References and Notes

1. C. B. Tanner, *Agron. J.* **55**, 210 (1963); D. M. Gates, *ibid.* **56**, 273 (1964); C. L. Wiegand and L. N. Namken, *ibid.* **58**, 582 (1966); W. L. Ehrler, *ibid.* **65**, 404 (1973).
2. S. B. Idso, R. D. Jackson, R. J. Reginato, *Science* **196**, 19 (1977); R. D. Jackson, R. J. Reginato, S. B. Idso, *Water Resour. Res.* **13**, 651 (1977); R. J. Reginato, S. B. Idso, R. D. Jackson, *Remote Sensing Environ.* **7**, 77 (1978); W. L. Ehrler, S. B. Idso, R. D. Jackson, R. J. Reginato, *Agron. J.* **70**, 999 (1978).
3. Infrared photography and infrared thermometry are fundamentally different. Leaf temperatures cannot be recorded with infrared photography because the film is not sensitive to emitted thermal energy [N. L. Fritz, *Photogramm. Eng.* **33**, 1128 (1967)]. Infrared radiation thermometers measure emitted thermal radiation; they thus provide a remote, noncontact means to observe radiant leaf temperatures and infer rates of evapotranspiration. Several earlier studies have demonstrated that infrared thermometry facilitates the identification of biological stress in certain forest and agricultural species [R. C. Heller, *Proc. Symp. Remote Sensing Environ.* **5**, 387 (1968); C. De Carolis, G. Baldi, S. Galli de Paratesi, G. M. Lechi, *ibid.* **9**, 1161 (1974); C. De Carolis, G. G. Conti, G. M. Lechi, *ibid.* **10**, 1219 (1976); C. E. Olson, Jr., *ibid.* **11**, 933 (1977); R. Kumar, L. F. Silva, M. E. Bauer, *Publication N78-29537* (National Technical Information Service, Springfield, Va., 1978)].
4. M. E. Stanghellini and E. L. Nigh, *Plant Dis. Rep.* **56**, 507 (1972); M. E. Stanghellini and J. D. Russell, *Phytopathology* **63**, 133 (1973).
5. R. B. Streets and H. E. Bloss, *Am. Phytopathol. Monogr. 8* (1973); H. E. Bloss and R. B. Streets, Sr., *Prog. Agric. Ariz.* **24**, 8 (1972); C. M. Rush and S. D. Lyda, in *Proceedings of the Beltwide Cotton Production Research Conferences* (National Cotton Council, Memphis, Tenn., 1978), p. 23.
6. For the sugar beets we used a Barnes PRT-5 infrared thermometer with a 2° field-of-view lens; for the cotton we used a Telatemp AG-42 infrared thermometer with a 2.5° field-of-view lens. Both were equipped with a 10.5- to 12.5-μm band-pass filter. Radiant temperatures were not corrected for target emissivity. Trade and company names are included for the benefit of the reader but imply no endorsement or preferential treatment of the product cited by the U.S. Department of Agriculture.
7. J. H. Zar, *Biostatistical Analysis* (Prentice-Hall, Englewood Cliffs, N.J., 1974), p. 70.
8. The frequency distributions of slightly diseased plants are not shown in Fig. 1, A and B, because they were essentially coincident with that of healthy plants.
9. We compared observed versus predicted disease incidence for healthy and moderately diseased categories, using 2×2 χ^2 contingency tables (sugar beets, $\chi^2 = 15.3$; cotton, $\chi^2 = 59.2$).

10. We thank T. N. Jordan, W. C. Kronland, and R. S. Seay for technical assistance. Contribution from the Agricultural Research, Science, and Education Administration, U.S. Department of Agriculture, Phoenix, Ariz.; the University of Arizona, Tucson; and the Amstar Corporation, Spreckels Sugar Division, Chandler, Ariz.

