Fourier models and the loci of adaptation

W. L. Makous

Center for Visual Science, University of Rochester, Rochester, New York 14627

Received January 9, 1997; revised manuscript received March 28, 1997; accepted March 31, 1997

First measures of sensitivity and the need for a model to interpret them are addressed. Then modeling in the Fourier domain is promoted by a demonstration of how such an approach explains spatial sensitization and its dependence on luminance. Then the retinal illuminance and receptor absorptions produced by various stimuli are derived to foster interpretation of the neural mechanisms underlying various psychophysical phenomena. Finally, the sequence and the anatomical loci of the processes controlling visual sensitivity are addressed. It is concluded that multiplicative adaptation often has effects identical to response compression followed by subtractive adaptation and that, perhaps as a consequence, there is no evidence of retinal gain changes in human cone vision until light levels are well above those available in natural scenes and in most contemporary psychophysical experiments; that contrast gain control fine tunes sensitivity to patterns at all luminances; and that response compression, modulated by subtractive adaptation, predominates in the control of sensitivity in human cone vision. [S0740-3232(97)01209-X]

1. SCOPE

This essay is part tutorial, part argument, and part review, according to my view of what needed to be said on the topics as they arose. The general topic of adaptation and the control of sensitivity has received excellent reviews by MacLeod,[1] by Shapley and Enroth-Cugell,[2] and by Hood and Finkelstein[3]; there is an authoritative review of recent findings by Walraven *et al.*[4]; and a new review of retinal mechanisms by Hood[5] is due to appear soon. Hence another summary of accumulated findings is hardly called for. I intend to focus mainly on integration of the classical findings on adaptation with more recent work on masking and contrast gain control. This required some introductory comments on models and a consideration of the mechanisms for the control of sensitivity, with their anatomical loci. As these topics depend critically on light levels, it was necessary to provide a means of going back and forth between the specifications of light levels used by neurophysiologists and those used by psychophysicists. But before taking up these topics, I first devote a few words to the concept of sensitivity.

2. CONCEPT OF SENSITIVITY

A. Definition

Sensitivity is a basic property of a sensory system, and everyone has an intuitive understanding of the concept. Nevertheless, it is worth reviewing the basic concept and how it is measured. In general, sensitivity refers to $\Delta R/\Delta I$, the ratio of the change at the output of a system (ΔR) to the change at its input (ΔI). In engineering it may be referred to as gain. In psychophysics, sensitivity is chiefly connected with detection and is usually defined as the reciprocal of the threshold for detection. Except in rare cases (such as in the classic paper by Hecht *et al.*[6]) the absolute sensitivity is of less interest than are the changes in sensitivity produced by changes in other variables, such as wavelength (λ).

B. Measurement

Ideally, observations stand on their own, independent of assumptions and theory. Regardless of whether this is ever possible, it is not so for measurements of sensitivity.

To measure sensitivity, either ΔR or ΔI is held constant and the other measured while some third variable, say, λ, is manipulated. Both approaches have their caveats. The problem can easily be seen in that prototypical vision experiment, the measurement of spectral sensitivity.

1. ΔI Constant

The most straightforward approach to a spectral sensitivity, and the approach that typifies many physiological experiments in vision, is to present flashes of varying wavelength but constant quantum density and to measure the magnitude of response, be it an electroretinogram, number of impulses, or whatever (it could even be a magnitude estimate[7]). Figure 1 shows how it might look if tested with stimuli separated by tenfold differences in quantum density. The point here is that the shape of the observed curve depends on the intensity used to measure it. (The fact that tenfold increases of intensity do not necessarily increase the response tenfold is a separate but important issue, discussed in Subsection 6.C.) Although this problem is almost universally recognized and has been for a long time,[10,11] it has nevertheless complicated comparison of physiological results with psychophysical results for decades, and, owing to differences between the objectives of many neurophysiological experiments and those of psychophysical experiments, it continues to be a problem.

2. ΔR Constant

The usual way around the difficulty associated with experiments in which the intensity of the test stimulus is constant is to determine the stimulus necessary to elicit a given response, such as a threshold. This is the approach typical of psychophysical experiments in vision.

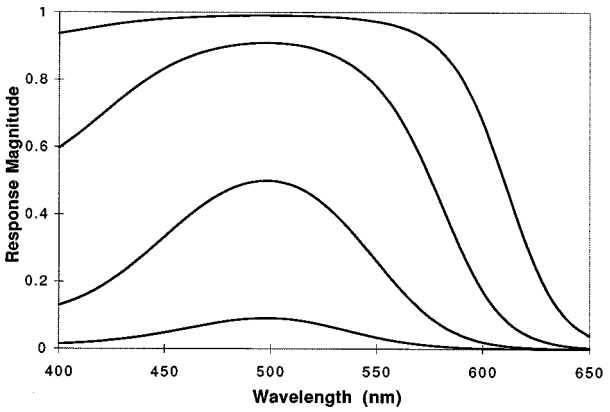

Fig. 1. Hypothetical action spectra obtained with a constant energy stimulus. Successive curves represent stimuli differing tenfold in energy. This assumes that the response follows the Michaelis–Menton equation,[8] which is simplified here to the form $R = 1/(1 + 1/I)$. The spectral sensitivity follows Eq. 6 of Baylor *et al.*[9]

The assumption is that a threshold represents a constant physiological response; that is, if a change of wavelength decreases the response, increasing the size of the stimulus until the original response is restored restores the original conditions leading to that response. In measurements of the spectral sensitivity of rods, for example, this assumption is valid: Changing the wavelength changes the proportion of incident quanta absorbed, and one can replace those not absorbed by increasing the incident number; hence, from the site of absorption and beyond, the state of the system is identical at both wavelengths. Moreover, the shapes of the spectral curves are the same, regardless of the magnitude of the criterion response—up to a limit, of course.

That limit is reached when the stimulus is sufficient to allow the cones to mediate the response. In this case, the assumption that works for rods fails. With the cone system, a change of wavelength may reverse the relative excitation of two classes of cones. Increasing the number of quanta until the same response is elicited from the observer no longer reestablishes the conditions that held before the change of wavelength, for a different class of cones may be mediating the response. One might be unable to reproduce the initial state after a change of wavelength, no matter what the intensity: The best that one can do is to restore conditions so that they are similar, or the same in limited ways.

The only similarity that such an experiment guarantees is that the probability of detection can be equalized. In the simplest case this means that after the change of wavelength the ratio of the signal from one cone system to whatever noise afflicts the detection mechanism is the same as the ratio of the signal from the other cone system to the noise present under those conditions. However, both the signal and the noise might be greater in the one case than in the other. The conditions at threshold might differ in other ways as well; for example, the number of cones contributing to the response may differ, or both cone systems might contribute to the response.

The point here is that sensitivity can be neither measured nor interpreted without a model, a model that often is not explicit. For a related discussion and a list of assumptions underlying psychophysical methods, see Graham.[12]

C. Threshold as a Response Criterion

1. Advantages

In psychophysics the criterion for constancy of response is almost universally the threshold for detection or discrimination. In principle, any constant response criterion could be used, e.g., a brightness approximately 3 times threshold[13] or a magnitude estimate of 10. However, thresholds have an advantage over other criteria, namely, that for a well-motivated observer they are likely to be pressed against physical limits like noise, whereas it is less clear what constrains other response criteria. This may render thresholds less variable than other responses and less susceptible to extraneous influences.

Another advantage of thresholds is that the theory of signal detection[14] provides a successful theoretical framework for interpreting them and for relating them to physiological processes, but, where this theory cannot be used, no comparable alternative exists.

I should like to suggest a third advantage, namely, that responses in a threshold or discrimination experiment (a class A observation according to Brindley's classification[15]) can be causally related to the stimuli without recourse to troublesome psychophysical linking laws,[15–19] laws that are needed to link the stimuli or the responses to inferred or hypothetical subjective states. The need for such laws arises only when one fails to distinguish between responses as physical events and responses as symbols. One is treating the response as a symbol if one has to know the language of the observer to interpret the response. In that case, the referents for the symbols delivered by the observer may also enter into a causal chain, but the events and the associated laws are psychological instead of physiological and hence are harder to relate to physiology (i.e., they require linking hypotheses). This argument is laid out in more detail in Appendix A.

2. Disadvantages

As stated above, thresholds are typically if not always limited by noise, but noise may have little or no influence on other responses, such as those associated with the appearance of stimuli or other suprathreshold properties. Similarly, subtractive adaptation (see below) may affect appearances with little effect on thresholds. Thresholds, then, may tell little about how things look.

Owing to the influence of noise on thresholds, they cannot be used to measure signal strength unless the noise is held constant or measured. This complicates the use of thresholds in observing adaptive changes of signal strength, as discussed below.

As a practical matter, nearly all the papers on visual sensitivity are based on a threshold criterion. Consequently, this essay deals primarily with thresholds.

3. MODELS IN VISION

A. Descriptive Models

It was pointed out above that a model is necessary to organize and to interpret observations. A model also light-

ens the memory load. That is, one goal of science is to summarize the results of as many experiments as possible by a law or theory, usually expressed as an equation. Then one can remember the law and forget the experiments. The ideal gas laws are an example. Such models are often called descriptive, sometimes only descriptive.

The modifier, "only," implicitly recognizes the deeper scientific goal of reducing laws to their underlying processes, as the gas laws can be reduced to molecular kinetics. However, such reduction, historically if not logically, occurs only after discovery of the descriptive laws. Reference to any handbook on vision shows that much of visual science is pretheoretic even in the descriptive sense.

So derivation of a descriptive model constitutes a scientific advance, even if it is not couched in terms of the underlying physiology. The ideal gas laws are useful and even now are considered a part of the science of physics. Explanation in terms of molecular kinetics is satisfying and gives one a better understanding, but for many purposes the descriptive formulation is entirely satisfactory. In vision we are striving for the analog to molecular kinetics, but, until we have attained that level of understanding, we settle for the gas laws. The tendency to dismiss such theories as merely descriptive overlooks what value they do have, especially in the absence of a theory of molecular kinetics.

B. Economy Versus Completeness

It is generally accepted that visual information passes through channels selective for different properties of the stimulus, such as spatial frequency. It is therefore natural to model visual mechanisms in terms of such channels. However, such channels have many properties requiring specification, including their bandwidth in spatiotemporal frequency and orientation; extent in three directions; density in space, orientation, and spatiotemporal frequency; phase sensitivity; rules of combination; variations with eccentricity; color coding; adaptability; and, finally, hierarchical organization (a similar set of parameters may have to be defined at each level of the hierarchy). Insofar as each of these parameters may vary among individuals, they represent a superabundance of free parameters. These problems are not necessarily insurmountable, as Wilson and Gelb[20] showed, for example, and one can minimize the number of free parameters by ignoring individual differences, describing instead the performance of a typical or "standard" observer, as Wilson and Gelb did. However, the laws of color matching were well established long before the properties of the color channels were known, and it may be possible to know some of the laws of spatial vision, for example, without knowing all the properties of the underlying channels. Giving up some of what we do know about the neural basis of vision may pay off by allowing us to finesse some of what we do not know about it.[21–23] Theories based on channels have intuitive appeal, and taking into account the properties of channels is sometimes necessary, but channels may not always be the best bases for a model.

4. TWO DOMAINS AND TWO APPROACHES

Three interrelated things that happened approximately simultaneously at the end of the 1960's and beginning of the 1970's changed practice and thought in many areas of vision, including those related to the control of sensitivity: Cathode-ray tube displays that allowed presentation and facile manipulation of sinusoidally modulated gratings came into widespread use; use of the Fourier domain to describe and manipulate stimuli became widespread; and the idea became accepted that visual information passes through the parallel channels, mentioned above, that are selectively sensitive to different regions of stimulus continua such as size or spatial frequency. Increasingly, (1) gratings tended to replace test and background disks; (2) gradual gating or modulation tended to replace flashes; (3) description of variables and modeling in the Fourier domain tended to replace the spatiotemporal domain; (4) the cortex tended to replace the retina as the locus of hypothesized sensitivity changes; and (5) explanation of sensitivity changes by masking and contrast gain control tended to replace adaptation.

This transition was neither complete nor universal, but it presented the visual scientist with three questions:

(1) To what extent are the newly hypothesized mechanisms simply different names for the same thing, e.g., is gain control or masking just a different name for adaptation? Many investigators prefer highly restrictive use of the word adaptation, but there is little agreement on which restrictions to use, and the reasons for the restrictions seem unconvincing; so I use the term here simply to apply to any process or visual property that brings about a change of sensitivity accompanying a change of luminance. This encompasses everything from photopigment bleaching to contrast gain control in cortical cells, including response compression, all discussed below. Then a better way to state the question here is whether the newly described phenomena can be explained on the basis of classical concepts, or whether the classical phenomena can be explained on the basis of the new concepts. Graham and Hood[24] have, in the temporal and the temporal-frequency domains at least, convincingly answered "no" in both cases. Much work remains, then, to integrate the phenomena and the theoretical concepts of the spatiotemporal and the Fourier domains.

(2) To what extent do the newly described phenomena affect observations of the classical phenomena, e.g., how does contrast gain control affect thresholds for test spots flashed on disk backgrounds? This also remains an open question that calls for more work.

(3) Introduction of the frequency domain (a phrase that I use interchangeably with "the Fourier domain") raises the question, In which domain should stimuli be described and theories be couched, the spatiotemporal domain or the Fourier domain? Although the experiments of Campbell and Robson[25] and of Enroth-Cugell and Robson[26] that introduced the concept of multiple spatial channels and those that were most convincing at the time (Pantle and Sekuler,[27] Blakemore and Campbell,[28] Graham and Nachmias,[29] and Sachs *et al.*[30]) all used grating stimuli and expressed them in Fourier terms, the first general and quantitative theory founded on multiple spatial channels, introduced by Thomas[31] as early as 1970, worked primarily in the spatial domain. One may argue that, as these two domains are linear transformations of

one another, they are simply alternative ways of representing stimuli and the systems that process them, and the choice is meaningless, devolving to personal preference; and indeed many later models (e.g., those by Thomas,[31] Thomas and Olzack,[32] Wilson and Bergen,[33] Wilson and Gelb,[20] and Watson and Ahumada[34]) explicitly can be expressed in either domain, although the Fourier domain tends to be used in practice. A recent trend toward models expressed solely in the Fourier domain[22,35–38] raises the question of whether it offers special advantages.

The sorting of spatial information into separate channels according to its spatial frequency is done in the cortex,[12,39] where the representation of the phase spectrum of the stimulus is, to some extent, separated from that of its amplitude spectrum. That is, many cortical cells, prototypically complex cells,[40] are indifferent to the spatial phase of grating stimuli, and, insofar as the sensitivity of the observer depends on such cells, the phase spectrum of the stimuli is irrelevant. Modeling such phase independence is awkward in the spatial domain but natural in the Fourier domain. It is hard to think of a comparable asymmetry favoring the spatial domain, and other nonlinearities acting on the signals after they have been sorted in the Fourier domain may favor theorizing in that domain; some are demonstrated below. One of the purposes of this essay is to point to some advantages of basing theory in the Fourier domain.

