would be 13%, which is more substantial but still small given the complete loss of cone-opponent input. Again, these gain changes do not appear to occur in P cells. However, the point is that, in such cells, with multiplexed sensitivity to chromatic and achromatic signals, gain adjustments could at best yield only weak compensation for the color deficiency.

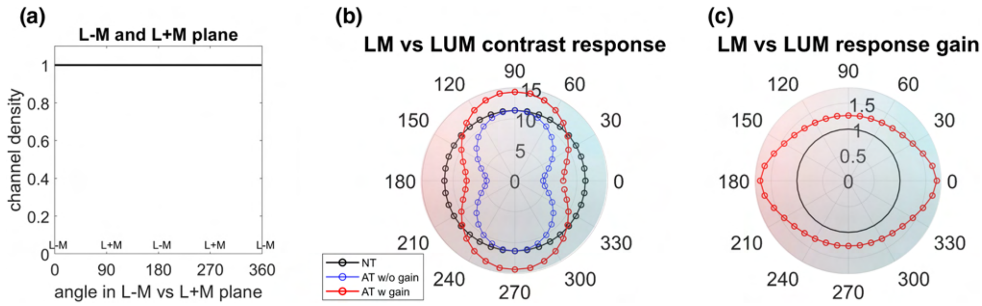


**Fig. 4.** Population codes and gain for luminance (L + M) and chromatic (L − M) signals, assuming a uniform distribution of underlying channels in normal-trichromat observers. (a) Simulated L − M and L + M channel density for normal trichromats. (b) Responses to stimuli in the L − M/L + M plane before (blue) and after (red) normalizing gains of individual channels to adjust for the chromatic losses. (c) Relative change in the gain of channel responses (red) after each channel renormalizes responses for the reduced chromatic contrast. The compensated responses have a residual loss in chromatic sensitivity and enhanced sensitivity for luminance contrast.

## 5. CHROMATIC AND ACHROMATIC CODING IN THE NEURAL POPULATION

The previous analysis considered only a single model neuron. Yet different P cells [36], as well as different cells in primary visual cortex [62], vary widely in their relative weighting of chromatic and achromatic contrast. Joint tuning has also been found psychophysically, e.g., in masking and adaptation studies [43,63–65]. This suggests that the luminance and chromatic information carried by the P pathway reflects a population code, in which there are multiple mechanisms tuned to different directions within the L − M and L + M plane, and that this code arises before the sites of the gain adjustment. What are the consequences of this representation for adjusting to a color deficiency?

To assess this, we used a simple model based on a set of 36 half-rectified mechanisms tuned to different linear combinations of the L and M cones to simulate uniform sampling of different directions in the L − M/L + M plane [Fig. 4(a)]. Because we are assessing only the change in relative sensitivity and not the absolute sensitivity (e.g., in terms of cone contrast), we assumed that, for the normal trichromat, sensitivity along the L − M and L + M axes was equal (e.g., in terms of multiples of threshold), and that the L/M peak separation in the anomalous trichromat was 30% of the normal separation. As noted, we also assumed that how the channels weighted the L and M cone inputs occurred before the stage of adaptation, which we presume is primarily in the cortex, since again P cells show little adaptation to contrast [52], and fMRI studies suggest that the compensation does not occur earlier than cortical areas V1 or V2 [14]. A final important property of the model was that contrast was coded by the summed activity of all channels and not merely the most sensitive. Consistent with the properties of cortical color coding, the channels were half-rectified so that the summed responses are not simply cancelled by the sum of excitation and inhibition in opponent mechanisms [32].