15 January 1979; revised 2 April 1979

Enchancement of Luminance Flicker by Color-Opponent Mechanisms

Abstract. *Color-opponent ganglion cells in the monkey retina respond to luminance flicker at high temporal frequencies. Color opponency, which makes these cells so selective of wavelength at low temporal frequencies, is progressively lost at high frequencies. This loss is due to a frequency-dependent phase shift between the responses of spectrally different center and surround mechanisms in the receptive field of each of these cells. Center and surround responses, which are antagonistic at low temporal frequencies, become synergistic at high ones, making these cells most responsive at high frequencies to those wavelengths to which they are least responsive at low frequencies. This phenomenon can explain the differences between chromatic and luminance flicker in human vision.*

Periodic changes in the luminance of a light produce a sensation of flicker. If these changes in luminance are large, flicker can be observed at frequencies as high as 70 to 80 Hz. Flicker can also be observed if the chromaticity (wavelength composition) of a light is varied, while luminance is kept constant. At low frequencies this chromatic flicker is more effective than luminance flicker but at high frequencies the converse is true (*1*, *2*). The neurophysiological basis for this difference between the perception of luminance and chromatic flicker is unknown. Suggestions have been made (*2*, *3*) that these perceptual differences could be due to the properties of two distinct classes of cells detectable in primate retina (*4*) and lateral geniculate nucleus (*5*): one class, which is wavelength selective (the so-called color-opponent cells), and the other, which does not show color opponency and is consequently selective only for the effective energy of luminance of light.

We have examined this hypothesis in single ganglion cells subserving the central retina of the rhesus monkey. The physiology of one class of cells, the color-opponent variety alone, may provide the entire explanation for the differences between chromatic and luminance flicker functions. Color-opponent cells respond better to chromatic changes at low temporal frequencies and better to luminance changes at high temporal frequencies. This phenomenon is due to a frequency-dependent phase shift between the color-opponent responses produced by the center and surround mechanisms in the receptive field of each of these cells. What are spectrally antagonistic responses at low frequencies become progressively synergistic at high frequencies.

We recorded the extracellular impulses of single ganglion cells in the retina of monkeys (*Macaca mulatta*) anesthetized with sodium pentobarbital (Nembutal, 5 mg per kilogram of body weight per hour, injected intravenously), paralyzed with gallamine triethiodide (Flaxedil, 10 mg kg^{-1} $hour^{-1}$, injected intravenously), and artificially ventilated; CO_2 in the expired air was continuously monitored and kept within the range of 4 to 5 percent. Recordings were made with glass micropipette electrodes filled with 3*M* KCl solutions, which were introduced into the eye through a closed chamber and guided to the retina by a hydraulic micromanipulator under direct vision (with a modified Bausch and Lomb fundus camera). The camera provided a means of presenting visual stimuli in Maxwellian view, which could be seen in sharp focus on the retinal surface (*4*). The light source was a 1000-W xenon arc lamp providing two independent beams. Flickering stimuli with equal periods of light and darkness were produced by a half-sectored disk rotated at a focal point in one beam by a variable-speed motor and monitored continuously by a photocell. The wavelength and energy in this beam were changed by narrow-band interference and neutral density filters, respectively; sharp cut absorption filters (Corning 2408, red; 3482, yellow; and 5543, blue) in the second beam provided selective chromatic adaptation. Fusion and response latencies to increasing flicker frequencies were determined ($\pm$ 1 msec) by superimposing 10 to 20 responses and stimuli on a double-beam storage oscilloscope, which provided a continuous monitor of the phase relationship of the response to the stimulus cycle.

We classified 385 single ganglion cells in the central retina (0° to 12° eccentricity) according to the position of its receptive field center and the cone mechanisms influencing its responses (*6*). The flicker fusion frequencies of 38 cells were studied intensively; 30 of these showed color opponency. In color-opponent cells both center and surround mechanisms in a cell's receptive field could usually be studied separately by an appropriate choice of wavelength. In general, the flicker fusion frequency of all cells increased with the intensity of light stimulation. The highest flicker fusion frequencies were attained by phasic ganglion cells that did not show color opponency. The average flicker fusion frequency of phasic cells was 62 Hz [$N = 8$, standard deviation (S.D.) = 11]. In the parafoveal retina, where such phasic cells are common, color-opponent cells also follow high flicker frequencies. Those whose receptive field centers were mediated by the red cone mechanism had an average maximum flicker fusion frequency of 53 Hz

0036-8075/79/0810-0587$00.50/0

(N = 12; S.D. = 13). Color-opponent cells with green and blue cone mechanisms mediating the responses of receptive field centers had lower maximum fusion frequencies, 49 Hz (N = 3, S.D. = 4) and 38 Hz (N = 2, S.D. = 2), respectively. In the fovea the maximum flicker fusion frequencies of color-opponent cells are lower; those with red, green, and blue cone mechanisms subserving their receptive field centers are, respectively, 33 Hz (N = 5, S.D. = 10), 36 Hz (N = 6, S.D. = 16), and 29 Hz (N = 2, S.D. = 9).