A classic phenomenon that touches on all three questions above is spatial sensitization,[41,42] otherwise known as the Westheimer effect (technically, sensitization is only part of the curve denoted as the Westheimer effect): It is measured by spots flashed on background disks, and it has typically been explained on the basis of the spatial properties of retinal mechanisms, without consideration of the phenomena associated with gratings, which were unknown when it was discovered. I next show that a theory couched exclusively in the Fourier domain, based on the phenomena measured by gratings and devoid of any obvious spatial counterpart, describes the Westheimer effect quantitatively without free parameters except for a vertical shift of the curve.

A. Westheimer Effect

1. Data

The phenomenon of spatial sensitization, first reported by Crawford,[43] has been studied most extensively under scotopic conditions,[41] but sensitization in cone vision[42] is most relevant here. In Westheimer's classic experiment,[42] threshold luminances were determined for a disk nominally subtending 1 arcmin and flashed at the center of a steady background disk of varying size and luminance. The curve in Fig. 2 (from Fig. 2 of Westheimer[42]) epitomizes the phenomenon: As the size of the background increases beyond that of the test flash, threshold luminance of the test flash increases to a peak, after which further increases in the size of the background actually decrease the amount of light necessary to detect the test flash. Hence, adding light at a distance from the test flash makes the test flash easier to see, i.e., sensitizes the observer to the test flash. This phenomenon is typical of the classical approach, insofar as the variables are spatial, consisting of a disk flashed briefly on a larger, steady disk; and the mechanism by which light at a distance increases sensitivity was thought to be retinal (discussed below).

It is argued above that the locus of the processes that account for a phenomenon is important because it bears on the appropriate domain of theory. Hence I next take up the question of the locus of this phenomenon.

2. Site of Underlying Processes

a. Psychophysics. Westheimer attributed sensitization to retinal processes because adding light to the nontest retina raised thresholds instead of lowering them as it did when the light was presented to the corresponding region of the test eye, though he did raise the possibility of a cortical contribution.[42] However, Markoff and Sturr[44] failed to replicate Westheimer's dichoptic result, and both Markoff and Sturr[44] and Sturr and Teller[45] reported robust dichoptic effects when the backgrounds were flashed. A wealth of evidence[46] has now accumulated that supports the Fox–Check discovery[47] that stimuli presented to one eye tend to raise thresholds in the other eye by activating the mechanisms associated with binocular rivalry. Even contralateral stimuli that are not normally considered rivalrous can raise thresholds.[48] These threshold-raising effects could well have exceeded any sensitizing effects and concealed their presence, but the flashed stimuli of Markoff and Sturr and of Sturr and Teller may have broken through the suppression of rivalry. Recently, Yu and Levi[49] have demonstrated that binocular rivalry interferes with such dichoptic tests of sensitization with steadily presented backgrounds and that the essential features of the curve shown in Fig. 2 can be obtained when the background is presented to one eye and the test flash to the other, as long as care is taken to avoid binocular rivalry. Further evidence of a cortical locus lies in the finding that the Westheimer effect can disappear if the test flash is presented only during Troxler fading[50] and in the findings of Lennie and MacLeod[51] (see also Blick and MacLeod[52] and Latch and Lennie[53]) that

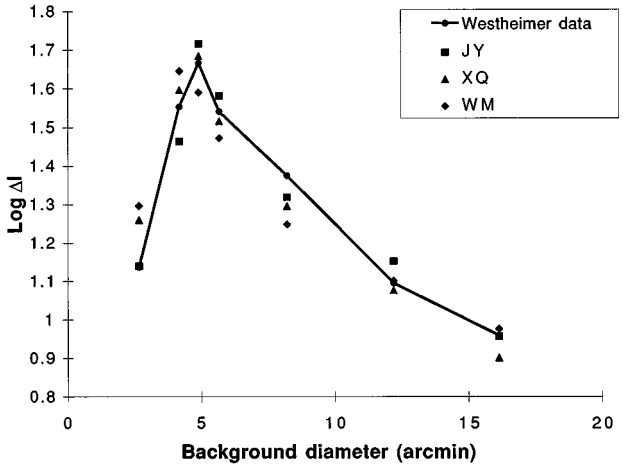

Fig. 2. Westheimer effect[42]: threshold for a small, centered spot flashed on steady backgrounds of varying diameter.

the operations known to elevate threshold in sensitizationlike paradigms all produce contours in the vicinity of the test flash.

Evidence against a cortical explanation was advanced by Hayhoe and Smith,[54] but that conclusion depends "on the fact that there is substantial reduction of the steady state signal by the level of the ganglion cells" (p. 466). This need not be true of P cells. Hayhoe[55] also presented evidence against explanations based on multiplicative gain changes after the nonlinearity (by imputation, cortical). However, since then, Wilson and Humanski[56] have shown that this is the wrong model for cortical contrast gain changes; moreover, the mechanism can be cortical without entailing a gain change, as described below.

The psychophysical evidence, then, favors a cortical locus.

b. Neurophysiology. The explanation of the Westheimer effect in terms of neural receptive fields has been laid out with varying degrees of completeness by many authors, including Westheimer,[41,42] Thomas,[31] and Teller.[16] The theory is that the information in a threshold test flash is carried by a cell that has its receptive field centered on the test spot, so that at that level of the hierarchy it is the cell most sensitive to the test flash. Light in the sensitizing annulus of the background activates the spatial antagonism of the receptive field surround, driving the firing rate of the cell in the direction opposite that produced by light in the center of the receptive field. Finally, this increases the ratio of signal to noise, so that the test spot is easier to detect.

Not all authors—specifically not Westheimer[42] himself—identified the neural elements responsible, but retinal ganglion cells have all the necessary properties and provide a ready substrate. In fact, a phenomenon analogous to the psychophysical Westheimer effect has been reported in cones of turtles[57] and in horizontal cells, bipolar cells, amacrine cells,[58] and ganglion cells[58–60] of the mudpuppy retina. Enroth-Cugell *et al.*[61] found that, in cat ganglion cells, sensitization required a retinal illumination of $1.5(10^6)$ quanta(q) $\mathrm{deg}^{-2}\,\mathrm{s}^{-1}$ or approximately 2 cat trolands (Td). Others[62,63] have also observed, in cat lateral geniculate nucleus cells, sensitization when the backgrounds exceeded the value found by Enroth-Cugell *et al.* and failed to observe it below that value.[64,65] Cleland and Freeman[66] failed to observe it at even higher backgrounds, $4.6(10^8)$q $\mathrm{deg}^{-2}\,\mathrm{s}^{-1}$, but these may have been too high. However, the lowest backgrounds at which sensitization has been observed in cat is some 400 times the corresponding value in humans.[41] More to the point, perhaps, is that no one has reported observing sensitization in subcortical primate neurons at any intensity; this is not to say that they have tried and failed; it simply means that the evidence allows any interpretation.

c. Anatomy. As Thomas[31] pointed out, the width of the background at which threshold is highest is approximately the same as the width of the center mechanism of the receptive field of the cell that detects the test flash or at least places an upper limit on it. Westheimer's data require a receptive field with a center mechanism 5 arcmin or 24 μm in diameter. This is half the diameter of the parasol ganglion cells of the human fovea,[67] more than twice that of the corresponding midget cells, and more than five times the width (at half height) of the summation area of the human retina, isolated interferometrically.[68–71] Although optical spread and eye movements increase the effective receptive field, their combined effects are insufficient to reconcile the anatomy with the psychophysics. The receptive field of a ganglion cell need not correspond to its dendritic spread, but, lacking data on receptive field sizes, one must conclude that insofar as the anatomy itself goes, it does not mesh well with the psychophysics.

d. Conclusion. It seems fair to say that the evidence of a retinal locus of spatial sensitization in humans is weak at best.

B. Modeling in the Fourier Domain

Explanation of spatial sensitization in terms of center–surround antagonism has been heuristically satisfying and serves well as long as the explanations are only qualitative, but it has so far spawned no quantitative theories, and the quantity that can be gleaned from the data, the size of the receptive field, does not support a retinal explanation. Moreover, there is now substantial psychophysical evidence for a cortical as opposed to a retinal locus. One must therefore take into account the effects of cortical mechanisms, with their size- or frequency-selective effects. As no route from spatial theories to a quantitative explanation of the Westheimer effect is obvious, I turn to the Fourier domain. Crossing between spatial and Fourier domains in either direction is hindered by violations of the assumptions of linearity and homogeneity, violations that are inherent in the functional architecture of the visual system. Ignorance about the properties of channels and how they interact also hinder the application of Fourier models to objects that are spatially simple but have complicated Fourier spectra, such as disks and annuli. However, these difficulties need not prevent the effort.

1. Results

To provide the reader with a reason to pay attention to the theory, I show the results before the theory. The triangles, squares, and diamonds shown in Fig. 2 are, aside from a vertical shift of each curve, parameter-free estimates of the thresholds computed from the model of Yang and colleagues[22,23]: No information from Westheimer's data (aside from a vertical shift of each curve) was used to derive these estimates, but the parameters used to make them are those estimated by Yang *et al.*[22] for their three observers. There is no statistically reliable difference between Westheimer's data and the estimated thresholds for these three observers ($F = 1.39$, $df = 6, 14$).

2. Method

To gain an intuitive understanding of the way in which a Fourier model produces the phenomenon of spatial sensitization, one begins with the Fourier spectrum of a disk:

$$A = \frac{J_1(2\pi f)}{\pi f} = \frac{D J_1(4\pi/D)}{2\pi}, \tag{1}$$

shown in Fig. 3 on semilogarithmic axes (the periodic function). D is the diameter of the disk in degrees, where

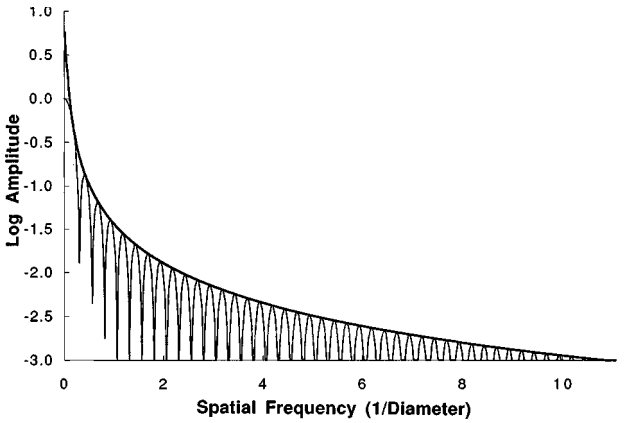

Fig. 3. Fourier spectrum of a disk.

f is spatial frequency in cycles per diameter. Note that, if -2.5 is taken as log contrast threshold, a disk has harmonics that could be detected at frequencies five times the reciprocal of the diameter of a disk, i.e., a 1-deg disk has detectable harmonics at 5 cycles per degree (c/deg). Appendix B explains the envelope of the curve shown in Fig. 3 and shows that the spectra of disks of varying size do not beat.

To estimate the threshold of a small disk on a larger one on the basis of their spatial frequencies, one needs their amplitude spectra. (Transforms of frequently encountered functions are available in convenient form in Bracewell's book on Fourier transforms,[72] which also contains references to other, larger sets of transforms.) Figure 4 shows the spectrum of the test flash (the nearly horizontal, heavy line intersecting the y axis at an amplitude of 10) and those of the five smallest backgrounds that Westheimer used. By assuming that the visual system is sensitive only to frequencies below 15 c/deg, one can get an intuitive idea of how the data of Fig. 2 might have arisen. As the size of the background increases through the first three smallest backgrounds, the level of the background increases at all the frequencies to which the system is sensitive. Assuming that a background component at a given frequency raises thresholds at that frequency, the amplitude (luminance) of the test stimulus must be likewise increased to be detected. However, the spectra of the two largest backgrounds each dip to zero within the sensitive range below 15 c/deg. As the test flash has nearly equal amplitude at all the frequencies, those components of the test stimulus at frequencies in which the background is deficient would be detected even when the amplitude of the test stimulus is low. Hence test flashes of lower amplitude, with lower spectra, can nevertheless be detected, and the phenomenon of sensitization results.

Transformation of these qualitative ideas into quantitative statements requires both a model that relates the amplitude of a background at a given frequency to the threshold at that frequency and a rule governing the contribution of sensitivity at each frequency to the observer's response. For the model, I adopted that of Yang and Makous[23] (see Table 1), a less general version of that by Yang *et al.*[22] This theory finesses ignorance about the properties of channels in a way that is analogous to use of primaries in lieu of fundamentals in color vision. It is based on thresholds for gratings presented on pedestal gratings of varying luminance, contrast, and spatial frequency.

To apply the model to the Westheimer paradigm, one considers each frequency in the spectrum of the test spot as a test grating and each frequency in the spectrum of the background as a masking grating, assuming that the visual system can respond to each separate test frequency, independent of all the other frequencies in the spectrum of the test stimulus. So the combination rule used assumes that threshold is determined solely by the test frequency at which threshold is lowest. (Probability summation among channels and other combination rules are folded into the expression for threshold.) Specifically, I found the minimum of the threshold ampli-

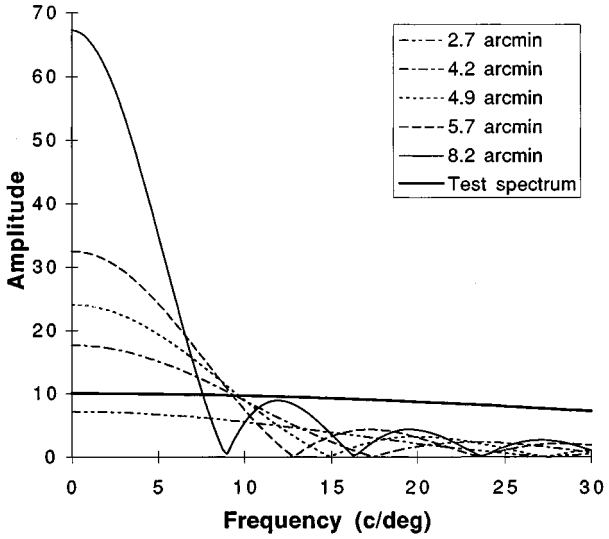

Fig. 4. Fourier spectra of the test stimulus and the five smallest backgrounds in Westheimer's experiment.

Table 1. Model by Yang and Makous[a,b]

Equation	Theoretic Component of Threshold Versus Amplitude Curve
$A_t = \exp(\alpha f)[$	Modulation transfer function (reciprocal)
ϕ	Absolute threshold
$+\beta L^{1/2}$	Noise
$+L\eta_0/(f^2 + \sigma_0)$	Implicit masking
$+\rho A_p \exp(-\alpha f)\eta f^{1/4}$	Effective pedestal
$]^{\gamma}$	Asymptotic slope
$-(1 - \rho)A_p$	Subthreshold summation

[a] Ref. 23.