The loss in L − M contrast owing to a color deficiency will reduce the L − M input to each individual unit, as illustrated in Fig. 3. At the population level, this will bias the distribution of preferred directions away from the L − M axis and toward the L + M axis, because neurons that respond to luminance and chromatic contrast will have a relatively weaker response to the chromatic component. For example, the tuning of a neuron that was equally sensitive to chromatic and achromatic contrast (relative to a normal trichromat) will rotate toward the achromatic axis. Gain changes within each unit could in principle restore its response range. However, like the example of the individual P cell given above, the gain for most mechanisms will be set by both the achromatic and chromatic inputs. Thus, with the exception of the pure L − M mechanism, gain changes can only partially compensate for the reduction in the chromatic input and also lead to amplified responses for the achromatic inputs [Fig. 4(b)]. The net effect of these gain changes for the summed responses across the population is a residual loss in chromatic contrast and an increased response to achromatic contrast [Figs. 4(b) and 4(c)].

In reality, the distribution of channel tuning in the L − M/L + M plane is not uniform. However, qualitatively similar consequences are predicted for nonuniform distributions, as long as the population includes mechanisms that jointly encode chromatic and achromatic contrast. For example, Fig. 5 shows the predictions for a distribution of channels that is biased along the L − M axis, consistent with the measured preferences of P cells [36]. In the anomalous trichromat, the tuning is again shifted toward the achromatic axis, and the compensatory gain similarly results in partial restoration for chromatic contrast while overshooting the normal trichromatic response for achromatic contrast.

Again, in these analyses, we assumed that the L + M and L − M signals are comingled and thus cannot be adapted independently; this is because P cells respond to both achromatic and chromatic signals. But what if, at later stages, the achromatic and chromatic responses do become separated? How and whether the achromatic and chromatic responses of P cells are decoupled in subsequent cortical processing stages is unresolved [66]. However, a number of important signal transformations in the primary visual cortex occur such as the emergence of "double-opponent" cells, which could more fully separate chromatic and luminance sensitivity [62,67,68]. If a separation of chromatic and achromatic signals arises, it could allow for more independent adjustments for a color loss. On the other hand, the fact that adaptation or masking can be selective for how achromatic and chromatic signals are combined indicates that at least some aspects of the cortical representation maintain a population

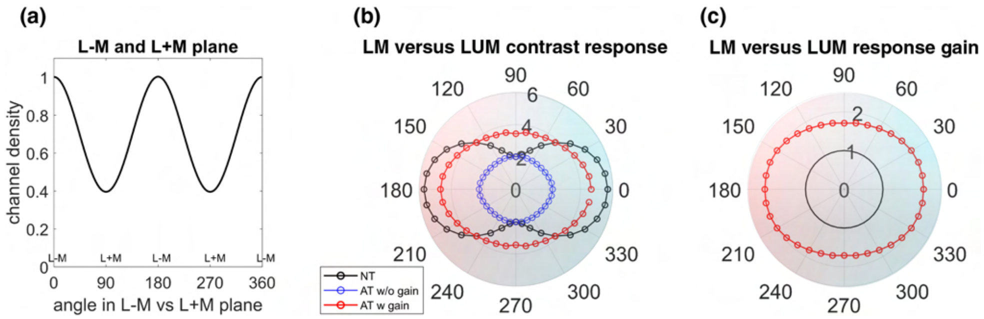


**Fig. 5.** Compensation and population codes for luminance and color with gain changes assuming a nonuniform distribution of underlying channels. (a) Simulated L − M and L + M channel density for normal trichromats, assuming more cells are tuned to the L − M axis. (b) Responses before (blue) or after (red) renormalizing each channel for a loss in chromatic contrast. (c) Relative change in the gain of channel responses (red) after each channel renormalizes responses for the reduced chromatic contrast. The compensated responses have a residual loss in chromatic sensitivity and enhanced sensitivity for luminance contrast.

