

The Eye's Mechanisms for Autocalibration

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As those of us who build optical instruments are all too aware, changes in the environment surrounding an instrument or changes within the instrument itself can quickly throw it out of calibration. This usually requires someone to intervene and restore the device to its calibrated state. The human visual system, on the other hand, does not enjoy the luxury of such a caretaker, and must calibrate itself continuously. Evolution has created ingenious mechanisms that maintain optimal visual performance over the three-quarters of a century that corresponds to a typical human lifespan. This autocalibration occurs on time scales of milliseconds to years and at all levels of the visual system, from the most fundamental optical processes to the most complex neural mechanisms. These mechanisms are truly self-calibrating in the sense that they are not simply prescribed processes for coping with expected changes, rather they rely on feedback to actively correct visual function in response to changes in the visual environment and the visual system itself. This article explores a few of the numerous autocalibration mechanisms employed by the human visual system, from optical calibrations to neural adaptations that depend on coordination between the visual system and other sensory modalities.

OPTICAL MECHANISMS

Maintaining a sharp retinal image

An essential capability in any imaging system is establishing and maintaining focus. For example, cameras require a mechanism to ensure that the image plane and the film plane coincide for a wide range of possible object distances. Modern cameras achieve this with a built-in autofocus mechanism based, for example, on sonic ranging or triangulation. Accommodation, the eye's reflexive autofocus mechanism, is an adaptive optical system consisting of a pliable lens, the focal length of which is under neural control. Unlike autofocus in a camera, which is controlled by a single source of distance information, the eye capitalizes on any of a number of sources depending on the visual scene. These sources include binocular disparity, vergence, chromatic aberration, and a host of monocular depth cues. This opportunistic strategy makes human accommodation impressively robust over a broad range of viewing conditions.

For accommodation to successfully bring images into focus on the retina at any object distance, the parameters of the eye's optics must be set correctly to begin with. For example, if the axial length

of the eye is too long, as in the case of myopia, distant objects are inevitably out of focus. This problem is greatly exacerbated by the fact that the eye, unlike a camera, grows during the first decade by an additional one-third of its size at birth. Maintaining a focused retinal image throughout development therefore requires the coordinated growth of the cornea, lens, and overall eye. One solution to this problem would be to genetically preprogram the eye's development to keep the growth of the cornea, lens, and eye in lock step at every stage. But this solution requires exquisite genetic control and fails if environmental influences intervene. Instead, the eye has evolved a feedback mechanism, called emmetropization, to maintain coordinated eye growth. Studies in chicks and monkeys have shown that the axial growth of the eyeball is under active control to maintain good focus.¹ For example, placing positive lenses on the eyes of a developing animal slows the eye's axial growth, whereas negative lenses accelerate the axial length increase to minimize the total refractive error of the eye. Remarkably, this mechanism does not require the brain: emmetropization proceeds even after severing of the optic nerve,² which precludes neural communication between eye and brain. Moreover, emmetropization operates locally even within the eye. In studies in which half of the visual fields of chick eyes were occluded, the chicks' eyeballs

grew asymmetrically, so that the half of the retina receiving normal visual stimuli exhibited no refractive error, while the half of the retina deprived of visual stimuli grew larger and became myopic.³

The most puzzling aspect is that emmetropization shows accurate directional behavior (both myopic- and hyperopic-induced refractive errors can be compensated), but a signed error signal has yet to be identified. Optical blur alone can indicate an error in focus, but cannot indicate the direction of the error. Other cues such as the eye's longitudinal chromatic aberration, which can indicate the direction of a focus error, are not required because emmetropization can proceed in their absence.⁴ We are also uncertain what specific retinal circuits code this signal and ultimately control eye growth. If we could solve this mystery, we might be able to learn how to prevent the development of refractive errors. This would have a major impact on the quality of life of the roughly 25% percent of the American population with refractive errors⁵ (the numbers are even higher in certain countries such as Singapore where as much as 80% of the population is myopic).⁶ Ultimately, it is conceivable that pharmacological control of refractive error could eventually replace glasses, contact lenses, and refractive surgery.