With increasing flicker frequencies, color-opponent cells begin to lose their color opponency and consequently their color selectivity (Fig. 1A). At low flicker rates this cell was excited by long wavelengths (666 nm) and inhibited by short wavelengths (456 nm). The converse pattern of excitation and inhibition occurs when the light goes off. Midspectral (530 nm) light is ineffective because it stimulates the opposing cone mechanisms similarly so that phases of excitation and inhibition antagonize each other almost equally and the modulated response to the flickering stimulus is lost. At medium flicker rates the effectiveness of midspectral light begins to increase, and modulated responses of the ganglion cell, time-locked to the stimulus, are produced. At higher flicker rates midspectral lights become more effective than any other for eliciting responses to flicker.

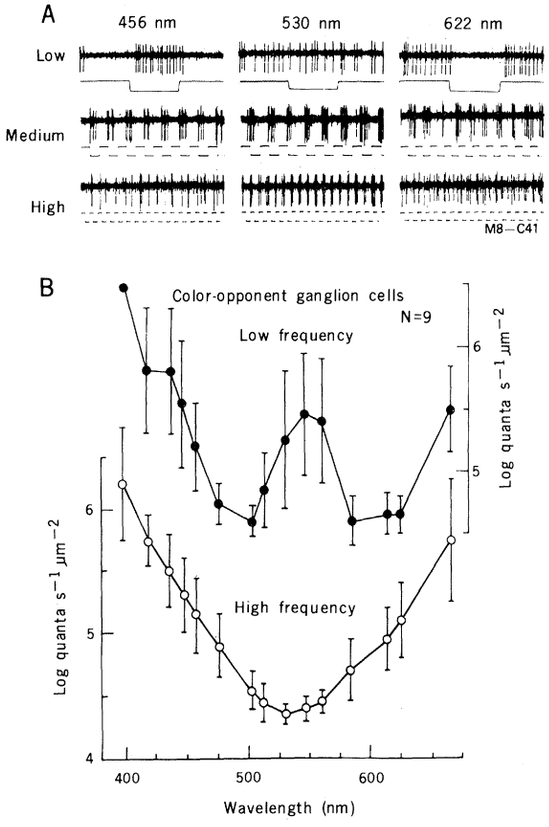

Fig. 1. (A) Flicker responses of a red ON-center, green OFF-surround color-opponent ganglion cell at three different frequencies (low, 1.1 Hz; medium, 15 to 18 Hz; high, 33 Hz) at three different wavelengths of equal energy ($10^{4.8}$ quanta/sec/μm^2). The stimulus was 20° in diameter and presented on a white background (20,000 trolands). At medium and high frequencies, three oscillographic records are superimposed. The response of a photocell to the light stimulus is shown below each response of the cell. (B) Averaged action spectra of nine red-green color-opponent ganglion cells based on a flicker threshold criterion at low (1.2 Hz, filled circles) and high (33 Hz, open circles) frequencies of stimulation on a white background of 20,000 trolands. Curves were combined at their mean maximum sensitivity; vertical axes are split (upper right, lower left) to avoid overlap.

The average action spectra of nine ganglion cells showing color opponency between red and green cone mechanisms (Fig. 1B) are based on the quantal energy required at different wavelengths to elicit a threshold flicker response at low (1 Hz) and high (33 Hz) frequencies. At low frequencies, the lowest thresholds for eliciting flicker occur in the orange-red and (with opposite polarity, that is, with responses 180° out of phase) in the blue-green parts of the visible spectrum. Midspectral wavelengths, at the peak of the human luminosity function, are ineffective at low frequencies because of color opponency. The spectral neutral point varies among red-green opponent cells but is mostly within the range of 500 to 600 nm. At high flicker rates, the thresholds of midspectral lights for producing flicker decrease, and this region becomes more effective than all other regions of the spectrum for producing a flicker response in color-opponent cells.