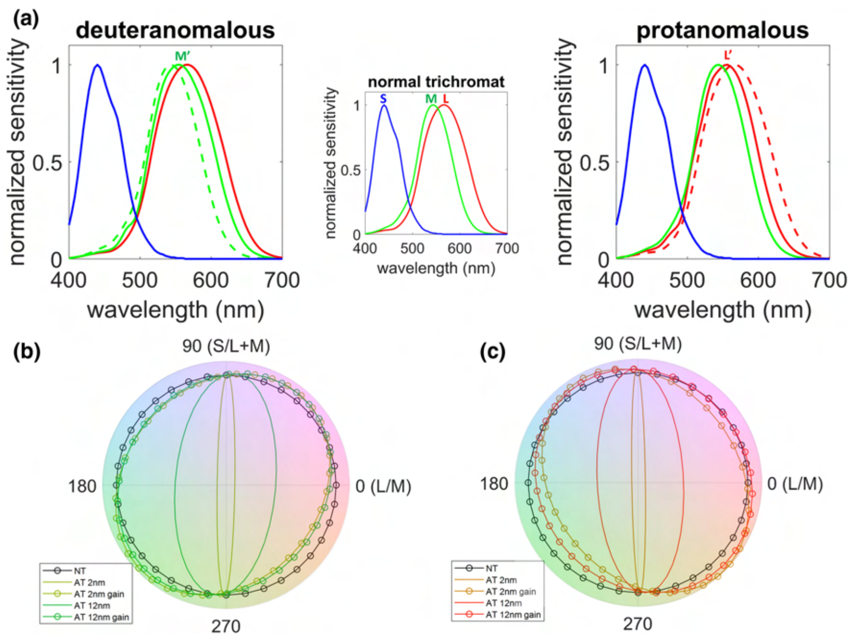


**Fig. 6.** Limits to gain in independent cardinal mechanisms. Decreasing the LM cone separation (as is the case for anomalous trichromats [AT]) is not equivalent to a gain loss, thus increasing the LM gain cannot fully discount the loss. (a) Spectral sensitivity curves of S, M, and L cones for deuteranomalous (M shifted toward L creates M' [the green dashed line shows the original M cone spectral sensitivity curve for the NT]), protanomalous (L shifted toward M creates L' [the red dashed line shows the original L cone spectral sensitivity curve for the NT]), and normal trichromats (NT). (b) L − M and S − (L + M) responses to naturalistic color signals for NTs and deuteranomalous observers with 2 or 12 nm of separation, before or after compensating for the color loss with a change in the L − M gain. (c) Similar results for protanomalous observers.

representation for achromatic and chromatic contrast and thus should show similar limits to compensation [63,64,69].

## 6. INDEPENDENT GAIN CHANGES WITHIN L − M AND S − (L + M) MECHANISMS

Thus far, we have only considered color coding and compensation in the signals from the L and M cones. What are the consequences of compensation for dimensions of color vision that also involve the S cones? The signals from the S cones are primarily conveyed through the koniocellular (K) layers of the lateral geniculate nucleus [70] and thus are initially confined to a different subsystem from the P and M pathways (though there is also evidence for a separate smaller opponent subsystem in which the S cones sum with the L or M cones and which has been suggested to mediate color appearance [39]). In terms of the P vs. K pathway, there is thus a clearer separation of the cardinal axes, at least at precortical stages. Moreover, early components of cortical adaptation may reflect sensitivity changes in the separate L − M and S − (L + M) mechanisms [71], though behaviorally adaptation also reveals multiple higher-order color mechanisms [42,43]). Accordingly, we first examine compensatory gain changes occurring within independent L + M and S − (L + M) mechanisms and then within the population of higher-order mechanisms (which are also assumed to reflect a stage after the initial gain change).

Figure 6 illustrates the responses to chromatic contrast after a complete and independent adjustment to the L − M and

$S-(L+M)$ signals available to an anomalous trichromat. If the visual system could adjust independently along the two cardinal chromatic axes, then the losses in $L-M$ signals owing to a color deficiency could be more fully compensated. However, even in this case, the renormalization should lead to residual errors. The reason for this is that the shifts in the peaks of the anomalous pigments also alter the spectral sensitivity of the mechanisms. This results not only in a reduction of contrast sensitivity but changes in the mechanism tuning, resulting in a "tilt" of the cardinal axes within the $L-M$ and $S-(L+M)$ chromatic plane relative to the normal trichromat [Figs. 6(b) and 6(c)]. Rescaling the sensitivity of the anomalous $L-M$ mechanism could equate the average responses for the normal and anomalous observers but cannot undo the distortions owing to the spectral sensitivity change. Note this is similar to the problem that the luminosity function (which depends on the summed responses of the L and M cones) is also necessarily different for the color deficient observer. Thus, even in the extreme of three independently adaptable cardinal dimensions, there is a limit to which the color percepts of anomalous trichromats could be fully compensated by gain changes.