[b] A_t is the amplitude of the threshold test grating; L is the mean luminance; f is the spatial frequency of test and pedestal (masking) gratings; A_p is the amplitude of the pedestal grating; $\rho = A_p[A^{*2}/(A_p{}^2 + A^{*2})]$, where A^* is A_t with no pedestal; and α, ϕ, β, η_0, σ_0, η, and γ are free parameters. Note: On reading a draft of this paper, Jian Yang pointed out that the exponent, 1/4, was from an earlier version of the model than the one that we published, where we chose an exponent of 1/2 instead, mostly on the basis of aesthetics (1/2 produced only a marginally better fit). However, the exponent, 1/2, introduces a systematic error here, yielding thresholds that are too low when the disk is small and too high when the disk is large. The value of 1/4 might have been a better choice after all, and I have used it here.

tudes, A_t, as spatial frequency (f) varied, using the equation in Table 1; the parameters obtained for each observer by Yang *et al.*; the luminance, L, from Westheimer's experiment; and masking amplitudes, A_p, at each frequency, from the Fourier spectrum of the disk in Westheimer's experiment. The threshold luminance of the test flash follows directly from the minimum threshold amplitude, A_t; the frequency, f, at which it is minimum; and the spectrum of the test flash. The results are the data points shown in Fig. 2 (the squares, triangles, and diamonds) after a vertical shift of the curve is made for each observer.

Note that this analysis differs from the spatial explanation in two ways. First, the explanation depends entirely on the location of the minimum in the Fourier transform of the background disk, a property of the Fourier approach that has no obvious counterpart in the spatial domain. Second, it has nothing to do with the spatial antagonism associated with the bandpass shape of the contrast sensitivity function. Eliminating this spatial antagonism (by setting $\eta_0 = 0$) improved the fit by 3% for one observer and worsened it by 1% and 2% for the other two, respectively.

This is not to say that the fit is generally insensitive to the parameters or the properties of the model. Aside from the coefficient for the magnitude of spatial antagonism, η_0 (and the related constant, σ_0, which is irrelevant when $\eta_0 = 0$), only the exponent (γ) was expendable: Setting it equal to unity actually improved the fit. Changing any other parameter disproportionately worsened the fit. (However, as the parameters are not orthogonal, the effects of changing one could be compensated by changes in another.) The point here is that the parameters that best describe an observer's performance in a grating experiment also describe, with no statistically reliable deviation, the observer's performance in an experiment on spatial sensitization. Adjusting all the parameters to fit Westheimer's data best does not produce a statistically reliable improvement.

It is worth noting that, while examination of the spectra of the backgrounds provides some insight, one cannot easily intuit the interaction between the spectra and the model. For example, lowering mean luminance has no effect on the shape of the Fourier spectra, but it does change the location of the peak of the sensitization curve, as shown in Subsection 4.B.*3*. Evaluating the applicability of this approach to other data requires the use of the model, an enterprise that is beyond the scope of this paper.

However, the validity of the model can be tested directly, for it dictates that masking at the minimum of the spectrum of the disk should be more effective than masking at other frequencies, and the effectiveness of masking should be independent of the phase of the masking grating. Note that models expressed in the spatial domain would hardly lead to this prediction. Preliminary tests made by Peter Bex and myself suggest that both predictions may be true.

3. Field Adaptation

Westheimer measured sensitization at five different luminances ranging from 0.0025 to 8 cd m^{-2}. Figure 5 shows how the theory describes the data at each of these luminances when the parameters of one of the observers of Yang *et al.*[22] (the observer whose data best match those of Westheimer, WM) are used. Again, the only parameter that has been used to fit the data is a vertical shift. Although the theory is intended to describe changes of sensitivity caused by changes of mean luminance without any parametric adjustment, each of these curves was in this case adjusted vertically; however, no adjustment exceeded 7% of the total range of log luminances in the figure.

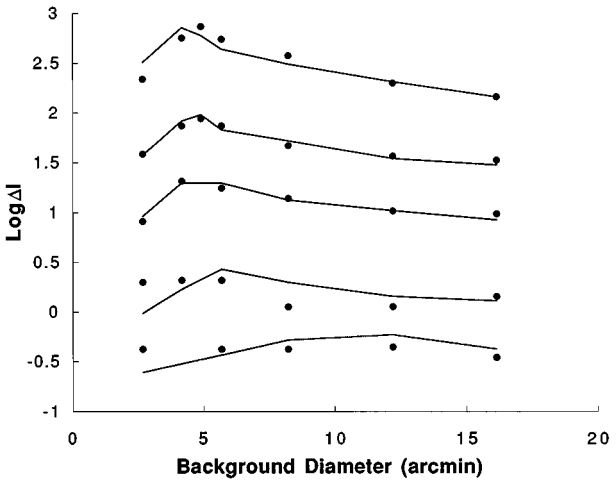

Fig. 5. Westheimer effect at five background luminances.

Aside from changing the heights of the curves, changing mean luminance also changes the shapes of the curves. The changes in the visual system that produce the changes in the shape of the curves shown in Fig. 5 are reflected in the model by changes in shape of the contrast sensitivity curve and in the function relating contrast discrimination to pedestal amplitude. The dependence of shape on mean luminance described by the model required no adjustments, although the fit is better at the high and the intermediate luminances than at the low luminances.

4. Concluding Comments

These observations and theory apply to photopic vision. The Westheimer effect observed in rods[41] may well depend on different mechanisms.

As I have tried no other models, there is no reason to believe that any other model of contrast sensitivity would fail to describe Westheimer's data as well as the one used here. The intention is not to compare models but to show that a model derived and expressed in the Fourier domain successfully applies to a phenomenon that otherwise seems more appropriately expressed and treated in the spatial domain and that such application can yield insight into underlying mechanisms. This encourages other efforts to cover the phenomena of both domains under a single theory operating in the Fourier domain, such as those treating vernier acuity.[73–75]

C. Temporal Domain

So far only the spatial domain and its Fourier counterpart have been considered. The question arises as to whether

analogous arguments can be made for the temporal-frequency domain. It was argued above that the special benefits of operating in the Fourier domain follow from the separation of information in different frequency bands within the visual system. How well the Fourier approach works in the temporal domain may depend on the degree to which the visual system performs an analogous Fourier analysis in the temporal domain.

1. Temporal Channels

The present evidence suggests fewer, less selective frequency channels in the temporal domain than in the spatial domain, but certainly more than one.[76] There is considerable evidence of at least three channels for spatial frequencies near 1 c/deg and below. Specifically, 1, 4, and 12 Hz can be discriminated from one another at threshold.[77] Also, masking at 1 Hz can raise thresholds at high frequencies with no effect on those of intermediate frequencies[78]: This requires at least three channels, one of which is broadly tuned. Finally, derivation of nearly identical channels by different investigators[77,79] by altogether different approaches (Fig. 6) strengthens one's confidence in such channels. Hence the evidence at present is that there are at least three temporal channels, although not all three may participate at all spatial frequencies or eccentricities.[80] Whether this is enough to give an advantage to theorizing in the Fourier domain is not clear, but it may well pose problems for single-channel theories.

2. Model by Graham and Hood

The notable effort to combine results observed in the temporal domain with those in the temporal frequency domain under the same theoretical rubric is that of Graham, Hood, and co-workers.[24,81,82] They showed that, while models from one domain could not predict fundamental phenomena predicted by models from the other domain,[24] models merged from both domains could predict some of the basic phenomena associated with both domains.[24,82] However, these merged models failed when tested with a paradigm in which sensitivity was measured with an aperiodic stimulus superimposed on a periodic background[81] (see also Wu *et al.*[83] in this issue). There are two aspects of the new findings that pose problems for the model: an abrupt shift of the phase of sensitivity variations relative to the background modulation as the frequency of the background modulation varied over the range from 4 to 8 Hz; and a large, sustained decrease of mean sensitivity that is independent of the phase of the background modulation.

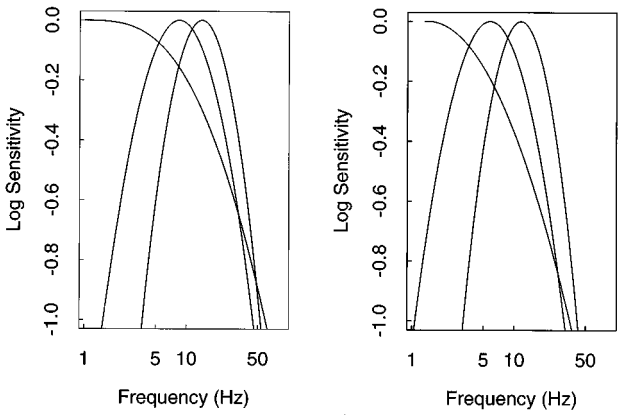

Fig. 6. Sensitivity profiles of the temporal channels inferred by Mandler and Makous[77] (left) and by Hess and Snowden[79] (right).

The model by Hood *et al.*[81] is an example of the kind of model promoted above in Section 3, namely, one that eschews the complications posed by incorporating the underlying channels as components of the theory. Those researchers avoided the complications of channel theory simply by assuming a single channel, but in this case it may have posed a problem. In a direct temporal analogy to Westheimer's spatially compact stimulus, they tested temporal sensitivity with a temporally compact test flash (10 ms) that splatters energy well over the range of visible frequencies, dropping to approximately 50% amplitude at 60 Hz. Such a stimulus is fine for a single-channel system but is not ideal for separating channels. Any putative temporal channels would be approximately equally sensitive to it, but the temporally modulated background constitutes a desensitizing or masking stimulus that acts selectively on the channels sensitive to it. The phase discontinuity observed could be due to a shift between channels that detect the test flash as a shift of the frequency of the masking stimulus changes the relative sensitivity of adjacent channels, for the discontinuity occurs at just the masking frequency where the sensitivities of the two lower-frequency channels cross. Channels tuned to different frequencies are bound to have different phase delays. So a likely interpretation of the problem encountered by Hood *et al.* is that a change in background frequency causes a shift between channels determining performance, with a corresponding shift of phase delay. Such abrupt shifts, when observed in sensory phenomena, always involve a shift between mechanisms, with the rod–cone break being the prototypical example.

3. Universal Dependence of Temporal Sensitivity on Spatial Properties

That the effects of spatial and temporal modulation are not independent is well known,[84] but contrast sensitivity can be decomposed into center and surround mechanisms within which, individually, the effects of spatial and temporal modulation are independent.[22,85,86] Any stimulus that modulates both mechanisms inevitably produces nonlinear interactions between these mechanisms. Interferometric methods of producing high spatial frequencies show that the surround mechanism is sensitive to spatial frequencies as high as 30 c/deg.[69,71] When only the center mechanism is modulated, by use of gratings of 35 c/deg,[69] the temporal contrast sensitivity becomes a shallow, low-pass curve with an asymptotic slope of -1 on log-log axes. (That this effect is not due to any peculiarity of the interferometric technique is shown by the fact that it replicates Robson's temporal contrast sensitivity curve[87] when tested at the spatial frequency of 22 c/deg that he used.) Modulation of the surround mechanism steepens the drop of sensitivity at both ends of the temporal contrast sensitivity curve, ultimately reaching a slope of -13 at the higher end of the curve when tested with large fields of 65 deg or so.[84]

Thus the effects of interactions between center and surround mechanisms extend from the lowest spatial frequencies to the highest that can pass through the optics of the eye. Owing to the universal effect of spatial interactions on temporal sensitivity, models that do not take them into account, such as those based solely on photoreceptor kinetics, cannot apply to psychophysical data obtained with the normal optics of the eye.

5. INTENSITY LEVELS

Any comparison of psychophysical performance with neurophysiological mechanisms must reconcile the different ways in which psychophysicists and neurophysiologists specify light. This section is devoted to such a reconciliation, and details are given so that the reader can know the basis of the estimates. Those not concerned with these details can safely bypass this section and use the tables as the need arises.

Calibrations of psychophysical stimuli determine the properties of the stimulus before it enters the eye, whereas neurophysiologists often illuminate the retina or the photoreceptors directly, without intervening ocular media. So they often specify their stimuli in terms of quantum flux per unit area on the retina, occasionally in terms of quantum flux per unit solid angle of visual field, or, if working with receptors, number of quanta per receptor absorbed or activating pigment molecules. The relationships among these are not yet clear, especially for cones. Moreover, the relationship varies with wavelength, retinal eccentricity, age, race, and individual. As a general expression covering all these variables is unwieldy, I present instead an estimate for a particular class of human observer, analogous to a standard observer, under a standard set of conditions.

The usefulness of such estimates is limited without an accompanying estimate of probable error. Consequently, the values given here include the estimated values one standard deviation above and below the mean estimate.

A. Ocular Media

Without losses in the ocular media, 1 scotopic (scot) Td at 510 nm would illuminate the retina with a flux density of 5.44 q $\mu m^{-2}\,s^{-1}$, and 1 photopic (phot) Td would illuminate the retina with a flux density of 14.65 q $\mu m^{-2}\,s^{-1}$. [This is from Wyszecki and Stiles,[88] Eq. 2.11.8. Note that in the second edition of Wyszecki and Stiles,[89] Eqs. 19(2.4.4) and 20(2.4.4) omit a factor equal to λ in the numerator. One can verify this omission in Eq. 20(2.4.4.), for example, by deriving it from Eqs. 6(2.4.4), 9(2.4.4), and 13(2.4.4) in the same edition or by comparison with the corresponding equation, 2.11.8, in the first edition,[88] which is correct. Note also that, in Eqs. 6(2.4.4) and 8(2.4.4) of the second edition, T_λ should be T'_λ and that the units for λ are centimeters in the first edition and meters in the second.] The best estimate of the losses in the ocular media are given in Table 2, with the estimated effects on retinal illuminance and receptor excitation.

The estimates of the losses within the ocular media follow Kraats *et al.*[90] These authors estimate that slightly under 30% of the light is lost in reflections and scatter within the media at all visible wavelengths, leaving 71% transmitted. Transmission through the lens at 555 and 507 nm are for ten observers spanning a wide range of ages, with a mean age of 32 years. (The macular pigment does not absorb significantly at 555 nm, the wavelength assumed here for cones, or beyond the macula, the retinal locus assumed here for rod vision.) The total proportion of light transmitted through the ocular media is approximately 0.64 at the peak wavelength for cones and approximately 0.60 at the peak wavelength for rods. As the standard errors are computed in absolute units but are expressed in logarithmic units, the probable error is asymmetrically spaced about the mean.