The reason for this phenomenon lies in temporal interactions between the spectrally different center and surround

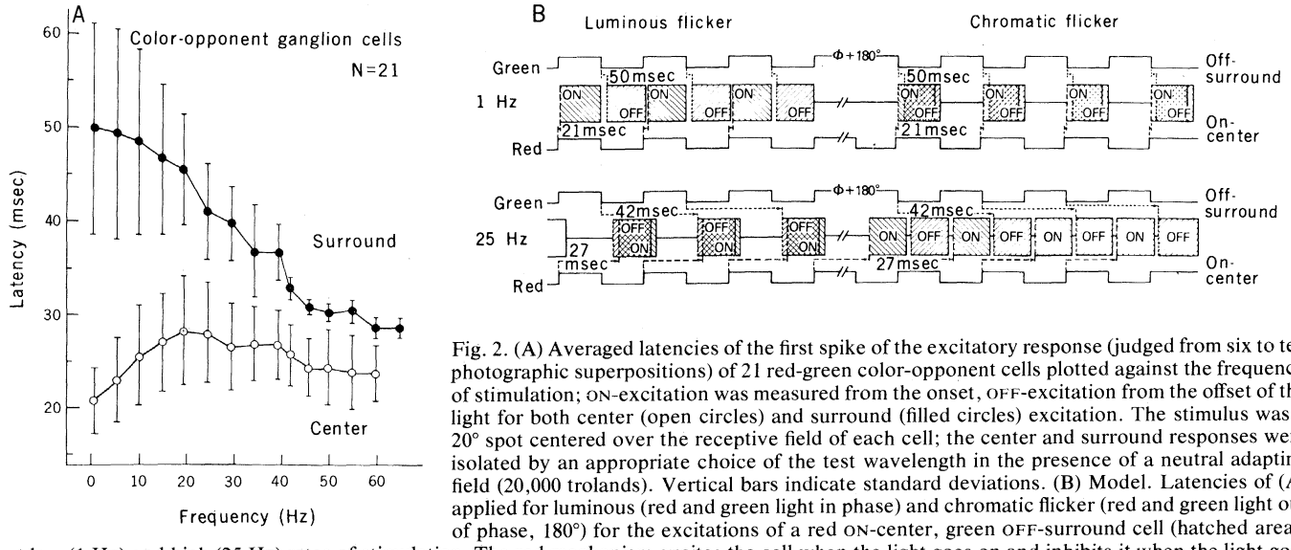

Fig. 2. (A) Averaged latencies of the first spike of the excitatory response (judged from six to ten photographic superpositions) of 21 red-green color-opponent cells plotted against the frequency of stimulation; ON-excitation was measured from the onset, OFF-excitation from the offset of the light for both center (open circles) and surround (filled circles) excitation. The stimulus was a 20° spot centered over the receptive field of each cell; the center and surround responses were isolated by an appropriate choice of the test wavelength in the presence of a neutral adapting field (20,000 trolands). Vertical bars indicate standard deviations. (B) Model. Latencies of (A) applied for luminous (red and green light in phase) and chromatic flicker (red and green light out of phase, 180°) for the excitations of a red ON-center, green OFF-surround cell (hatched areas) at low (1 Hz) and high (25 Hz) rates of stimulation. The red mechanism excites the cell when the light goes on and inhibits it when the light goes off; the green mechanism acts conversely. Only the excitatory responses are illustrated. A resonance between center and surround responses occurs for chromatic flicker at low and for luminous flicker at high frequencies.

mechanism of color-opponent cells. Figure 2A shows the average latencies at different flicker frequencies of the excitatory responses generated by the receptive field center and surround mechanisms of 21 color-opponent cells showing color opponency between red and green cone mechanisms. Excitation from the surround occurs later than that from the center mechanism in both ON- as well as OFF-center cells. The difference decreases with increasing flicker frequencies but is never eliminated. This frequency-dependent change in phase difference between the responses of center and surround mechanisms progressively turns the cone antagonism (color opponency) so characteristic of these cells at low frequencies into synergism at high frequencies.