## 7. COMPENSATION AND HIGHER-ORDER CHROMATIC MECHANISMS

As we described above, within the cortex, the signals from the $L-M$ and $S-(L+M)$ dimensions are combined to form higher-order mechanisms tuned to intermediate chromatic directions [72–75]. However, a difference from the $L-M/L+M$ plane is that, as noted, the $L-M$ and $S-(L+M)$ dimensions are initially segregated, allowing potential gain changes to be applied before their inputs are combined. Some cortical amplification of S-cone mediated signals is assumed in normal trichromacy because the S cones have a disproportionately large contribution to color perception despite making up only a small fraction of the cone mosaic. However, the evidence for this amplification is mixed [62,76,77].

Figures 7 and 8 show the effect of gain changes if these occurred independently for $L-M$ and $S-(L+M)$ mechanisms, prior to their combination. Specifically, these simulations show how compensating for signals in the $L-M/L+M$ population, as in Figs. 4 and 5, should spill over to affect the channels constructed in the $L-M$ and $S-(L+M)$ plane. Because the $L-M$ response can only be partially restored, this reduces the $L-M$ input to cardinal mechanisms within the chromatic plane, but without a concomitant increase in the overall response to the S signals.

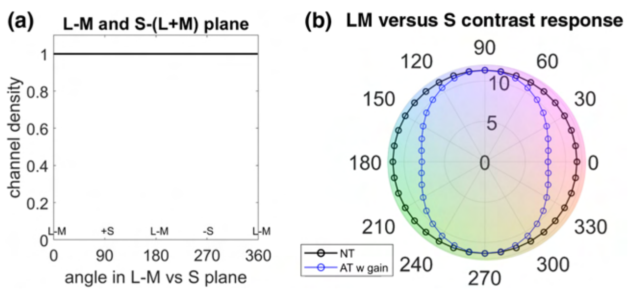


**Fig. 7.** Compensation and population codes for LM and S signals with gain changes assuming a uniform distribution of underlying channels in normal trichromats. (a) Uniform distribution of channels in the $L-M$ and $S-(L+M)$ plane for NT observers. (b) Responses to different directions in the plane assuming renormalization before the $L-M$ and $S-(L+M)$ signals are combined (blue). The compensated responses have a residual loss in $L-M$ sensitivity with no net change in sensitivity for $S-(L+M)$ contrast.

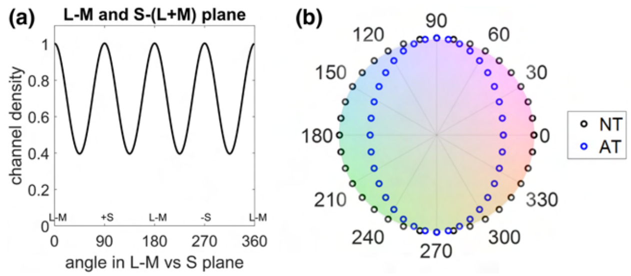


**Fig. 8.** Compensation and population codes for LM and S signals with gain changes assuming a nonuniform distribution of underlying channels in normal trichromats. (a) Distribution of channels in the $L-M$ and $S-(L+M)$ plane for NT observers, consistent with higher density of mechanisms tuned to the cardinal axes. (b) Responses to different directions in the plane assuming renormalization before the $L-M$ and $S-(L+M)$ signals are combined (blue). The compensated responses have a residual loss in $L-M$ sensitivity with no net change in sensitivity for $S-(L+M)$ contrast.