These losses drop the retinal illuminance produced by 1 Td to approximately 9.4 q $\mu m^{-2}\,s^{-1}$ for cones and 3.2 q $\mu m^{-2}\,s^{-1}$ for rods, where q stands for quanta. The flux density on the retina produced by 1 phot Td is roughly 3 times as great as that produced by 1 scot Td. However, a direct comparison of rods and cones is likely to be done close to the central fovea, where the proportion of 507-nm light transmitted through the macular pigment is 0.65 ± 0.06.[88] This drops the quantum flux per scotopic troland to 2.1 ± 0.3 q $\mu m^{-2}\,s^{-1}$, so that the flux density for cones is 4.5 times that for the rods at the same retinal locus.

Table 2 shows a difference between rods and cones that

Table 2. Quantum Densities on the Human Retina and at the Receptor[a]

	Cones, 555 nm			Rods, 507 nm		
Parameter	(mn − sd)	(mn + sc)	mean	(mn − sd)	(mn + sd)	mean
Total Transmission	0.62	0.67	0.64	0.56	0.64	0.60
Scatter and reflection	0.71	0.71	0.71	0.71	0.71	0.71
Lens	0.87	0.94	0.91	0.79	0.91	0.85
Retina, q/(μm^2 s)	**9.05**	**9.79**	**9.41**	**3.01**	**3.46**	**3.23**
Capture	0.89	2.74	1.76	0.23	0.23	0.23
Aperture, μm^2	1.19	3.04	2.10			
Absorption	0.75	0.90	0.84			
Receptor, q/s	8.06	26.79	16.61	5.54	7.58	6.46
Receptor, q*/s	**5.37**	**17.87**	**11.08**	**3.70**	**5.05**	**4.31**

[a] All the spatial dimensions are in micrometers; transmission and absorption are proportions of incident quanta. Quanta absorbed by a receptor are represented by q, and quanta that activate the absorbing pigment are represented by q^*. The labels mn − sd and mn + sd represent the mean minus 1 standard deviation and the mean plus 1 standard deviation, respectively. Key results are entered in boldface.

is worth noting in passing: The estimated variability of quantal absorptions is much greater for cones than for rods. One standard deviation for the distribution of effects on rod absorption is approximately 16% of the mean estimate, or approximately 0.06 units on a logarithmic scale. The corresponding value for cones is much greater, roughly 60% or 0.2 unit on a log scale. Even when the variability of macular pigment absorption is added to the rod estimates, it increases the standard deviation only to 30% of the mean, or 0.11 logarithmically. This variability among observers in quantum absorptions should be reflected in their performance, and it is: The root mean square of the standard deviation of the absolute rod thresholds of different subjects tested under identical conditions, normalized relative to the mean threshold, is 0.125 on a logarithmic scale (8 studies, 33 subjects), close to the estimated cumulative variability of 0.11 from Table 2. This suggests that the estimated variability of the quantities that affect rod excitation are approximately correct.

By contrast, cone thresholds are much more variable, even more variable than estimated from Table 2. The standard deviation of absolute cone thresholds (divided by the mean) is 0.586 (7 studies, 15 subjects), which is to be compared with the estimated value of 0.2.

Almost the identical difference between rod and cone variability is seen when rod and cone thresholds are measured at the same retinal locus in the same observers (0.125 on a logarithmic scale for rods and 0.648 for cones).[91] Either the variability of one or more of the factors that influence cone excitation has been underestimated here, or some variable not considered affects cone thresholds more in some observers than others. (One can exclude the Stiles–Crawford effect as the cause, for all these studies limited pupillary entry to 2 mm or less, which reduces the standard deviation caused by differences in the Stiles–Crawford effect, inferred from the data of Applegate and Lakshminarayanan,[92] to less than 5%, or less than 0.01 on a log scale.)

B. Quantum Activation Rate

1. Cones

The rate at which quanta activate a receptor, Q^*, is the product of the quantum flux density falling on it, n; the area of the optical aperture, A; the density of the photopigment, D; and the quantum efficiency, η:

$$Q^* = n\,\eta A(1 - 10^{-D}). \quad (2)$$

The quantum efficiency of rods is well established at $\eta = 0.67$, and although that of cones is not known, it is usually assumed to be the same (e.g., Baylor *et al.*[9]). The optical aperture of a foveal cone can be described as a Gaussian window with a width at half-height of 1.36 μm,[68–71] which has an equivalent diameter of 1.60 μm and an equivalent area [A in Eq. (2)] of 2.10 μm^2, or 55% of the 3.8-μm^2 anatomical cross section of a foveal cone.[93] The optical density of a long-wavelength-sensitive cone within the central 1/2 deg of the fovea may be as great as 0.8 (see the figure and the equation on p. 708 of Pokorny and Smith[94]), corresponding to an absorption of 84%. The product of the two yields an absorption of 46% of the light incident within the anatomical boundaries of the cone. This is greater than the 30% observed by Packer and Williams[95] in peripheral cones. However, peripheral cones can have up to ten times the cross-sectional area of a foveal cone, so that the rate of quantal absorptions in peripheral cones may equal or exceed that in foveal cones despite the difference in optical density.[94]

Then, from the equation given above, a reasonable estimate of the rate of quantal absorptions in a cone at 1 phot Td is approximately 17 q s^{-1}, of which some 11 cause activations. The probable range of error is from 5 to 18 activations. Differences from the particular conditions assumed here would of course increase the range, and when a large area of the retina is illuminated, for example, absorptions per cone are likely to differ across the retina. This estimate of 11 q* s^{-1} is close to the 12.6 estimated by Hood and Birch,[96] by a different approach, with a correction for the Stiles–Crawford effect not included here and omission of the receptor aperture used here.

Here a parenthetical note on the Stiles–Crawford effect[97] may be of use. Its effects depend on pupil size and on the breadth and position of the curve expressing the individual's Stiles–Crawford effect. According to the statistics on normal observers reported by Applegate and Lakshminarayanan,[92] light entering the pupil 3 mm from the maximum in an average observer has only 25% of maximum effectiveness (down 0.6 on a log scale), but the integrated effectiveness of all the light entering a 6-mm pupil is roughly 62% of maximum (down only 0.2 on a log scale). For a 3-mm pupil the integrated effectiveness is 86%, down only 0.06 on a log scale. So failure to correct for the Stiles–Crawford effect introduces only modest errors under normal conditions.

Variations in the Stiles–Crawford effect among observers also contributes to interobserver variability, but these are even smaller. If one simply adds the effects of variations in breadth and position (an overestimate), the standard deviation of individual differences in the summed effectiveness of light is only approximately 10% even with a 6-mm pupil, a factor that contributes variability of less than 0.05 unit on a log scale.

2. Rods

The optical aperture of rods is unknown, but it may not be needed, for Packer and Williams[95] have observed that, at an eccentricity of 30 deg, 23% of the quanta incident upon the retina are absorbed by the rods. The mean density of rods at that eccentricity is 105,000 to 125,000 rods mm^{-2}, depending on meridian.[98] Taking the middle of that range, 115,000 rods mm^{-2}, yields exactly 2 q (0.23/0.115) absorbed per rod for every quantum per square micrometer falling on the retina.

Of course, the density of rods varies with eccentricity and among individuals, with the mean peak density being 176,200 rods mm^{-2} at an eccentricity of approximately 15 deg,[98,99] a favorite location to study rod vision; the standard deviation of peak density is 13,200 rods mm^{-2}.[98] Although rod density is higher at 15 deg than at 30 deg, sensitivity also tends to be somewhat higher,[100] so the proportion of incident quanta absorbed per rod may not

be much affected by density, as would be the case if rods were of approximately constant size but of varied packing density.

Then 2 q absorbed per rod for 1 q $\mu m^{-2}\,s^{-1}$ falling on the retina yields 6.5 (5.5–7.6) q s^{-1} absorbed, and 4.3 (5.1–3.7) activations s^{-1} per rod at 1 scot Td when the eccentricity is 30 deg. This can be taken as a general, rough estimate in the parts of the retina in which rod vision is good. Close to the central fovea, where rod and cone vision can easily be compared, 1 scot Td corresponds to 4.2 (3.3–5.4) absorptions/s/rod and to 2.8 (2.2–3.6) activations.

The estimate of 6.5 absorptions and 4.3 activations is close to but slightly above the estimate of 5 absorptions s^{-1} per rod by Westheimer[41] and by Rushton and Henry[101] and of 4 absorptions by Barlow,[102] and it is close to but slightly below Pugh's estimate[103] of 1.47(10^6) absorptions/mm^2, which corresponds to 8.35 absorptions and 5.6 activations, assuming a mean density of 176,200 rods/mm^2.[98,99] However, it is half the 8.5 and 8.6 activations estimated by Kraft *et al.*[104] and by Breton *et al.*[105] respectively, owing principally to a factor of 2 and 1.8, respectively, allowed by them for a funneling of light by rods that has been inferred from deformation phosphenes[106] but which has not been quantified. The rod absorptions measured by Packer and Williams,[95] used here, would have included any such funneling.

C. Check on the Estimates

A rough check on these estimates can be obtained by reference to bleaching experiments. In an equilibrium bleach, the rate of regeneration equals the rate of bleaching; so, on the assumption that the rate of regeneration in the light is equal to the maximum rate of regeneration in the dark,[107] the maximum rate of regeneration tells how many molecules per second are bleached by a given light during an equilibrium bleach and hence the rate of quantum activations at that light level. The time constant of regeneration of cone pigments is 111 ± 8 s;[101,108–110] that for rhodopsin, 394 ± 7 s [Alpern's Table 1 (Ref. 107)]. Thus the maximum rate of pigment regeneration is 394/111 = 3.6 times faster in cones than in rods.

A light of 4.39 ± 0.14 log phot Td keeps the cone pigment at half-concentration,[101,108–110] and the corresponding value for rods is 4.40 log scot Td.[111] So it takes approximately equal numbers of scotopic and photopic trolands to keep the corresponding pigments at half-concentration. If each photopic troland bleaches 5 to 18 molecules/s with a full complement of pigment, then when half are bleached, half of 5 to $18 \times 10^{4.37}$, or 60,000 to 210,000 molecules/s, are being bleached in each cone; correspondingly, each scotopic troland bleaches half of $4.3 \times 10^{4.4}$, or 54,000 molecules of rhodospin/s (the variability of the rod estimate is so small relative to the cone estimate that it is ignored). Hence bleaching this is 1.1 to 3.9 times faster in cones than rods.

The ratios of estimated quantum activation rates by photopic and scotopic trolands spans an admittedly large range, but it does include the ratio of the corresponding bleaching rates, so the relative estimated rates of cone and rod activations are consistent with the regeneration

Table 3. Photopic Conversion Table[a]

Quantity	Troland	cd m^{-2}, 2-mm pupil	cd m^{-2}, d-mm pupil	q $str^{-1}\,s^{-1}$ on retina	q $deg^{-2}\,s^{-1}$ on retina	q $mm^{-2}\,s^{-1}$ on retina
1 Troland=	1	3.142	$0.785\,d^2$	2.62×10^9	7.98×10^5	9.41×10^6
1 cd m^{-2}, 2-mm pupil=	0.318	1	$0.249\,d^2$	8.34×10^8	2.54×10^5	3.00×10^6
1 cd m^{-2}, d-mm pupil=	$1.273\,d^2$	$3.99722\,d^2$	1	$3.33 \times 10^9\,d^2$	$1.02 \times 10^6\,d^2$	$1.20 \times 10^7\,d^2$
1 q $str^{-1}\,s^{-1}$=	3.82×10^{-10}	1.20×10^{-9}	$3.00 \times 10^{-10}\,d^2$	1	3.05×10^{-4}	3.59×10^{-3}
1 q $deg^{-2}\,s^{-1}$=	1.25×10^{-6}	3.94×10^{-6}	$9.84 \times 10^{-7}\,d^2$	3.28×10^3	1	1.18×10^1
1 q $mm^{-2}\,s^{-1}$=	1.06×10^{-7}	3.34×10^{-7}	$8.34 \times 10^{-8}\,d^2$	2.78×10^2	8.48×10^{-2}	1
1 q $\mu m^{-2}\,s^{-1}$=	0.106	0.334	$0.083\,d^2$	2.78×10^8	8.48×10^4	1.00×10^6
1 q s^{-1}/cone, absorbed=	0.060	0.189	$0.053\,d^2$	1.58×10^8	4.80×10^4	5.67×10^5
1 q s^{-1}/cone, active=	0.090	0.284	$0.080\,d^2$	2.36×10^8	7.20×10^4	8.49×10^5
1 Macaque Td=	1.700	5.341	$1.335\,d^2$	4.45×10^9	1.36×10^6	1.60×10^7
1 Cat Td=	1.667	5.236	$1.308\,d^2$	4.36×10^9	1.33×10^6	1.57×10^7

	q $mm^{-2}\,s^{-1}$ on retina	q s^{-1}/cone, absorbed	q s^{-1}/cone, active	Macaque Td	Cat Td
1 Troland=	9.410	16.610	11.080	0.588	0.600
1 cd m^{-2}, 2-mm pupil=	2.995	5.287	3.527	0.187	0.191
1 cd m^{-2}, d-mm pupil=	$11.98\,d^2$	$18.81\,d^2$	$12.55\,d^2$	$0.749\,d^2$	$0.76\,d^2$
1 q $str^{-1}\,s^{-1}$=	3.59×10^{-9}	6.34×10^{-9}	4.23×10^{-9}	2.25×10^{-10}	2.29×10^{-10}
1 q $deg^{-2}\,s^{-1}$=	1.18×10^{-5}	2.08×10^{-5}	1.39×10^{-5}	7.37×10^{-7}	7.52×10^{-7}
1 q $mm^{-2}\,s^{-1}$ =	1.00×10^{-6}	1.77×10^{-6}	1.18×10^{-6}	6.25×10^{-8}	6.38×10^{-8}
1 q $\mu m^{-2}\,s^{-1}$=	1	1.765	1.177	0.063	0.064
1 q s^{-1}/cone, absorbed=	0.567	1	0.667	0.035	0.036
1 q s^{-1}/cone, active =	0.849	1.499	1	0.053	0.054
1 Macaque Td=	15.997	28.237	18.836	1	1.020
1 Cat Td=	15.683	27.683	18.467	0.980	1

[a] For human vision except where otherwise specified. str, steradians. Assumes that cat and macaque eyes differ from human eyes only in size.