Recent work has demonstrated that the cornea and lens also cooperate to optimize

retinal image quality. Artal *et al.*⁷ have shown that the aberrations of the cornea and lens partially compensate for each other, affording the whole eye with better optical quality than either optical component alone. This is illustrated in Fig. 1 with plots of the wave aberration and point-spread functions of the cornea, internal optical surfaces (primarily the lens), and the eye as a whole, for one subject. They found partial compensation for corneal and lens aberrations such as spherical aberration and astigmatism, which are systematic from individual to individual. Compensation for these aberrations could result from evolutionary selection pressure, and need not imply the existence of active ocular aberration balancing mechanisms in each individual. However, surprisingly, they also found partial compensation for aberrations such as coma, which vary unsystematically across individual eyes in both sign and direction. Compensation of these aberrations in individual eyes appears less likely to result from evolution; it is conceivable that it involves some type of aberration-balancing mechanism in individual eyes. However, it is difficult to imagine in this case what the error signal could be and how it could be selective enough to control specific aberrations.

Cone phototropism

Autocalibration is an important feature of the photoreceptor mosaic as well as the eye's optics. The cone mosaic has a self-alignment mechanism that maximizes retinal image quality. Cones are long and thin and behave as optical waveguides because their refractive index is higher than the matrix surrounding them. Their optical axes are aligned with the eye's pupil so that they favor photons passing through the pupil center (see Fig. 2). A visual consequence of this alignment is the Stiles-Crawford effect, in which light entering through the pupil margins appears dimmer than light entering through the pupil center. Angular tuning of cones also rejects light scattered inside the eye, which is obliquely incident and cannot couple efficiently into each waveguide. This selective rejection of scattered light is a large effect: obliquely incident light is as much as 10 times less effective than light entering through the pupil center. The rejection of scattered light afforded by cone angular tuning is presumably especially valuable when the organism's survival depends on

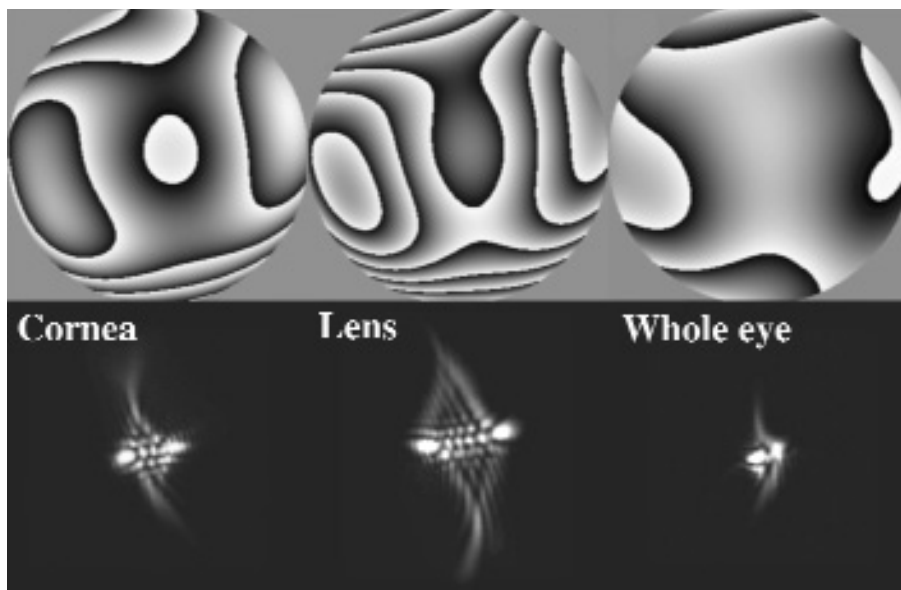


Figure 1. Grayscale plots of the wave aberration function and point-spread function of the front corneal surface, internal ocular surfaces (dominated by the eye's crystalline lens), and the whole eye, for one subject. The aberrations of the cornea and lens partially compensate, providing the whole eye with better optical quality than either element alone. (From Ref. 7.)

detecting dimly illuminated objects in an otherwise brightly illuminated scene, such as fruit shaded by sun-lit foliage, or more ominously, a predator lurking in the shadows.