Figure 7 illustrates these predictions for uniform distribution of chromatic mechanisms, while Fig. 8 shows that similar effects are again predicted if the distribution is biased along both of the cardinal axes [43].

The limits to compensation can also be evaluated if the gain changes instead occur after the $L-M$ and $S-(L+M)$ mechanisms are combined. As described above, short-term chromatic contrast adaptation exhibits selective response changes along directions intermediate to the cardinal axes, implicating adaptation in higher-order mechanisms [42,43,78]. A model based on setting the gains after the distribution is formed may therefore be more plausible (at least for short-term adjustments). In this case, predicted effects for the color-anomalous observer in the chromatic plane parallel the predictions for the $L-M/L+M$ plane, i.e., a partial recovery of color signals along the $L-M$ dimension with enhanced responses to the $S-(L+M)$ dimension.

## 8. DISCUSSION

To summarize, several lines of evidence point to compensation for color deficiencies [2,16]. However, the processes underlying these adjustments and what limits them remains poorly understood. In this study, we evaluated the potential role of one possible factor—simple neural gain changes for compensating for the reduced $L-M$ signals available to anomalous trichromats [17,18]. These gain changes were assumed to arise within individual neurons and to result from short- or long-term adaptation to match their output range to the range of prevailing inputs. The compensation thus rests on a plausible, widely assumed, and pervasive property, i.e., that visual coding adapts to changes in the visual diet, whether these changes arise from variations in the environment or the observer [79]. However, as we have shown, the compensation for color losses predicted by this adaptation is limited by the properties of the neurons and the populations, such that adaptation cannot completely undo losses in one component of the input if the mechanism or the population respond and set their gain for multiple types of inputs.

For our analyses, we assumed adaptation and compensation arise in the cortex. This is consistent with the findings that fMRI BOLD responses to chromatic contrast in anomalous trichromats are weak in primary visual cortex but, in some individuals, approach the magnitude of normal trichromatic responses in the next visual area, V2 [14]. It is intriguing that cells earlier in the visual pathway do not appear to adjust to the changes in their chromatic inputs produced by a color deficiency, since this suggests that, at the level of the retina and geniculate, the cells are not fully optimized for encoding contrast. Lutze *et al*. suggested that this may reflect a more hard-wired gain in the cells [54]. The reasons for this, and more generally for why cortical cells and M cells show much stronger adaptation than P cells, are unclear. One possibility is that the more linear responses of P cells are less likely to lead to response saturation, so that the pressure to adjust their dynamic range is weaker. However, as we have shown here, an added potential factor is that, for natural stimulus distributions, the responses of the cells may tend to be dominated by the achromatic inputs so that adjustments for changes in the chromatic inputs are less imperative.

In modeling the perceptual consequences of gain changes in the cortical population, we assumed that the perceived contrast of the stimulus depended on the sum of the responses across all the channels that were sensitive to the stimulus and not merely on the channel(s) that were most sensitive. This assumption has also been used to model contrast responses from the activity of cells in primary visual cortex [80,81]. In the case of color, one example of evidence supporting this assumption comes from contrast adaptation [43,69]. Even though the channels tuned to the $L-M$ and $S-(L+M)$ cardinal axes are independent, there is some cross adaptation between them in measures of perceived contrast. This can be explained by assuming that adaptation to one chromatic axis (e.g., $L-M$) also reduces sensitivity in mechanisms tuned to intermediate axes, and that these then contribute weaker responses to target stimuli confined to the other (e.g., S) cardinal axis [43]. In other conditions, such as detection thresholds, performance may depend more strongly on the most sensitive mechanisms. However, in this case, the color-deficient observer may also be limited by the lower signal-to-noise ratio. In fact, it is for this reason that compensation may most often occur only for suprathreshold stimuli. As noted, the impact of noise will in part depend on whether it arises before or after the site of gain change [17,82]. In the former case, gain changes will amplify both the signal and the noise, which could impact suprathreshold discrimination while still producing a richer perceived gamut of color contrast.