Table 4. Scotopic Conversion Table[a]

Quantity	Troland	cd m^{-2}, 2-mm pupil	cd m^{-2}, d-mm pupil	q str^{-1} s^{-1} on retina	q deg^{-2} s^{-1} on retina	q mm^{-2} s^{-1} on retina
1 Troland=	1	3.142	$0.785\ d^2$	8.99×10^8	2.74×10^5	3.23×10^6
1 cd m^{-2}, 2-mm pupil=	0.318	1	$0.249\ d^2$	2.86×10^8	8.72×10^4	1.03×10^6
1 cd m^{-2}, d-mm pupil=	$1.273\ d^{-2}$	$3.99722\ d^{-2}$	1	$1.14 \times 10^9\ d^{-2}$	$3.49 \times 10^5\ d^{-2}$	$4.11 \times 10^6\ d^{-2}$
1 q str^{-1} s^{-1}=	1.11×10^{-9}	3.49×10^{-9}	$8.77 \times 10^{-10}\ d^2$	1	3.05×10^{-4}	3.59×10^{-3}
1 q deg^{-2} s^{-1}=	3.65×10^{-6}	1.15×10^{-5}	$2.87 \times 10^{-6}\ d^2$	3.28×10^3	1	1.18×10^1
1 q mm^{-2} s^{-1}=	3.10×10^{-7}	9.73×10^{-7}	$2.43 \times 10^{-7}\ d^2$	2.78×10^2	8.48×10^{-2}	1
1 q μm^{-2} s^{-1}=	0.310	0.973	$0.243\ d^2$	2.78×10^8	8.48×10^4	1.00×10^6
1 q s^{-1}/cone, absorbed=	0.155	0.486	$0.188\ d^2$	1.39×10^8	4.24×10^4	5.00×10^5
1 q s^{-1}/cone, active=	0.232	0.729	$0.182\ d^2$	2.09×10^8	6.35×10^4	7.49×10^5
1 Macaque Td=	1.700	5.341	$1.335\ d^2$	1.53×10^9	4.66×10^5	5.49×10^6
1 Cat Td=	1.667	5.236	$1.308\ d^2$	1.50×10^9	4.56×10^5	5.38×10^6

	q mm^{-2} s^{-1} on retina	q s^{-1}/ cone, absorbed	q s^{-1}/ cone, active	Macaque Td	Cat Td
1 Troland=	3.230	6.460	4.310	0.588	0.600
1 cd m^{-2}, 2-mm pupil=	1.028	2.056	1.372	0.187	0.191
1 cd m^{-2}, d-mm pupil=	$4.11\ d^{-2}$	$8.22\ d^{-2}$	$5.49\ d^{-2}$	$0.749\ d^{-2}$	$0.76\ d^{-2}$
1 q str^{-1} s^{-1}=	3.59×10^{-9}	7.19×10^{-9}	4.79×10^{-9}	6.54×10^{-10}	6.67×10^{-10}
1 q deg^{-2} s^{-1}=	1.18×10^{-5}	2.36×10^{-5}	1.57×10^{-5}	2.15×10^{-6}	2.19×10^{-6}
1 q mm^{-2} s^{-1}=	1.00×10^{-6}	2.00×10^{-6}	1.33×10^{-6}	1.82×10^{-7}	1.86×10^{-7}
1 q μm^{-2} s^{-1}=	1	2.000	1.334	0.182	0.186
1 q s^{-1}/cone, absorbed=	0.500	1	0.667	0.091	0.093
1 q s^{-1}/cone, active=	0.749	1.499	1	0.136	0.139
1 Macaque Td=	5.491	10.982	7.327	1	1.020
1 Cat Td=	5.383	10.767	7.183	0.980	1

[a] For human vision except where otherwise specified. Assumes no macular pigment. Assumes that cat and macaque eyes differ from human eyes only in size.

rates. If there is any difference, the rod estimate is high relative to the cone estimate.

D. Conversion Tables

Tables 3 and 4 show various units in which stimuli in vision experiments are likely to be expressed, with the corresponding retinal illuminances, rod and cone absorptions and activations and the conversion factors among them, according to the discussion above. The macaque and cat trolands are based on the assumption that a given solid visual angle covers 1.7 times less area on a macaque (*Macaca fascicularis*) retina than on a human's[112] and that 1 mm on the cat retina corresponds to 4.4 deg of visual angle.[113,114] Tables for conversion from other photometric units to those in Table 3 are available from Judd[115] or from Wyszecki and Stiles.[89] Older and more arcane units are available from LeGrand.[116]

6. CONTROL OF SENSITIVITY

As argued above, whether models of visual sensitivity are best expressed in the spatial or the Fourier domain depends on whether the sensitivity-controlling mechanisms operate before or after the signal enters frequency-selective channels. Here I examine the evidence on the locus at which sensitivity-controlling mechanisms act, with the focus being chiefly on human foveal vision, which dominates human psychophysics and human vision as it is used under natural conditions. It leads to the surprising conclusion that in human cone vision there is little evidence of retinal gain change at intensities below 3.5 log Td but that response compression and subtractive adaptation predominate as early as the outer segments of foveal cones. Specifically, I argue that most of the neurophysiological evidence comes from poikilotherms with retinas much different from those of primates (especially the receptors), is contaminated by rod input in nearly every case, and often may be subtractive when treated as multiplicative. Likewise, the psychophysical evidence shows phase independence, does not distinguish well between retinal and cortical mechanisms, and often can confuse multiplicative adaptation with response compression followed by subtractive adaptation.

A. Multiplicative Versus Subtractive Adaptation

Before proceeding, it is necessary to ensure that the concepts of subtractive adaptation and multiplicative adaptation (also called a gain change) are clearly in mind (the word additive is arguably more general and appropriate than "subtractive," and if it is called "subtractive" then perhaps for consistency the other ought to be called divisive, but usage favors the terms subtractive and multiplicative). Detailed equations are laid out clearly by Adelson,[117] but for present purposes only an intuitive understanding is required. For simplicity, assume that the visual response, R, is linearly proportional to the stimulus intensity, I:

$$R = aI - s, \tag{3}$$

where the value of a determines the magnitude of multiplicative adaptation; that of s, the magnitude of subtractive adaptation.

If a and s are set by the background only, then adding an incremental test flash, ΔI, produces

$$\Delta R + R = a(\Delta I + I) - s. \tag{4}$$

Substituting Eq. (3) into Eq. (4) yields

$$\Delta R = ak\Delta I. \tag{5}$$

The point here is simple but important: Subtractive adaptation reduces the visual response to the adapting stimulus, but, insofar as a system is linear, it has no effect on the response to a superimposed test stimulus that is too brief to elicit an adaptive response; multiplicative adaptation to any stimulus, however, always affects the response to any other stimulus. Where the system is nonlinear, of course, preceding subtractive adaptation can affect the responses to all stimuli by moving the system away from a saturated state.[117–119]

The distinction is illustrated graphically in Fig. 7, taken from Werblin.[119] Here the surround mechanism of ganglion cells seems to have a subtractive as opposed to a multiplicative effect on stimulation at the center of the receptive field.[64,65,119–121] Figure 7(A) shows the response of a *Necturus* bipolar cell to a flickering stimulus confined to the center mechanism of its receptive field. During the period labeled b, the surround was steadily illuminated. Although illumination of the surround decreased the response of the cell by 50%, it had no effect on the amplitude of the response to flicker confined to the receptive field center: Hence the effect of the surround is subtractive, not multiplicative.

Figure 7(B) shows the membrane potential of the bipolar cell in response to steady illumination of either the center alone (left-hand curve) or the center and surround (right-hand curve). It shows that over a wide range of intensities the response of the cell is a linear function of log stimulus intensity. Although this result is interesting in its own right and is discussed below, the point here is that the slope of the function relating the cell's response to the flickering stimulus was the same regardless of whether the surround was on or off.

It is important to note that if the flickering stimulus had been extended to cover the entire receptive field so that the stimulus to both center and surround was flickering, the response to the flicker would have been attenuated by the subtractive adaptation (assuming that the surround mechanism is fast enough to follow the flicker). The same is true in the spatial-frequency domain: Subtractive spatial antagonism reduces sensitivity to low spatial frequencies but has no effect on stimuli that are of frequencies too high to excite the antagonistic surround (but low enough to stimulate center mechanisms). An important consequence of all this is that presence of the bandpass contrast sensitivity function that is produced by attenuation of low spatial or temporal frequencies can be due to either subtractive or multiplicative adaptation, even though such band-pass shape is sometimes taken as a sign of multiplicative adaptation and can be modeled as such.[122]

B. Contrast Gain Control

In the past 20 years a new mechanism for the control of sensitivity has been introduced, namely, contrast gain control. Insofar as the gain control operates on contrast, the response to the mean luminance, i.e., the component at 0 frequency, is unaffected, so contrast gain control is somewhat frequency selective by its very nature. However, both psychophysical[28] and neurophysiological[39] evidence reveal a mechanism, located in the cortex, that is considerably more selective in spatial frequency (at least) than is necessary to exclude the mean luminance.

C. Response Compression

All neurons have a limit on the size of signal that they can produce, and the approach to that limit is gradual. This approach can be satisfactorily described in all the peripheral units by the hyperbolic function introduced to vision research by Naka and Rushton[123]:

$$\frac{V}{V_{\max}} = \frac{1}{1 + \left(\frac{I_0}{I}\right)^n}, \tag{6}$$

where I_0 is the stimulus intensity (I) at which the response (V) is half-maximum ($V_{\max}$) and n is a shape parameter that is usually equal to or close to unity for re-

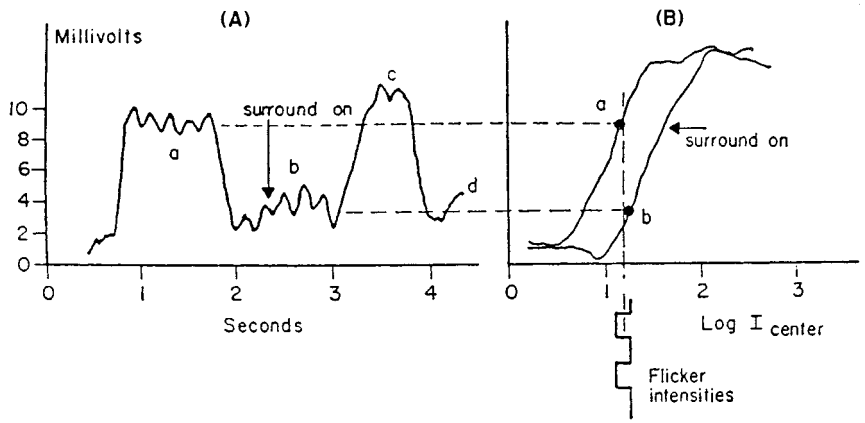

Fig. 7. Reproduction of Fig. 11 from Werblin,[119] showing (A) the subtractive nature of the surround in bipolar cells of the mudpuppy, and (B) linear portions of log intensity response curves.

ceptors. As I gets large, V approaches an asymptote of V_{max}. Approach to that asymptote is commonly referred to as saturation. Obviously, an incremental test stimulus that is added to a background light produces a smaller and smaller incremental response as the background increases. This can be seen in Fig. 1, where successive tenfold increases in stimulus magnitude produce responses that fall successively shorter of tenfold increases. Since the background light diminishes the response to a fixed test stimulus, this is a form of adaptation, as the term is used here. Usually referred to as response compression, it is a universal form of adaptation.

Multiplicative adaptation is incorporated in the expression for response compression by a change in the value of the semisaturation constant, I_0, or as a separate coefficient, a, of the intensity: aI. As the intensity I, increases, attenuation also increases (i.e., the value of a decreases). As both response compression and multiplicative adaptation have the effect of reducing the response to modulation superimposed on high background levels, the two have similar effects, and any attempt to infer the presence of multiplicative adaptation must separate the two (not an easy task).

Response compression is usually treated as a static function that is independent of temporal variables, but this is a fiction either forced by ignorance of the temporal properties or adopted for convenience in situations in which the time scale is long compared with the temporal processes that affect the observed response. In many models of adaptation, such as that of Baylor *et al.*,[124] part of the very process that produces response compression is a multiplicative adaptation that takes effect as the response to a given stimulus develops. In most experiments this can be treated as a static response compression, but it complicates attempts to distinguish multiplicative adaptation from response compression.

Nevertheless, adaptive changes do require a finite time to take effect. The failure of adaptive effects on modulation at high temporal frequencies[125] is a sign of this. Recent observations in macaques by Lee *et al.*[126] show a 35-ms delay between an adaptive stimulus and its effect at the level of the horizontal cell. Psychophysical observations of the time course of multiplicative adaptation[127] suggest that there is a fast process that is complete in less than 50 ms. Of course, there are also much slower multiplicative processes—pigment bleaching and regeneration, for example—that extend over many seconds.[128] Contrast gain control has a time course similar to but perhaps slightly slower than the fast multiplicative process, with a time constant of perhaps 60 ms[129] and no detectable effects at 30 ms.[56]

Response compression also complicates separation of subtractive and multiplicative adaptation. While subtractive adaptation preceding response compression protects the system from the insensitivity associated with saturation of the response, subtractive adaptation following response compression can have effects identical to those of multiplicative adaptation, as explained in the next section.

D. Sequence and Loci of Adaptive Stages

Since the idea of separate subtractive and multiplicative adaptation has taken hold, attempts have been made to fit them into a serial model with a nonlinear stage.[24,55,81,117,127,128,130–132] These models have been tested almost exclusively by their descriptions of the shapes of threshold-versus-intensity (tvi) curves, curves expressing the thresholds for brief, test flashes superimposed on backgrounds of varying intensity, also flashed briefly on steady adapting fields. Deriving such a systematic account of the sequence of operations performed by the visual system from psychophysical observations and relating them to physiological mechanisms is hard, and the difficulties force simplifications and limitations that warrant explicit recognition. Some of them are as follows.

1. *Caveats*

(a) The experimental tests of the sequence of stages pit a few models against one another, leaving untested alternatives, as has been acknowledged from the beginning.[117] In particular, the models seldom take into account parallel organization (with an exception in the realm of the first stages of color vision[133]).

(b) These tests depend on the assumption that each stage of the system participates equally at all levels of adaptation, but it is clear (cf. the discussion below) that different processes predominate at different levels: This assumption can lead to false conclusions. That is, a curve is fitted to a tvi curve that spans from near-absolute threshold to 4 or 5 log Td, and the conclusions are then based on the fitted parameters and are applied to the entire range covered. However, Kortum and Geisler[131] report that the exponent of the Naka–Rushton equation used to fit such data depends on whether intensities greater than 3 log Td are used in the fit. As the model fits high and low intensities differently, the question arises as to whether different conclusions apply to high and low intensities; Kortum and Geisler say that this difference affects only the details of the interpretation, not the basic conclusions.

(c) These studies use either the method of limits or the method of adjustment. The large amounts of data required for these experiments put a premium on efficient methods of collection, but both methods are sensitive to shifts of the observer's criterion. Comparing such thresholds to those obtained by forced choice, Kortum and Geisler[131] report small differences varying from 0 to 0.3 on a log scale, but according to Hayhoe *et al.*[132] the estimates of some of the parameters are sensitive to such small differences, sensitive, evidently, even to the curve fitting procedures used.[131] (The psychophysical methods used, along with this sensitivity to small differences, may also account for some of the large individual differences that plague this work.[127,131,132]) Any systematic effects of criterion shifts can be incorporated into the nonlinearity of the model, as Hayhoe *et al.*[132] have done, but then, as Hayhoe *et al.*[132] caution, one must give up interpretations based on the properties of specific neural elements.[132] This is a serious drawback.