It has been known for some time that the retina has a mechanism for automatically realigning photoreceptors in recovery from retinal detachment^{9,10} but the signal for realignment has not been clear. Smallman *et al.*¹¹ have recently provided conclusive evidence that the retina, like a phototropic plant, uses the direction of incident light itself for cone alignment. They studied the Stiles-Crawford effect in a patient who had lived with congenital cataracts, which obscured his natural pupils, for over 40 years. Throughout this period, he used eye drops to dilate his pupils enough so that light could pass around the cataract. Due to the asymmetry of the cataract, most of the light falling on his retina passed through the temporal edge of the pupil. Immediately after surgery was performed to remove the cataract, light passing through this same location at the temporal edge of the pupil was most effective in stimulating the retina, evidence that the cones were pointing toward the major source of the light. However, as shown in Fig. 3(b), soon after surgery, the peak of the Stiles-Crawford function shifted systematically toward the center of the new pupil, indicating a realignment of the cones over a period of about a week. This experiment clearly shows that photoreceptors are phototropic and actively align themselves to maximize their light-gathering capabilities while simultaneously rejecting unwanted stray light. Just how the retina senses the direction of incoming light, and what the particular mechanism is for tilting cones toward the light, remain a mystery.

NEURAL MECHANISMS

Autocalibration in the visual system is by no means confined to the eye's optics. It has long been appreciated that the neural machinery of vision provides exquisite compensation for changes in the visual environment as well as internal properties of the visual system. Barlow¹² has suggested that neural autocalibration is the visual system's way of removing redundancy in the neural code. A closely related idea is predictive coding,^{13,14} an efficient strategy in which only changes in a signal

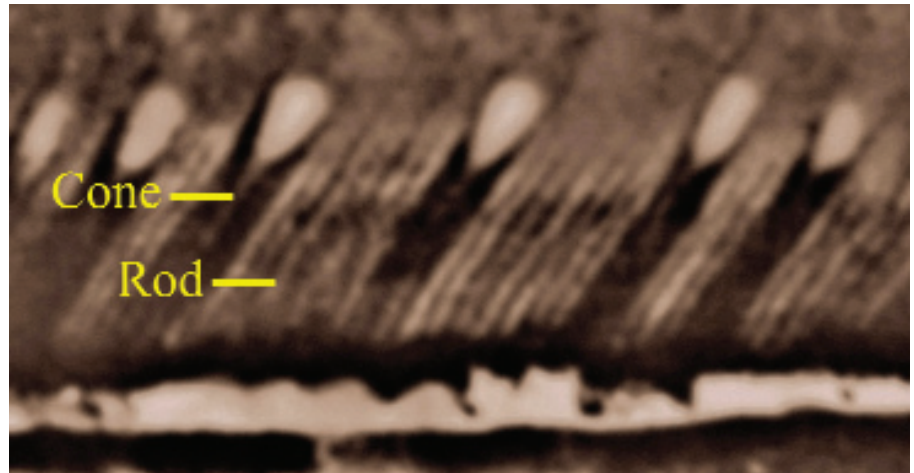


Figure 2. Fluorescence microphotograph of freeze-dried monkey retina showing the tilting of rods and cones toward the pupil center. At this eccentric retinal location the angle of the photoreceptors with respect to the underlying retinal surface is approximately 60 degrees. (From Ref. 8.)