Recently, Knoblauch and Werner explored the role of noise specifically in the context of compensation [83]. They showed that multiplicative noise, combined with a contrast response function that has a compressive nonlinearity, leads to a weaker maximum response in the mechanisms and thus can also limit compensation. Their model better encapsulates the actual properties of neural responses than the simple linear and noise-free model we considered, and a useful extension would be to combine these approaches to simulate realistic neural responses in the population. However, it is unlikely that this would qualitatively change the limitations resulting from the multiplexing of $L-M$ contrast with other color dimensions.

For modeling these population responses, we also assumed that the cortical representation of color involves multiple chromatic mechanisms in both normal and anomalous trichromats. However, to our knowledge, these higher-order mechanisms have not been evaluated for anomalous observers. The gain changes we modeled make testable predictions for the characteristics of these channels. In particular, they suggest that the density of the channels should be biased away from the $L-M$ axis. In turn, this predicts that, while adaptation and masking should still show selectivity for multiple directions in color space, there should be asymmetries such that the effects of the sensitivity or response changes are more selective along the $L-M$ axis compared with normal trichromats, even when the contrast scaling of the signals is adjusted for the observer's $L-M$ and $S-(L+M)$ sensitivity [18].

The simulations also predicted that compensation for the weakened $L-M$ signal should in principle also result in increased responses for achromatic contrast. Previous studies have documented various visual enhancements from color deficiencies [10,84–87], but when and how they might be manifest is complex. We examined the influence of gain changes on the P pathway; as noted, however, $L+M$ signals are also carried through the M pathway, which is the likely substrate of the luminosity function [50]. The M pathway is insensitive to chromatic contrast and therefore should not undergo gain changes with a color loss. Accordingly, tasks that differentially depend on this pathway (e.g., flicker photometry [88] or contrast discrimination under appropriate conditions [57]) should not show an enhancement. Conversely, the achromatic signals in P cells have been proposed to play a role in surface lightness perception and in fine spatial discriminations [88]. Tasks that depend on the achromatic signals from the P cells may be more likely to show enhancements with color deficiencies. Enhancements could similarly occur for signals carried by the S cones if the gains are set after the $L-M$ and $S-(L+M)$ signals are combined in the cortex. However, enhancements in this case are less evident. As described in our analyses, this could be because the gain changes occur before the cardinal mechanisms are combined to form higher-order mechanisms. Yet, as also noted, these mechanisms can be readily adapted at short time scales. The sites of the potential adjustments for a color deficiency, and whether these are different for short-term vs. long-term gain changes, remain important questions.

Gain changes and noise are only some of the many factors that could contribute to or limit adjustments to a color deficiency. For example, as the cone spectral sensitivities become more similar, it may be harder for the visual system to correctly distinguish them [31,89]. A further class of processes that are likely to play a role are post-perceptual. For example, color naming shows comparatively little deficit with color deficiencies, and this could in part be because it reflects how observers learn to categorize and label colors, apart from how the stimuli are actually perceived [90]. Similarly, even individuals without vision can have conceptual knowledge of color and color relations that resemble the representations of normally sighted individuals [91,92]. As these examples illustrate, the role of different factors will also depend on the nature of the task and the observer's judgment. A major challenge in understanding compensation is in distinguishing adjustments in actual sensory signals vs. these more cognitive strategies and the contexts under which they are manifest. In any case, our analyses suggest that neural gain changes, a plausible sensory mechanism for counteracting weakened color signals from the cones, could provide an important but ultimately incomplete process for normalizing color experience among observers with varying forms of trichromatic color vision.

**Funding.** National Eye Institute (EY-010834); American Australian Association Graduate Education Fund.

**Disclosures.** The authors declare no conflicts of interest.

**Data availability.** Data underlying the results presented in this paper are not publicly available at this time but may be obtained from the authors upon reasonable request.

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