(d) The test flashes are brief, and the backgrounds also are briefly present to probe the properties of the system before these stimuli change them, but as Geisler[128] and Hayhoe *et al.*[127] (p. 325) point out, both "signals may be affected by adaptational changes they themselves gener-

ate." If so, the results may depend on the temporal properties of the test stimuli, a dependence that has not been tested.

(e) Present methods of representing serial models have no good way to represent uncertainty about the sequence of processes: A diagram of a serial system must place components in serial order with or without data on their relative position, and choices made for expediency tend to persevere and to influence thinking.

(f) Response compression complicates the distinction between multiplicative and subtractive adaptation. Distinguishing multiplicative adaptation preceding response compression from subtractive adaptation following response compression may be difficult, for the two can be formally identical. The Naka–Rushton equation [Eq. (6)], for example, closely approximates a logarithmic transformation over a large part of its range; this can be seen as the linear portion of the curve when plotted on semilogarithmic axes, as in Fig. 7(B). Then, inserting a logarithmic response compression into Eq. (3) yields

$$R = a \log I - s; \tag{7}$$

and, if

$$s = \log s', \tag{8}$$

then

$$R = a \log\left(\frac{I}{s'}\right), \tag{9}$$

and multiplication of $1/s' \times I$ before the transformation is identical to subtraction of s after the logarithmic transformation, $a \log I$. This can be seen in Fig. 7(B), where the effect of the surround could be considered either a reduction of membrane potential by a fixed amount within the range between its upper and lower limits, or else it could be considered a lateral shift on the $\log I$ axis. Many compressive transformations are close enough to logarithmic to thwart practical efforts to distinguish between multiplication before and subtraction after the transformation. The difficulty of the discrimination depends on the details of the model. Geisler[129] shows how one particular class of tvi curves might be affected by pairing of multiplicative or subtractive adaptation with a Naka–Rushton function, either before or after it.

As noted above, all neurons are subject to response compression, and the compression increases as the signal passes through the hierarchy of visual stations.[134] Any multiplicative adaptation is likely to be followed by some response compression, which may make it look like subtractive adaptation. Conversely, almost all synapses tend to time differentiate the signal, which is a subtractive process (the lower the frequency, the greater the subtraction). So response compression at one synapse is likely to be followed by subtraction at the next, and that may look like multiplicative adaptation. So multiplicative adaptation before response compression is at least approximately the same as response compression before subtractive adaptation, and the physiological basis for all three phenomena are rife within the nervous system.

(g) Most psychophysical tests of adaptation are based on thresholds, which depend on noise as well as signal strength. Diagrams and equations used in the formulations and interpretations of such experiments deal exclusively with the signal, with the concept of noise rarely being introduced. This is no problem if one simply substitutes the ratio of signal to noise in place of I or R in these formulations. However, in practice, a decrease in observed response magnitude, R, is uniformly interpreted as a change of signal caused by a gain change or subtractive adaptation, for example, when it could instead be due to an increase of noise. The difference becomes especially important when one tries to relate the model to physiology. The possibility that the gain is set by the level of noise[135,136] instead of by the signal increases the importance of paying attention to the noise. In any case, in the experiment of Kortum and Geisler,[131] at least, the conclusions were unaffected if noise was assumed to be proportional to the mean response.

(h) Finally, note that at present neither the best model available in the temporal domain,[82] at least, nor any plausible variant can be reconciled with new results obtained by Hood *et al.*[81] and with other results reported in this issue by Wu *et al.*[83] This is, of course, a normal part of scientific progress, but it raises questions about the validity of inferences made on the basis of these models.

2. Subtractive Adaptation

With these caveats in mind, we take up the evidence on the sequence and the loci of adaptive stages, beginning with subtractive adaptation. All psychophysical tests and associated models since that of Sperling and Sondhi[137] agree in that both multiplicative and subtractive of adaptation precede the principal nonlinearity. However, as shown above, multiplication before the nonlinearity might be hard to separate from subtraction after the nonlinearity. Subtractive adaptation has happened to be diagrammed after multiplicative adaptation, although there was no evidence for either sequence until comparatively recently.[55,133] As early as Adelson's 1982 paper,[117] the idea of two different subtractive processes, a fast and a slow process, was broached.[128,132]

Hayhoe[55] has argued that a subtractive process that mediates spatial antagonism must follow the (multiplicative) gain change. However, Schnapf *et al.*[138] observed a different subtractive process that is local and is seen in the transmembrane current of the outer segments of macaque cones, producing their biphasic response[138]; Hayhoe elected to place the local process after both the multiplicative stage and after the spatially subtractive stage. But a process within cone outer segments that is independent of stimulation of neighboring cones, like that reported by Schnapf *et al.*, must precede any spatially antagonistic process, and it could precede multiplicative adaptation as well.

If the physiologically observed process is the same as that inferred from the psychophysics, the time constants should be the same. A rough estimate from Fig. 6 of Schnapf *et al.*[138] suggests that the delay of the subtractive process in cones varies by a factor of 2, with approximate time constants of 100 to 300 ms. The time constants that I have estimated roughly from the figures in the reports of psychophysics are at least as variable: $<$50 ms in Geisler[128]; for the two observers in Hayhoe *et al.*,[127] $<$25 ms and 200 ms, respectively; for the two ob-

servers in Hayhoe *et al.*,[132] 400 ms and 10 s at 1.9 log Td, 1 and 10 s at 2.9 log Td, and 50 and 200 msec when the luminance is increased from 1.9 to 2.9 log Td. These numbers are consistent with almost any interpretation, but the suggestion[132] that the lateral process may be much faster than the local process would account for the psychophysical data that are faster than those from the cone itself.

So far, then, the evidence shows a subtractive process within the cone with a time constant of a few hundred milliseconds, and a lateral subtractive process that may be much faster, with the multiplicative adaptation sandwiched in between.

3. Multiplicative Adaptation

a. Receptors. Most of what is known about the neurophysiology of retinal adaptation, both within receptors and in other parts of the retina, comes from poikilotherms and other species with receptors and retinal organization fundamentally different from those of macaques and humans. For example, differences in size of receptors,[139] numerical apertures of the eyes, and sensitivity of the cone system all increase the need for adaptive mechanisms in poikilotherm rods to retard saturation until luminances are high enough for the cone system to take over. Most current investigations of adaptation in receptors are limited to conditions in which a significant amount of pigment has been bleached,[140] but human rods saturate[141] before significant bleaching occurs.[111] In any case, macaque[142] and human[141] rods appear to differ from all the others that have been studied[2,143,144] in that they alone are not protected from saturation by gain changes. (Rat rods were previously thought not to adapt in this way, but Nakatani *et al.*[143] reported typical gain changes in rat rods, and their reanalysis of the data of Penn and Hagins[145] confirms this.) So present evidence is that human rods adapt neither multiplicatively nor subtractively.

It is of fundamental importance that human rod and cone systems adapt differently. Since Rushton introduced the concept of adaptation pools[146–148] it has been accepted that, over the entire range of rod vision, the gain changes occur at a site proximal to the rods themselves. At the top of the scotopic range, the average rod is activated by fewer than one quantum per integration time: Adaptation of human rods is not needed, and it is only at the site of convergence that there is danger of saturation. Signals from foveal cones, however, are subject to adaptation even without pooling of signals.[68,70,149,150]

There are four sources of neurophysiological evidence on adaptation in primate cones. Two involve recording massed potentials from the intact retina, but they use different techniques to isolate the cone potentials. Boynton and Whitten[151] recorded the intraretinal potential while clamping the retinal circulation, leaving only the choroidal flow to nourish the retina. This technique inactivates most of the retinal neurons except the receptors, but it leaves intact an unidentified layer of cells in the inner nuclear layer, and the S-potential likewise survives retinal clamping.[152,153] Brown *et al.*[152] argued convincingly that the potentials recorded by this technique must include receptor potentials, but their argument does not address the issue of whether they are exclusively receptor potentials. Baron and Boynton[154] tested for postreceptoral contributions to these recordings by blocking such contributions with sodium aspartate and comparing observations before and after its application. Although Baron and Boynton chose to emphasize the similarities, there are clear differences. The evidence favors the conclusion that the potentials recorded by Boynton and Whitten with this technique are contaminated by S-potentials.

Valeton and van Norren[155] recorded the potential difference across the photoreceptor layer with dual electrodes. Although this technique selectively enhances the signal from photoreceptors relative to other signals, it does not exclude a contribution from other sources. If their observations reflected pure cone signals, they would differ from those of Boynton and Whitten. However, the two results are nearly identical, as Hood and Birch[156] have shown, so it follows that the data of Valeton and van Norren are likewise contaminated by signals from retinal elements other than cones.

Schnapf *et al.*[138] recorded the transmembrane current of the outer segment of individual cones by sucking the outer segment of the isolated cone into a pipette. The principal drawback of this technique is the possibility that the electrode alters the state of the cone and its environment.

Finally, Hood and Birch[156] inferred the cone response from the leading edge of the a-wave of the electroretinogram. The main drawback of this technique is that it does not show the full time course of the response and any effects that light adaptation might have on it, although they have recently inferred the rest of the curve from double-flash experiments.[157]

All four studies are consistent in showing less than a tenfold change of sensitivity from multiplicative adaptation below the level at which sensitivity in the steady state is entirely controlled by photopigment bleaching. However, the adaptation index (the adaptation level at which sensitivity is reduced to half) inferred from the Boynton–Whitten and Valeton–van Norren data is approximately 2 log Td, while that of Schnapf *et al.*[138] corresponds to a background that isomerizes some 70,000 molecules/s/cone. On the assumptions of Schnapf *et al.*, that is 3.5 log Td, but according to Table 3 it is 3.9 log Td. The results obtained by Hood and Birch[156] lie somewhere between 3.5 and 3.9 log Td, depending on the correction for the Stiles–Crawford effect.

The evidence on cone vision, then, favors the conclusion that human and macaque cones do not show significant multiplicative adaptation until the light levels approach 3.5–4 log Td. Note that 4 log Td is higher than the light levels that one is likely to encounter under natural conditions, excluding the Sun and adjacent clouds.[158] At lower light levels any multiplicative adaptation must occur beyond the cones, after the local subtractive adaptation that does lie within the cones. However, as the psychophysical curves from which inferences about the sequence of processes are inferred typically do extend to 4 log Td or more, cone adaptation could affect their shapes and hence the conclusions about the sequence of processes based on psychophysics.

b. Postreceptoral Retina: Neurophysiology. To orient the reader, I note that this and the next section lead to the conclusion that there is no conclusive evidence of multiplicative adaptation in the retina at light levels below approximately 3.5 log Td.

As stated above, most of what is known of the retina comes from poikilotherms, and most of what little is known of the mammalian retina comes from cats. But the retinas of the poikilotherms are very different from the human retina, beginning at the receptors (in coupling, gain changes, and size), and the cat retina is dominated by rods, whereas human psychophysics is dominated by cones.

Owing to the predominance of rods throughout the cat retina, any given ganglion cell is almost certain to receive rod input.[159] It was noted above that rod signals undergo gain changes at a retinal site proximal to the rods. Therefore experiments conducted on cat ganglion cells under scotopic or mesopic conditions are certain to show retinal gain changes. To be sure that they are working in the photopic range of cats, physiologists have been forced to work at 3.5 log Td,[66] well above the level of many psychophysical experiments, where receptor adaptation begins to become significant even in humans (see subsection 6.D.*3.a*).

Such results from cat ganglion cells are frequently cited to support a retinal locus of gain changes in humans, but they need not apply to parvocellular ganglion cells in primates. Lee *et al.*[160] found no evidence of a gain control in parvo-ganglion cells even at 3.3 log Td (but did for magno-cells). Purpura *et al.*[122] have shown that increasing the mean luminance of a grating reduces the response of macaque ganglion cells to low temporal frequencies relative to that at higher temporal frequencies. Although the authors chose to model this as a gain change produced by negative feedback, their data do not exclude an explanation based on subtractive adaptation (see Subsection 6.A above), as they acknowledge.

Recently, Lee *et al.*[126] have demonstrated that the amplitude of the response of primate horizontal cells to low-amplitude, high-frequency flicker fluctuated in synchrony with a high-contrast, low-frequency flicker on which it was superimposed. This could be due either to multiplicative adaptation, presumably at the synapse between cone and horizontal cell, or to response compression (discussed above).

Such neurophysiological evidence as is applicable to primate cone vision, then, shows no clear evidence of retinal multiplicative adaptation in the cone pathway of the parvo-ganglion cells that form some 80% of the output of the retina and carry the signals on which, some argue,[161] nearly all visual performance is based. At least such multiplicative adaptation is not noticeable until luminance levels exceed almost unphysiological levels, where the cones themselves adapt.

c. Postreceptoral Retina: Psychophysics. Distinguishing the effects of retinal processes from those of central processes by psychophysical methods is problematic. The principal techniques used to distinguish retinal from central processes are tests of interocular transfer and tests of the relative importance of spatial location or spatial phase relative to spatial frequency (although occasionally more indirect tests have been used[55]). Both have their problems.

If stimulation of one eye affects the consequences of stimulating the other eye, one can conclude that the cortex is involved in the interaction (unless the pupil or fixation are uncontrolled). If no such effects are observed, however, one cannot infer that the effect is retinal, for the failure could be due to binocular rivalry, as in the case of the Westheimer effect.[49] Note also that adapting one eye can affect the signal that it sends to the brain, where it can interact with test signals from the other eye, thus producing interocular transfer without requiring a central process of adaptation.[162]

Observations that are unaffected by changes of the spatial phase of a grating or a Fourier component necessarily implicate the cortex, for the primate retina contains no mechanisms to subserve such independence. However, observing an effect that does depend on phase excludes neither retina nor cortex, for both retinal mechanisms and many cortical cells are sensitive to phase.

Typically, thresholds for gratings are the same no matter what their phase relative to a masking grating,[163] as long as the luminances are 25 cd m^{-2} or less and the spatial frequencies greater than 1 c/deg.[23,164,165] However, the significance of this should not be overrated, for demonstrating true phase independence is difficult. Changing the spatial position of a component of a complex pattern usually changes its Fourier spectrum, so one must exclude the consequences of those spectral changes before their effects can be attributed to retinal mechanisms. If sensitivity is tested with a grating superimposed on a masking grating, changing from a 0-deg phase difference between a masking and test gratings to a 180-deg difference simply changes the task from an increment threshold to a decrement threshold in the bright bars of the masking grating and from a decrement threshold to an increment threshold in the dark bars. Classically, increment and decrement thresholds differ little.[166] The greatest difference occurs in quadrature phase, but here allowance must be made for the fact that the increment in contrast of the mask plus test grating does not equal the contrast of the test grating: The total contrast equals the square root of the sum of the squared contrasts of the two gratings, and the phase of the composite grating also lies between that of test and mask.