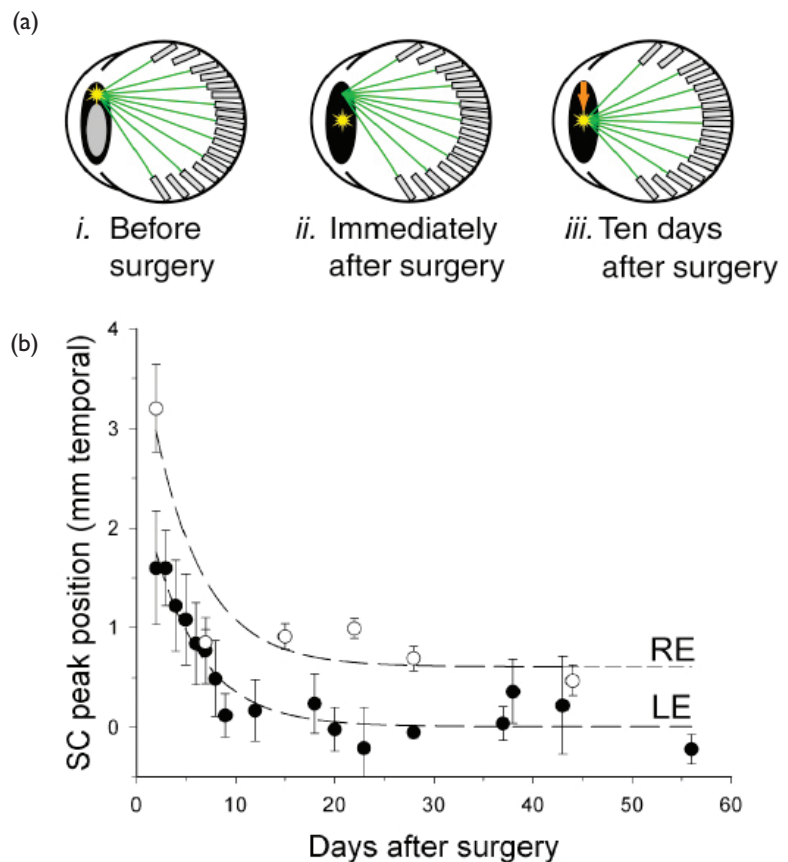


Figure 3. a) Schematic horizontal cross section through the cataract patient's eyes illustrating the pre-operative cataract position and photoreceptor alignment before, immediately after, and ten days after cataract removal surgery. The yellow sun indicates the average location through which light enters the pupil before and after surgery. b) Quantitative measurement of the Stiles-Crawford effect peaks, from which photoreceptor alignment can be inferred, for both of the subject's eyes in the days following cataract removal. After 10 days, photoreceptors had realigned towards the pupil center. (From Ref. 11.)