The way this occurs can be determined from latency measurements (Fig. 2A) for both luminous and chromatic flicker; Fig. 2B shows this for a red ON-center, green OFF-surround cell. For luminance flicker a mixture of red and green light (equals yellow when in phase) was used to stimulate the cell at low and high frequencies. At low frequencies the latency difference between the response of the center and surround mechanisms was insignificant relative to the long duration of the stimulus. Excitatory (hatched blocks) as well as inhibitory responses (not shown) produced by the two mechanisms were totally out of phase, so that they canceled each other. Consequently, the cell generated little to no modulated response to yellow light at low frequencies (*7*). As flicker frequency increased, the difference in latency between the responses of center and surround mechanisms began to turn antagonism into synergism in what appears to be a resonance between these two responses; that is, the excitatory as well as the inhibitory responses in both center and surround occurred simultaneously. This resonance was most effective at precisely those wavelengths which produced strong responses from both center and surround mechanisms and consequently had the weakest responses at low temporal frequencies. Therefore, action spectra based on flicker criteria for color-opponent cells gradually changed with frequency; midspectral wavelengths, which are relatively ineffective at low, became more effective at high flicker frequencies and color opponency was progressively lost. The broadband and single peaked luminosity function based on heterochromatic flicker photometry (*1*) may be an inevitable consequence of this phenomenon, since all cells become single peaked and broadband at higher flicker frequencies. A similar synchronization between center and surround mechanisms induced by eye movements may also broaden the action spectrum of color-opponent cells detecting fine lines or borders (*8*).

Chromatic flicker produced the converse behavior (Fig. 2B). The red and green stimuli were 180° out of phase, producing flicker that alternated only in chromatic composition but not in luminance. At low frequencies, the excitatory (and inhibitory) responses from the center (ON) and surround (OFF) mechanisms of such a cell synchronized and a strong response to flicker was produced. At high frequencies, these two responses became completely out of phase and the modulated response to chromatic flicker was lost.

The center and surround mechanisms of primate color-opponent retinal ganglion cells seem organized to enhance successive color contrast at low and luminance contrast at high temporal frequencies. At low frequencies, chromatic flicker, the most effective stimulus for these cells, must underlie the low subjective thresholds to such flicker (*1*, *2*); at high frequencies, the increasingly greater effectiveness of luminance flicker must account for its becoming more effective subjectively. This enhancement of flicker by center-surround interaction may be a general property of all cells with concentrically organized receptive fields (*9*). The phenomenon is particularly striking in color-opponent cells, where both spectral and spatial opponency are combined to enhance luminance flicker (*10*).

The loss of color opponency in ganglion cells at high flicker frequencies implies that their impulses alone cannot unambiguously code for color. The color signals they transmit must depend upon comparisons between the phase relationship of different cells, subserving neighboring areas of the retina. This detector, presumably the visual cortex, must work optimally only at low temporal frequencies because color-opponent signals that carry the information upon which this detector depends are progressively lost at higher frequencies.

P. GOURAS

Columbia University,
College of Physicians and Surgeons,
630 West 168th Street,
New York 10032

E. ZRENNER

Max-Planck-Institute for Physiological and Clinical Research,
W. G. Kerckhoff Institute,
Parkstrasse 1, D-6350 Bad Nauheim,
West Germany