Testing the effects of phase is also complicated by involuntary eye movements, which reduce the time-integrated contrast. (Note, however, that the attenuation is complete only under singular conditions, and a sine-wave grating of detectable contrast can survive linear drift over as many as 15 periods.) Eye movements are especially troublesome if the adapting and test gratings are not simultaneous.

A solution to the problem posed by eye movements is to conduct the experiment with stabilized retinal images, as Jones and Tulunay-Keesey did.[165] They tested contrast sensitivity during brief intervals between periods of adaptation to a grating of 75% contrast and found that adaptation reduced sensitivity tenfold for a 3-c/deg grating, no matter what its phase (0, 30, 45, 60, and 90 deg). Explaining this result in the spatial domain (i.e., by retinal mechanisms) is a challenge. The greatest threshold in-

crease that one could expect from Weber's law, for example, is 75%, but instead the increase was 1000%. However, the changes of contrast sensitivity produced in cortical neurons by adaptation to gratings[167] are sufficient to explain this result.

Hence this experiment, carried out at a luminance of 16 cd m^{-2}, showed no multiplicative adaptation that can be attributed to the retina, but by exclusion implicates cortical mechanisms of contrast gain control. Although contrast gain control operates on contrast instead of luminance, it is a multiplicative mechanism that is preceded by at least two kinds of subtractive adaptation: the local and the spatially antagonistic processes discussed above.

Thus the only clear evidence of retinal gain changes in the fovea, whether physiological or psychophysical, stems from the interpretation of tvi curves, discussed in Subsection 6.D.*2*, which places the first spatially antagonistic subtractive process—which can only be retinal—after a gain change. This likewise places the preceding gain change in the retina. Using the same paradigm, based on tvi curves, Kortum and Geisler[131] found that both subtractive and multiplicative adaptation are almost independent of spatial frequency. This places them distal to the cortical cells that are selective for spatial frequency, leaving open the possibility of a locus near the entrance to the cortex. It is hard to believe that this technique could be so consistently wrong about so fundamental a question, but the conflicting evidence from Lee *et al.*[160] and from Jones and Tulunay-Keesey,[165] and the many caveats listed above, must leave the question open.

d. Response Compression. As stated above, response compression is a ubiquitous form of adaptation, and it undoubtedly accounts for much of the sensitivity regulation at moderate photopic adapting levels. However, the effects of response compression are difficult to quantify without a model, for it occurs at multiple sites (at least two in the retina[133,168]) and is modified by the action of prior subtractive adaptation that varies with the spatial and the temporal properties of adapting and test stimuli (see Subsection 6.D.*2*), with wavelength,[133,168] and with eccentricity[169] (also, compare Hayhoe and Smith,[54] e.g., with almost any foveal tvi curve.[130,133,170–172]). The model by Wilson and Humanski[56] makes an impressive start on solving this tough problem.

e. Contrast Gain Control. Evidently, for the parvocellular system, at least, a major portion of the task of multiplicative adaptation (below 3.5 log Td)—aside from that accomplished by subtraction following response compression—falls to the mechanism of contrast gain control, a process that is both fast and labile[173–175] but perhaps of limited magnitude[165,168–171] (although complete loss of the signal in stabilized images within the cortex[176] suggests otherwise). How this mechanism might enter into general models of the visual system is just beginning to be worked out,[56,177,178] some of it in this feature.[32,179,180]

4. Chromatic Adaptation

The phenomena of color adaptation are outside the scope of this essay. It is concluded here that little multiplicative adaptation may occur in human cone vision below 3.5 log Td, aside from contrast gain control. If opponent pathways are subject to the same processes as the achromatic signals, which probably pass through the same retinal pathways,[161] one would look mainly to subtractive processes, response compression, and cortical mechanisms for explanations of the phenomena of color adaptation at natural light levels.

E. Conclusion

Much of what one would like to know about processes that control sensitivity and their loci is not known. Although not conclusive, present evidence favors the following schema. Sensitivity of the foveal cone system is probably strongly governed by the poorly understood cortical mechanisms of contrast gain control even after relatively brief exposure to patterned stimuli, but the effects may be limited to a single order of magnitude. Aside from fine tuning by such mechanisms, all the sensitivity changes over the lower 2–3 orders of magnitude of photopic luminance—the luminances spanned by most natural stimuli and the great majority of contemporary psychophysical experiments—can be attributed to the joint action of subtractive adaptation and response compression; multiplicative adaptation may contribute as well but cannot easily be distinguished from the other processes. Over a range of 1 order of magnitude, from approximately 3.5 to 4.5 log Td, the effect of multiplicative adaptation in receptors predominates, and, above that, bleaching of the photolabile pigments keeps the output of cones constant as the mean level of retinal illumination increases toward unphysiological levels. This view assigns to response compression a major burden of adjustment of sensitivity to the prevailing light level, as suggested by Fechner[181] almost a century and a half ago.

APPENDIX A

The argument proceeds by a sequence of nine propositions:

1. A symbol and its referent are different things. The words billiard ball constitute a symbol standing for the round, hard, physical object used in the game of billiards. This object is the referent for the symbol consisting of the words. The referent for the symbols 2, two, and zwei is a number. If you do not know the language, you do not know the referent and must deal only with the symbol, not its referent.

2. A symbol and its referent have different properties. One can roll a billiard ball but not the words that stand for it. Numbers have mathematical properties: They are ordered, additive, and so forth. Numerals, *per se*, cannot be added, and their ordering is ambiguous. For example, numerals might be ordered according to the area covered by the ink needed to print them, but they have no arithmetic value.

3. Causes that affect the referent do not affect its symbol. Hitting a billiard ball has no direct effect on the words for it.

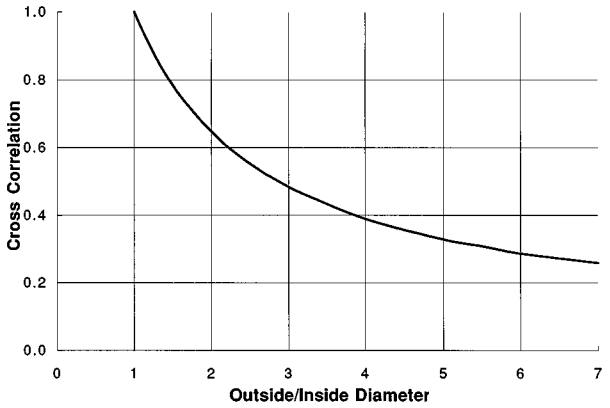

Fig. 8. Cross correlation of the spectrum of a disk of varying diameter.

4. Causes affecting the symbol do not affect its referent. Saying, "Hit the billiard ball" has no necessary effect on the billiard ball; nor does shredding the words affect the ball itself.

5. A physical cause has a physical effect. In a vision experiment, a light stimulus starts a causal chain in the nervous system that results in a response. If the response is a word, it is the symbol that is caused, not its referent. So the responses "yes," "no," and "two" are just words and do not necessarily have the properties of numbers, for example.

6. An abstract cause has an abstract effect. The question, "What is two plus three?" may cause the response, "Five." The response could be denoted by any of the symbols for the number 5 and could be caused by the same question, regardless of whether the question is expressed in numerals or words.

7. The physical and the abstract levels are not completely separate. Experiments that use magnitude estimation, for example, show that the symbols emitted as responses to physical stimuli that have no obvious symbolic significance (e.g., lights of varying luminance) sometimes do have the properties of the numbers for which they stand.[7] The experiment described under proposition 6 can be described at either the physical or the abstract level, but we cannot (at present) use the physical analysis to predict the physical response without knowing the referents for the symbols in the stimulus and the response.

8. We do not know the rules relating the physical and the abstract levels. Such rules are often called psychophysical linking hypotheses.

9. Therefore conclusions based on what observers mean depend on unknown psychophysical linking hypotheses, but we can avoid the need for such hypotheses by avoiding use of the meaning in observers' responses.

APPENDIX B

It may be useful to have an expression that describes the envelope of the function shown in Fig. 3, as shown by the heavy, smooth curve. I obtained this by modifying an expression for the upper bounds of Bessel functions (Eq. 9.1.63 in Abramowitz and Stegun[182]) to apply to Eq. (1):

$$A = \frac{1}{4}\log\left\{\frac{\exp[1-(4\pi f)^2]^{1/2}}{1+[1-(4\pi f)^2]^{1/2}} - 1.5077f\right\} - 0.8353. \tag{10}$$

The periodicity in Eq. (1) suggests the possibility that the spectra of two disks may fall in and out of spatial synchrony as their relative sizes vary; however, the smoothness of the cross-correlation function (Fig. 8) shows that this is not true.

ACKNOWLEDGMENTS

This research was supported by U.S. Public Health Service grants EY-4885 and EY-1319. I thank Peter Bex, Mary Hayhoe, Don Hood, Barry Lee, Peter Lennie, Jim Muller, Jim Thomas, and Dave Williams for comments on an earlier draft.

REFERENCES

1. D. I. A. MacLeod, "Visual sensitivity," Annu. Rev. Psychol. **29**, 369–645 (1978).
2. R. Shapley and C. Enroth-Cugell, "Visual adaptation and retinal gain controls," in *Progress in Retinal Research*, N. Osborne and G. Chader, eds. (Pergamon, New York, 1984), Vol. 3, pp. 263–346.
3. D. C. Hood and M. A. Finkelstein, "Sensitivity to light," in *Handbook of Perception and Human Performance*, K. R. Boff, L. Kaufman, and J. P. Thomas, eds. (Wiley, New York, 1986), Vol. 1, pp. 5-1–5-66.
4. J. Walraven, C. Enroth-Cugell, D. Hood, D. I. A. MacLeod, and J. L. Schnapf, "The control of visual sensitivity: receptoral and postreceptoral processes," in *Visual Perception: The Neurophysiological Foundations*, L. Spillmann and J. S. Werner, eds. (Academic, New York, 1990), pp. 53–101.
5. D. C. Hood, "Retinal control of sensitivity," Annu. Rev. Psychol. (to be published).
6. S. Hecht, S. Shlaer, and M. H. Pirenne, "Energy, quanta, and vision," J. Gen. Physiol. **25**, 819–840 (1942).
7. L. E. Marks, *Sensory Processes: The New Psychophysics* (Academic, New York, 1974).
8. L. Michaelis and M. L. Menten, "Die Kinetik der invertinwirkung," Biochem. Z. **49**, 333–369 (1913).
9. D. A. Baylor, B. J. Nunn, and J. L. Schnapf, "Spectral sensitivity of cones of the monkey *Macaca fascicularis*," J. Physiol. (London) **390**, 145–160 (1987).
10. J. C. Armington, *The Electroretinogram* (Academic, New York, 1974).
11. H. Autrum, "The physiological basis of colour vision in honeybees," in *Colour Vision*, A. V. S. de Reuck and J. Knight, eds. (Little, Brown, Boston, Mass., 1965), pp. 286–300.
12. N. V. S. Graham, *Visual Pattern Analyzers* (Oxford U. Press, New York, 1989).
13. S. Hecht, C. Haig, and A. M. Chase, "The influence of light adaptation on subsequent dark adaptation of the eye," J. Gen. Physiol. **20**, 831–850 (1937).
14. H. L. Van Trees, *Detection, Estimation, and Modulation Theory. Part I. Detection, Estimation, and Linear Modulation Theory* (Wiley, New York, 1968).
15. G. S. Brindley, *Physiology of the Retina and Visual Pathway* (Williams and Wilkins, Baltimore, Md., 1970).
16. D. Y. Teller, "Locus questions in visual science," in *Visual Coding and Adaptability*, C. S. Harris, ed. (Erlbaum, Hillsdale, N.J., 1980), pp. 151–176.
17. D. Y. Teller, "Linking propositions," Vision Res. **24**, 1233–1246 (1984).
18. D. Y. Teller and E. N. Pugh, Jr., "Linking propositions in color vision," in *Colour Vision: Physiology and Psychophysics*, J. D. Mollon and T. Sharpe, eds. (Academic, New York, 1983), pp. 577–589.