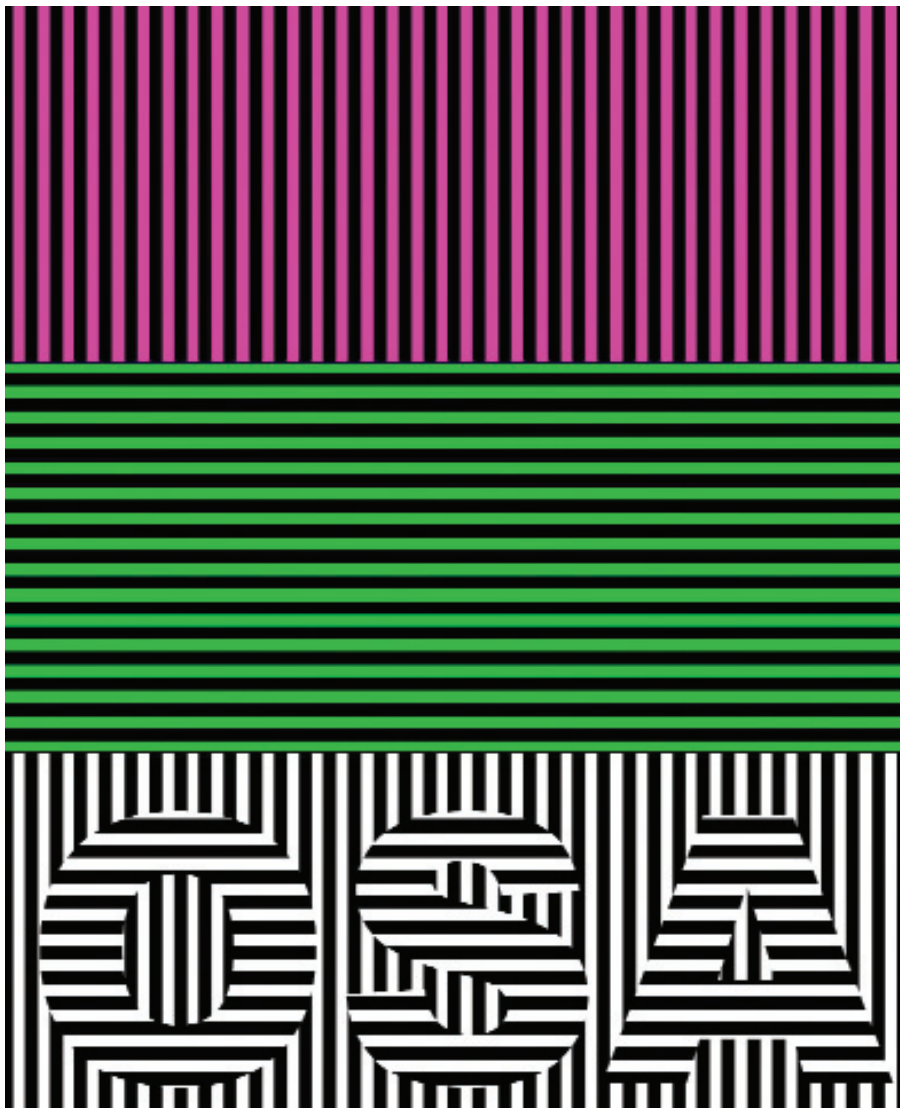


Figure 4. Demonstration of chromatic adaptation in orientation selective mechanisms, known as the McCollough effect after Celeste McCollough who discovered it in 1965. To induce the effect, look at one colored grating for approximately 15 seconds, then look at the other for 15 seconds. Continue to alternate gaze between the pink and green gratings in this way for five minutes. Feel free to move your eyes around the gratings, as constant fixation is not important for this effect. After five minutes of adaptation, look at the lower stimulus: all sections with vertical bars will appear pale green and the sections with horizontal bars will appear pale pink. Note that the induced color depends only on the orientation of the black and white bars, not on retinal location. Try looking again after an hour or even a day has passed: although it takes only a few minutes to induce, this effect can last for months or even years.

are transmitted and not the signal itself.

The process of light and dark adaptation provides a simple example. This process allows the eye to operate over the 11 orders of magnitude of light intensities encountered in everyday scenes even though the output neurons from the retina can only operate over two-three orders.¹⁵ This is accomplished with machinery that continuously matches the sensitivity of the retina to the range of intensities present in a particular scene. Information about ab-

solute intensity, which is not especially valuable, is discarded, while information about contrast, which contains critical information about objects and their locations, is preserved. The mechanism works by modulating sensitivity based on an estimate of light intensity averaged over some spatio-temporal window. If the averaging window is too short in time, the signal itself will be lost. If it is too long, the visual system will not be nimble enough to adapt to rapid changes in ambient light level.

An inevitable consequence of the averaging window is the existence of an after effect when the light intensity has changed too much in too short a time. The after image caused by a flash bulb is a familiar example. Indeed, the literature on human vision is replete with examples of after effects beyond the simple after images produced by light adaptation. After effects have been reported that involve all fundamental dimensions of visual stimuli, including color, motion, orientation, spatial frequency, and form. These effects, while often described as errors in vision, actually reflect valuable autocalibration mechanisms that compensate for many different kinds of distortions or changes in scenes that would otherwise interfere with a stable appearance of the visual world.

Many people experience a visual field distortion when they first don a new pair of spectacles. Straight lines can look curved and objects may be displaced in the field of view. These effects dissipate rapidly in time as autocalibration mechanisms restore an undistorted interpretation of the scene. The visual system probably compensates for curvature distortion by a perceptual renormalization that largely discounts the average curvature in the visual scene,¹⁶ and by requiring a constant shape of rigid objects as they move across the visual field.¹⁷ Additional autocalibration mechanisms involve sensory modalities other than vision. For example, people can learn to operate in a world that is optically inverted, as the motor system adjusts to respond appropriately despite the visual distortion.¹⁸

Color autocalibration

Color vision also reveals neural autocalibration mechanisms at play. For example, the fact that after images of colored objects often have the complementary hue reflects a color balancing process that contributes to keep the colors of objects constant despite changes in the illumination falling on them. As another example, it is well known that the chromatic fringes that can be clearly seen when first wearing dispersing prisms disappear completely within a few weeks.¹⁹ When the prisms are removed, complementary fringes appear. Some readers may have experienced this calibration first hand, since a new pair of spectacles, especially with a strong prescription, can induce colored fringes as well as optical distortions that disappear