References and Notes

1. H. De Lange, *J. Opt. Soc. Am.* **48**, 784 (1958); Y. Galifret and H. Pieron, *Rev. Opt. Theor. Instrum.* **28**, 154 (1949).
2. D. H. Kelly and D. van Norren, *J. Opt. Soc. Am.* **67**, 1081 (1977).
3. P. E. King-Smith and D. Carden, *ibid.* **66**, 709 (1976); C. R. Ingling, Jr., *ibid.* **68**, 1143 (1978).
4. P. Gouras, *J. Physiol. (London)* **199**, 533 (1968); *ibid.* **204**, 407 (1969); F. De Monasterio and P. Gouras, *ibid.* **251**, 167 (1975); E. Zrenner and P. Gouras, *Pfluegers Arch.* **377** (Suppl.) R48 (1978).
5. R. L. De Valois, *Cold Spring Harbor Symp. Quant. Biol.* **30**, 567 (1965); T. N. Wiesel and D. H. Hubel, *J. Neurophysiol.* **29**, 1115 (1966); P. H. Schiller and J. G. Malpeli, *ibid.* **40**, 428 (1977); B. Dreher, Y. Fukada, R. W. Rodieck, *J. Physiol. (London)* **258**, 433 (1976); O. D. Creutzfeldt and B. B. Lee, *ibid.* **284**, 116P (1978); J. Krüger, *Exp. Brain Res.* **30**, 297 (1977).
6. The receptive field center of each cell was mapped with a small (10′ of arc) spot of light (either white or monochromatic); the cell's action spectrum was determined with 15 different wavelengths extending from 399 to 666 nm on a neutral (white) background with the small and a large (20°) stimulus. Phasic cells formed 29 percent of our sample; 55 ON-center and 37 OFF-center cells; none showed color opponency. Tonic cells formed 67 percent of our sample; 86 percent showed color opponency. Color opponency was established when the response of a cell changed from ON-excitation–OFF-inhibition to ON-inhibition–OFF-excitation by changing the wavelength of stimulation. In some cells this could be established only by redetermining the cell's action spectrum in the presence of a chromatic adapting field. The cone mechanism sensitive to short wavelengths (blue) was considered to be present only when the action spectrum, obtained on a strong red adapting field (approximately 300,000 trolands, Corning filter 2408), had a maximum sensitivity in the range of 440 to 460 nm and decreased at least tenfold in sensitivity at 500 nm. By this criterion, 22 of 385 cells had an input from blue cones; all were color opponent, and in 21 the blue cone input was excitatory. Most of the color-opponent cells (180 of 202) appeared to involve antagonistic interactions between red and green cone mechanisms. This report is concerned with these color-opponent cells, although a similar phenomenon appears to occur in those receiving inputs from blue cones.
7. The phasic ganglion cell system may compensate for the unresponsiveness of the tonic or color-opponent system to low-frequency midspectral lights.
8. The minimum distinct border technique reveals broadband functions similar to that obtained by heterochromatic flicker photometry [G. Wagner and R. M. Boynton, *J. Opt. Soc. Am.* **62**, 1508 (1972)].
9. An amplification of flicker by facilitative interaction between center and surround mechanisms was shown in Limulus [F. Ratliff, B. W. Knight, J. Toyoda, H. K. Hartline, *Science* **158**, 392 (1967); F. Ratliff, B. W. Knight, N. Graham, *Proc. Natl. Acad. Sci. U.S.A.* **62**, 733 (1969)] and is also implied in the analysis of G. V. Bekesy [*Vision Res.* **8**, 1483 (1968)]. Its occurrence in vertebrates is suggested by several flicker studies of cat retinal ganglion cells [C. Büttner, U. Büttner, O.-J. Grüsser, *Pfluegers Arch.* **322**, 1 (1971); L. Maffei, L. Cervetto, A. Fiorentini, *J. Neurophysiol.* **33**, 276 (1970)]. Isolation and interaction of center and surround mechanisms are difficult to achieve without changing spatial variables in cat ganglion cells in which the spectral properties of these opposing mechanisms seldom differ.
10. This possibility was suggested by R. L. De Valois, D. M. Snodderly, E. W. Yund, N. K. Hepler [*Sens. Processes* **1**, 244 (1977)], but several studies of the flicker properties of cells in the lateral geniculate nucleus of monkeys had not yet revealed such an effect [H. Spekreijse, D. van Norren, T. J. T. P. van den Berg, *Proc. Natl. Acad. Sci. U.S.A.* **68**, 2802 (1971); P. K. Kaiser, D. W. Blick, J. R. Tuttle, J. L. Brown, *J. Opt. Soc. Am.* **67**, 1379 (1977)].
11. We thank R. Nelson for his suggestions and E. Dodt for his encouragement and support. E.Z. was supported by PHS international research fellowships F05TW2429-01 and F05TW2429-02. P.G. was supported by NIH research grant EY02591 from the National Eye Institute and in part by a professorship award from Research to Prevent Blindness, Inc.

4 December 1978; revised 21 May 1979