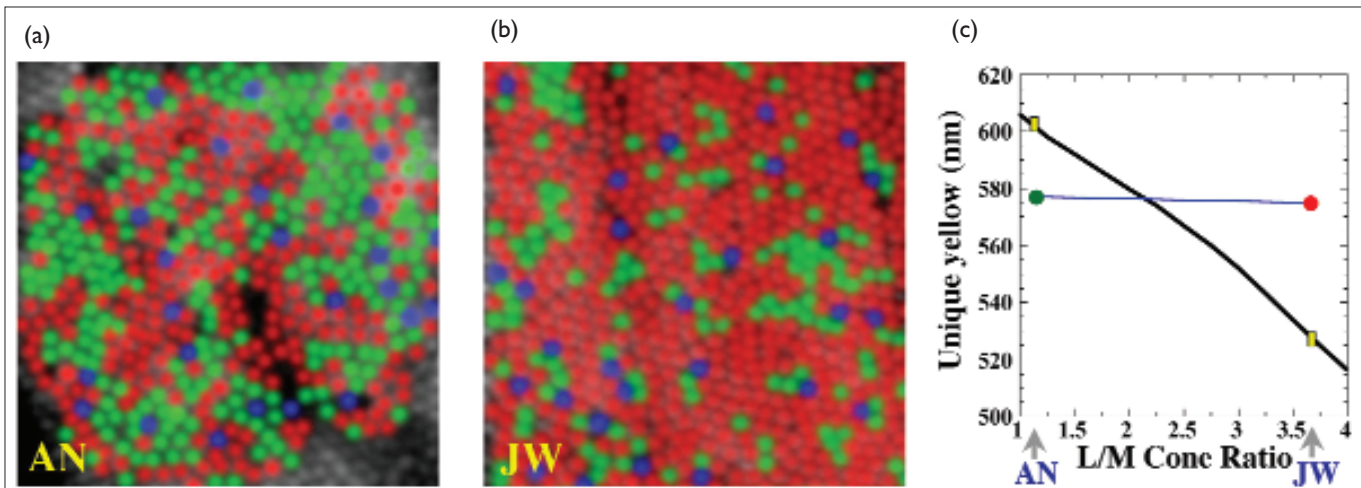


Figure 5. (a) and (b) False color images of the trichromatic retinal mosaics at 1 degree retinal eccentricity for two human subjects determined by direct retinal imaging with adaptive optics. L, M, and S cones are indicated by red, green, and blue, respectively. The two subjects exhibit very different relative numerosities of L and M cones. L:M is 1.15 for AN and 3.79 for JW. (c) Despite the large differences in relative cone numbers, both subjects see the same wavelength as uniquely yellow. The green and red circles indicate each subject's uniquely perceived yellow; the yellow rectangles indicate the unique yellow setting for each subject predicted by a theory of color vision in which the relative numbers of L and M cones determine red-green color perception.²¹ [(a) and (b) from Ref. 20; (c) from Ref. 22.]

over time. The optics of the eye can create its own chromatic fringes due to the existence of transverse chromatic aberration. Perhaps chromatic fringe adaptation is the visual system's way of compensating for this optical flaw. These fringes are most prominent for edges that are oriented perpendicular to the direction of transverse chromatic aberration. Thus, an effective compensation mechanism must be capable of modifying color appearance depending on the orientation of edges. Just such a mechanism is revealed by the after effect demonstrated in Fig. 4.

Autocalibration mechanisms may play an even more significant role in vision. Perhaps they are responsible for establishing the fundamental properties of visual experience during development. For example, what is it that establishes in each of us the relationship between the three cone signals upon which color vision depends and the subjective hues we experience? Recent evidence makes it clear that people with very different retinas perceive colors in the same way. Using adaptive optics to image the living human retina at high resolution, Roorda and Williams²⁰ were able to determine the arrangement of the three cone classes in the retinal mosaic for two subjects, shown in Fig. 5 (a) and (b). These two subjects have extremely different retinas: the numbers of S cones are similar but JW has nearly four times more L cones per M cone than AN. According to one theory of color vision, this should



Figure 6. In the long-term chromatic adaptation experiment of Yamauchi *et al.*²⁵ subjects either spent several hours a day in the red or green room, wore colored contacts, or wore colored goggles. (Image courtesy: Yasuski Yamauchi.)

cause the subjects to have very different hue perception.²¹ For example, light that looks yellow to AN should look more reddish to JW, since he has more L cones, and thus a stronger L cone signal. Remarkably, despite their very different retinas, Fig. 5 (c) shows that these subjects had nearly identical color perception.²² Carroll *et al.*²³ have provided additional evidence that people with very different retinal mosaics share similar color perception. They found, for a large subject population, that the perception of unique yellow was simi-

lar from subject to subject, and did not correlate with the relative numerosity of L and M cones as estimated by flicker-photometric electroretinography.²⁴

Though different people have very different retinas, we all share a similar visual environment during development. If the visual system contains an autocalibration mechanism that tunes itself to the chromatic environment, then people with different relative numbers of cones could develop similar color vision. If this autocalibration mechanism exists, then it should

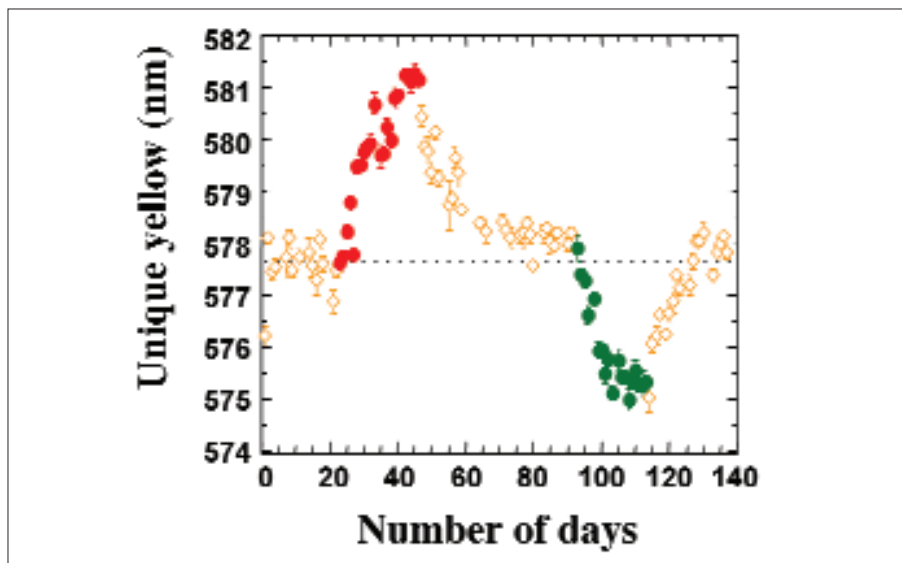


Figure 7. The shift in the wavelength perceived as uniquely yellow for one subject during the time course of chromatic adaptation. Measurements were performed each morning prior to that day's adaptation period, and thus do not reflect the effects of short-term color adaptation. Red and green symbols represent measurements made during the periods of adaptation to a red or green chromatic environment. Orange symbols represent measurements made before adaptation and during recovery to the chromatic adaptation. (From Ref. 25.)

be possible to alter a person's color perception by exposing them to an unusual chromatic environment for an extended period of time. Recently, Yamauchi *et al.*²⁵ confirmed this hypothesis by exposing subjects to an altered chromatic environment (either more red or more green than normal) for 4-12 hours per day for many days. Adaptation was accomplished by wearing tinted contact lenses, tinted goggles, or spending time in a red or green room (see Fig. 6). After several days, the wavelength which subjects perceived as uniquely yellow, which represents a boundary between reddish and greenish lights in the spectrum, significantly shifted in the direction required to compensate for the chromatically altered environment. The time course of this shift is shown for one subject in Fig. 7. The maximum shift occurred after 15 days of adaptation, and it took more than a week, once adaptation was complete, for this after effect to decay. These shifts are consistent with observations that color vision remains remarkably constant across the lifespan despite large changes in the spectral transmittance of the lens with age.²⁶ The phenomenon is consistent with predictive coding of color information in which the visual system calibrates itself so that only departures from the average chromaticity are transmitted. If the visual system normalizes its

response in this way,²⁷ then perhaps the hue boundaries that we experience follow naturally from a simple and efficient coding scheme. The mechanism responsible for the adaptation effect described by Yamauchi *et al.* in adulthood could be the remnants of a more vigorous mechanism that operates during development to establish hue perception. Perhaps future work on developing eyes will establish whether this process does indeed shepherd vision toward a consistent pattern of hue perception in adults.

Summary

The human visual system relies on a host of autocalibration mechanisms that have evolved to maintain proper visual function throughout life despite enormous internal and environmental changes. Optical mechanisms exist that optimize the quality of the retinal image and the retina's capability to detect it throughout development. Two classes of neural mechanisms exist, some correct for internal imperfections in the visual system, such as optical distortion, transverse chromatic aberration, and irregularities in the retinal mosaic. Others, such as light adaptation and color calibration, compensate for changes in the visual environment that would otherwise interfere with our ability to recog-

nize objects or visual scenes under different circumstances. After effects that occur with sudden changes in the visual environment reflect the valuable workings of these mechanisms. Although the existence and utility of optical and neural autocalibration in vision is clear, the actual machinery that accomplishes autocalibration largely remains to be discovered.

References

1. E. L. Smith, *Optom. Vision Sci.* **75**, 388-98 (1998).
2. C. F. Wildsoet and J. Wallman, *Invest. Ophthalmol. Visual Sci. (Suppl.)* **33**, 1806 (1992).
3. J. Wallman, M. D. Gottlieb, V. Rajaram, and L. A. Fugate-Wentzke, *Science* **237**, 73-7 (1987).
4. C. F. Wildsoet, H. C. Howland, S. Falconer, and K. Dick, *Vision Res.* **33**, 1593-603 (1993).
5. R. D. Sperduto, D. Seigel, J. Roberts, and M. Rowland, *Arch. Ophthalmol. (Chicago)* **101**, 405-7 (1983).
6. H. M. Wu, B. Seet, E. P. Yap, S. M. Saw, T. H. Lim, K. S. Chia, *Optom. Vision Sci.* **78**, 234-9 (2001).
7. P. Artal, A. Guirao, E. Berrio, D. R. Williams, *J. Vis.* **1**, 1-8 (2001).
8. A. M. Laties, J. M. Enoch, *Invest. Ophthalmol.* **10**, 69-77 (1971).
9. J. M. Enoch, J. A. Van Loo, and E. Okun, *Invest. Ophthalmol.* **12**, 849-53 (1973).
10. F. Fankhauser, J. M. Enoch, and P. Cibis, *Am. J. Ophthalmol.* **52**, 767-83 (1961).
11. H. S. Smallman, D. I. A. MacLeod, and P. Doyle, *Nature* **412**, 604-5 (2001).
12. H. B. Barlow, A theory about the functional role and synaptic mechanism of visual after-effects, in: C. Blakemore, *Vision: Coding and Efficiency*, Cambridge University Press (1990).
13. A. L. Fairhall, G. D. Lewen, W. Bialek, and R. R. de Ruyter van Stevenick, *Nature* **412**, 787-92 (2001).
14. M. V. Srinivasan, S. B. Laughlin, and A. Dubs, *Proc. R. Soc. Lond. B* **216**, 427-59 (1982).
15. S. W. Kuffler, *J. Neurophysiol.* **16**, 37-68 (1953).
16. D. P. Andrews, *Q. J. Exp. Psych.* **16**, 104-15 (1964).
17. I. Rock, *The Nature of Perceptual Adaptation*, New York, Basic Books (1966).
18. G. M. Stratton, *Psychol. Rev.* **3**, 341-60 (1897).
19. R. Held, "The rediscovery of adaptability in the visual system: effects of extrinsic and intrinsic chromatic dispersion," in: C. S. Harris, Lawrence Erlbaum Associates, Inc. (1980).
20. A. Roorda and D. R. Williams, *Nature* **397**, 520-2 (1999).
21. S. Otake and C. M. Cicerone, *J. Opt. Soc. Am. A - Optics & Image Science*, **17**, 615-27 (2000).
22. D. H. Brainard, A. Roorda, Y. Yamauchi, J. B. Calderone, A. Metha, M. Neitz, J. Neitz, D. R. Williams, and G. H. Jacobs, *J. Opt. Soc. Am. A - Optics & Image Science* **17**, 607-14 (2000).
23. J. Carroll, J. Neitz, M. Neitz, Evaluation of the flicker photometric electroretinogram as a method for estimating L:M cone ratios in human eyes, OSA Annual Meeting, Providence, RI, (2000).
24. J. Carroll, C. McMahon, M. Neitz, and J. Neitz, *J. Opt. Soc. Am. A - Optics & Image Science* **17**, 499-509 (2000).
25. Y. Yamauchi, D. R. Williams, J. Carroll, J. Neitz, and M. Neitz, Chromatic adaptation can cause long term shifts in color appearance that arise in binocular visual pathways, Assoc. for Res. in Vis. and Ophthal. Annual Meeting, #3873 (2001).
26. J. S. Werner and B. E. Scheffrin, *J. Opt. Soc. Am. A - Optics & Image Science*, **10**, 1509-16 (1993).
27. J. Pokorny and V. C. Smith, *Die Farbe* **34**, 53-7 (1987).

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