19. D. Y. Teller, "The domain of visual science," in *Visual Perception: The Neurophysiological Foundations*, L. Spillmann and J. S. Werner, eds. (Academic, New York, 1990), pp. 11–21.
20. H. R. Wilson and D. J. Gelb, "Modified line element theory for spatial frequency and width discrimination," J. Opt. Soc. Am. A **1**, 124–131 (1984).
21. Q. Hu, S. A. Klein, and T. Carney, "Can sinusoidal vernier acuity be predicted by contrast discrimination?" Vision Res. **33**, 1241–1258 (1993).
22. J. Yang, X. Qi, and W. Makous, "Zero frequency masking and a model of contrast sensitivity," Vision Res. **35**, 1965–1978 (1995).
23. J. Yang and W. Makous, "Modeling pedestal experiments with amplitude instead of contrast," Vision Res. **35**, 1979–1989 (1995).
24. N. Graham and D. C. Hood, "Modeling the dynamics of light adaptation: the merging of two traditions," Vision Res. **32**, 1373–1393 (1992).
25. F. W. Campbell and J. G. Robson, "Application of Fourier analysis to the visibility of gratings," J. Physiol. (London) **197**, 551–566 (1968).
26. C. Enroth-Cugell and J. G. Robson, "The contrast sensitivity of retinal ganglion cells of the cat," J. Physiol. (London) **187**, 517–552 (1966).
27. A. Pantle and R. Sekuler, "Size-detecting mechanisms in human vision," Science **162**, 1146–1147 (1968).
28. C. Blakemore and F. W. Campbell, "On the existence of neurones in the human visual system selectively sensitive to the orientation and size of retinal images," J. Physiol. (London) **203**, 237–260 (1969).
29. N. Graham and J. Nachmias, "Detection of grating patterns containing two spatial frequencies: a comparison of single-channel and multiple channel models," Vision Res. **11**, 251–259 (1971).
30. M. B. Sachs, J. Nachmias, and J. G. Robson, "Spatial-frequency channels in human vision," J. Opt. Soc. Am. **61**, 1176–1186 (1971).
31. J. P. Thomas, "Model of the function of receptive fields in human vision," Psychol. Rev. **77**, 121–134 (1970).
32. J. P. Thomas and L. A. Olzak, "Contrast gain control and fine spatial discriminations," J. Opt. Soc. Am. A **14**, 2392–2405 (1997).
33. H. R. Wilson and J. R. Bergen, "A four mechanism model for threshold spatial vision," Vision Res. **19**, 19–32 (1979).
34. A. B. Watson and A. J. Ahumada, "A hexagonal orthogonal-oriented pyramid as a model of image representation in visual cortex," IEEE Trans. Biomed. Eng. **36**, 97–106 (1989).
35. J. M. Foley, "Human luminance pattern-vision mechanisms: masking experiments require a new model," J. Opt. Soc. Am. A **11**, 1710–1719 (1994).
36. J. Rovamo, J. Mustonen, and R. Näsänen, "Modelling contrast sensitivity as a function of retinal illuminance and grating area," Vision Res. **34**, 1301–1314 (1994).
37. P. G. J. Barten, "Simple model for spatial frequency masking and contrast discrimination," in *Human Vision, Visual Processing, and Digital Display VI*, B. E. Rogowitz and J. P. Allebach, eds., Proc. SPIE **2411**, 142–158 (1995).
38. P. G. J. Barten, "Physical model for the contrast sensitivity of the human eye," in *Human Vision, Visual Processing, and Digital Display III*, B. E. Rogowitz, ed., Proc. SPIE **1666**, 57–72 (1992).
39. L. A. Bauman and A. B. Bonds, "Inhibitory refinement of spatial frequency selectivity in single cells of the cat striate cortex," Vision Res. **31**, 933–944 (1991).
40. D. H. Hubel and T. N. Wiesel, "Receptive fields and functional architecture of monkey striate cortex," J. Physiol. (London) **195**, 215–243 (1968).
41. G. Westheimer, "Spatial interaction in the human retina during scotopic vision," J. Physiol. (London) **181**, 881–894 (1965).
42. G. Westheimer, "Spatial interaction in human cone vision," J. Physiol. (London) **190**, 139–154 (1967).
43. B. H. Crawford, "The effect of field size and pattern on the change of visual sensitivity with time," Proc. R. Soc. London Ser. B **129**, 94–106 (1940).
44. J. I. Markoff and J. F. Sturr, "Spatial and luminance determinants of the increment threshold under monoptic and dichoptic viewing," J. Opt. Soc. Am. **61**, 1530–1537 (1971).
45. J. F. Sturr and D. Y. Teller, "Sensitization by annular surrounds: dichoptic properties," Vision Res. **13**, 909–918 (1973).
46. A. Arditi, "Binocular vision," in *Handbook of Perception and Human Performance*, K. R. Boff, L. Kaufman, and J. P. Thomas, eds. (Wiley, New York, 1986), Vol. 1, pp. 23-1–23-41.
47. R. Fox and R. Check, "Binocular fusion: a test of the suppression theory," Percept. Psychophys. **1**, 331–334 (1966).
48. W. Makous and R. K. Sanders, "Suppressive interactions between fused patterns," in *Visual Psychophysics and Physiology*, J. C. Armington, J. Krauskopf, and B. R. Wooten, eds. (Academic, New York, 1978), pp. 167–179.
49. C. Yu and D. M. Levi, "Cortical components of the Westheimer function," Vision Res. (to be published).
50. S. L. Buck, W. Makous, and T. Piantanida, "Background visibility and increment thresholds," Vision Res. **23**, 1107–1113 (1983).
51. P. Lennie and D. I. A. MacLeod, "Background configuration and rod threshold," J. Physiol. (London) **233**, 143–156 (1973).
52. D. W. Blick and D. I. A. MacLeod, "Rod threshold: influence of neighboring cones," Vision Res. **18**, 1611–1616 (1978).
53. M. Latch and P. Lennie, "Rod–cone interaction in light adaptation," J. Physiol. (London) **269**, 517–534 (1977).
54. M. M. Hayhoe and M. V. Smith, "The role of spatial filtering in sensitivity regulation," Vision Res. **29**, 457–469 (1989).
55. M. M. Hayhoe, "Spatial interactions and models of adaptation," Vision Res. **30**, 957–965 (1990).
56. H. R. Wilson and R. Humanski, "Spatial frequency adaptation and contrast gain control," Vision Res. **33**, 1133–1149 (1993).
57. D. A. Burkhardt, "The influence of center-surround antagonism on light adaptation in cones in the retina of the turtle," Visual Neurosci. **12**, 877–885 (1995).
58. D. A. Burkhardt, "Sensitization and centre–surround antagonism in *Necturus* retina," J. Physiol. (London) **236**, 593–610 (1974).
59. C. J. Karwoski and L. M. Proenza, "Transient adaptation and sensitization in the retina of *Necturus*," J. Gen. Physiol. **76**, 479–497 (1980).
60. C. J. Karwoski and D. A. Burkhardt, "Ganglion cell responses of the mudpuppy retina to flashing and moving stimuli," Vision Res. **16**, 1483–1495 (1976).
61. C. Enroth-Cugell, P. Lennie, and R. M. Shapley, "Surround contribution to light adaptation in cat retinal ganglion cells," J. Physiol. (London) **247**, 579–588 (1975).
62. E. A. Essock, S. Lehmkuhle, J. Frascella, and J. M. Enoch, "Temporal modulation of the background affects the sensitization response of *X*- and *Y*-cells in the dLGN of cat," Vision Res. **25**, 1007–1019 (1985).
63. K. Nakayama, "Local adaptation in cat LGN cells: evidence for a surround antagonism," Vision Res. **11**, 501–509 (1971).
64. B. Sakmann, O. D. Creutzfeldt, and H. Scheich, "An experimental comparison between the ganglion cell receptive field and the receptive field of the adaptation pool in the cat retina" Pfluegers Arch. Gesamte Physiol. Menschen Tiere **307**, 133–137 (1969).
65. H. B. Barlow and W. R. Levick, "Threshold setting by the surround of cat retinal ganglion cells," J. Physiol. (London) **259**, 193–208 (1976).
66. B. G. Cleland and A. W. Freeman, "Visual adaptation is highly localized in the cat's retina," J. Physiol. (London) **404**, 591–611 (1988).
67. D. M. Dacey and M. R. Petersen, "Dendritic field size and

morphology of midget and parasol ganglion cells of the human retina," Proc. Natl. Acad. Sci. USA **89**, 9666–9670 (1992).
68. D. I. A. MacLeod, D. R. Williams, and W. Makous, "A visual nonlinearity fed by single cones," Vision Res. **32**, 347–363 (1992).
69. B. Chen, W. Makous, and D. R. Williams, "Serial spatial filters in vision," Vision Res. **33**, 413–427 (1993).
70. S. He and D. I. A. MacLeod, "Contrast-modulation flicker: dynamics and spatial resolution of the light adaptation process," Vision Res. (to be published).
71. X. Qi, "Spatial summation and antagonism of foveal cone signals at different illuminances on the human retina," Ph.D. dissertation (University of Rochester, Rochester, New York, 1997).
72. R. N. Bracewell, *The Fourier Transform and Its Applications* (McGraw-Hill, New York, 1978).
73. H. R. Wilson, "Responses of spatial mechanisms can explain hyperacuity," Vision Res. **26**, 453–469 (1986).
74. A. C. Naiman and W. Makous, "Vernier acuity modeled by one-dimensional Fourier analysis," Invest. Ophthalmol. Visual Sci. (Suppl.) **37**, S734 (1996).
75. S. J. Waugh and D. M. Levi, "Orientation, masking, and vernier acuity for line targets," J. Opt. Soc. Am. A **12**, 2305–2317 (1995).
76. A. B. Watson, "Temporal sensitivity," in *Handbook of Perception and Human Performance*, K. R. Boff, L. Kaufman, and J. P. Thomas, eds. (Wiley, New York, 1986), Vol. 1, pp. 6-1–6-43.
77. M. B. Mandler and W. Makous, "A three channel model of temporal frequency perception," Vision Res. **24**, 1881–1887 (1984).
78. X. Qi, J. Yang, and W. Makous, "Further evidence of a third temporal channel," Invest. Ophthalmol. Visual Sci. (Suppl.) **34**, 780 (1993).
79. R. F. Hess and R. J. Snowden, "Temporal properties of human visual filters: number, shapes, and spatial covariation," Vision Res. **32**, 47–59 (1992).
80. S. J. Waugh and R. F. Hess, "Suprathreshold temporal-frequency discrimination in the fovea and the periphery," J. Opt. Soc. Am. A **11**, 1199–1212 (1994).
81. D. C. Hood, N. Graham, T. E. von Wiegand, and V. M. Chase, "Probed-sinewave paradigm: a test of models of light-adaptation dynamics," Vision Res. **37**, 1177–1191 (1997).
82. T. E. von Wiegand, D. C. Hood, and N. Graham, "Testing a computational model of light-adaptation dynamics," Vision Res. **35**, 3937–3051 (1995).
83. S. Wu, S. A. Burns, A. E. Elsner, R. T. Eskew, Jr., and J. He, "Rapid sensitivity changes on flickering backgrounds: tests of models of light adaptation," J. Opt. Soc. Am. A **14**, 2367–2378 (1997).
84. D. H. Kelly, "Flicker," in *Visual Psychophysics*, Vol. VII/4 of *Handbook of Sensory Physiology,* D. Jameson and L. Hurvich, eds. (Springer, New York, 1972), pp. 273–302.
85. C. A. Burbeck and D. H. Kelly, "Spatiotemporal characteristics of visual mechanisms: excitatory–inhibitory model," J. Opt. Soc. Am. **70**, 1121–1126 (1980).
86. J. Yang and W. Makous, "Spatiotemporal separability in contrast sensitivity," Vision Res. **24**, 2569–2575 (1994).
87. J. G. Robson, "Spatial and temporal contrast sensitivity functions of the visual system," J. Opt. Soc. Am. **56**, 1141–1142 (1966).
88. G. Wyszecki and W. S. Stiles, *Color Science: Concepts and Methods, Quantitative Data and Formulas* (Wiley, New York, 1967).
89. G. Wyszecki and W. Stiles, *Color Science: Concepts and Methods, Quantitative Data and Formulae*, 2nd ed. (Wiley, New York, 1982).
90. J. van de Kraats, T. T. J. M. Berendschot, and D. van Norren, "The pathways of light measured in fundus reflectometry," Vision Res. **36**, 2229–2247 (1996).
91. S. L. Buck, D. R. Peeples, and W. L. Makous, "Spatial patterns of rod–cone interaction," Vision Res. **19**, 775–782 (1979).
92. R. A. Applegate and V. Lakshminarayanan, "Parametric representation of Stiles–Crawford functions: normal variation of peak location and directionality," J. Opt. Soc. Am. A **10**, 1611–1623 (1993).
93. C. A. Curcio, K. A. Allen, K. R. Sloan, C. L. Lerea, J. B. Hurley, I. B. Klock, and A. H. Milam, "Distribution and morphology of human cone photoreceptors stained with anti-blue opsin," J. Comp. Neurol. **312**, 610–624 (1991).
94. J. Pokorny and V. C. Smith, "Effect of field size on red–green color mixture equations," J. Opt. Soc. Am. **66**, 705–708 (1976).
95. O. S. Packer and D. R. Williams, "The quantum efficiency and directional sensitivity in peripheral primate photoreceptor mosaic," presented at OSA 1996 Annual Meeting, Rochester, N.Y., October 20–24 (Optical Society of America, Washington, D.C., 1996).
96. D. C. Hood and D. G. Birch, "Phototransduction in human cones measured using the a-wave of the ERG," Vision Res. **35**, 2801–2810 (1995).
97. W. S. Stiles and B. F. Crawford, "The luminous efficiency of rays entering the eye pupil at different points," Proc. R. Soc. London Ser. B **112**, 428–450 (1933).
98. C. A. Curcio, K. R. Sloan, R. E. Kalina, and A. E. Hendrickson, "Human photoreceptor topography," J. Comp. Neurol. **292**, 497–523 (1990).
99. G. Østerberg, "Topography of the layer of rods and cones in the human retina," Acta Ophthalmol. **13**, Suppl. 6, 1–103 (1935).
100. E. Aulhorn and H. Harms, "Visual perimetry," in *Visual Psychophysics*, Vol. VII/4 of *Handbook of Sensory Physiology*, D. Jameson and L. M. Hurvich, eds. (Springer, New York, 1972), pp. 102–145.
101. W. A. H. Rushton and G. H. Henry, "Bleaching and regeneration of cone pigments in man," Vision Res. **8**, 617–631 (1968).
102. H. B. Barlow, "Retinal noise and absolute threshold," J. Opt. Soc. Am. **46**, 634–639 (1956).
103. E. N. Pugh, Jr., "Rhodopsin flash photolysis in man," J. Physiol. (London) **248**, 413–435 (1975).
104. T. W. Kraft, D. M. Schneeweis, and J. L. Schnapf, "Visual transduction in human rod photoreceptors," J. Physiol. (London) **464**, 747–765 (1993).
105. M. E. Breton, A. W. Schueller, T. D. Lamb, and E. N. Pugh, Jr., "Analysis of ERG a-wave amplification and kinetics in terms of the G-protein cascade of phototransduction," Invest. Ophthalmol. Visual Sci. **35**, 295–309 (1994).
106. G. S. Brindley, "The deformation phosphene and the funnelling of light into rods and cones," J. Physiol. (London) **188**, 24–25P (1966).
107. M. Alpern, "Rhodopsin kinetics in the human eye," J. Physiol. (London) **217**, 447–471 (1971).
108. M. Hollins and M. Alpern, "Dark adaptation and visual pigment regeneration in human cones," J. Gen. Physiol. **62**, 430–447 (1971).
109. A. C. C. Coolen and D. van Norren, "Kinetics of human cone photopigments explained with a Rushton–Henry model," Biol. Cybern. **58**, 123–128 (1988).
110. M. Alpern, F. Maaseidvaag, and N. Ohba, "The kinetics of cone visual pigments in man," Vision Res. **11**, 539–549 (1971).
111. M. Alpern and E. N. Pugh, Jr., "The density and photosensitivity of human rhodopsin in the living retina," J. Physiol. (London) **237**, 341–370 (1974).
112. T. Yeh, B. B. Lee, and J. Kremers, "The time course of adaptation in macaque retinal ganglion cells," Vision Res. **36**, 913–931 (1996).
113. H. B. Barlow, R. Fitzhugh, and S. W. Kuffler, "Change of organization in the receptive fields of the cat's retina during dark adaptation," J. Physiol. (London) **137**, 338–354 (1957).
114. P. O. Bishop, W. Kozak, and G. J. Vakkur, "Some quantitative aspects of the cat's eye: axis and plane of reference, visual field, co-ordinates and optics," J. Physiol. (London) **163**, 466–502 (1962).
115. D. B. Judd, "Basic correlates of the visual stimulus," in

Handbook of Experimental Psychology, S. S. Stevens, ed. (Wiley, New York, 1951), pp. 811–867.
116. Y. LeGrand, *Light, Colour and Vision*, 2nd ed. (Chapman and Hall, London, 1968).
117. E. H. Adelson, "Saturation and adaptation in the rod system," Vision Res. **22**, 1299–1312 (1982).
118. H. B. Barlow, "Optic nerve impulses and Weber's law," Cold Spring Harbor Symp. Quant. Biol. **30**, 539–546 (1965).
119. F. S. Werblin, "Control of retinal sensitivity. II. Lateral interactions at the outer plexiform layer," J. Gen. Physiol. **63**, 62–87 (1974).
120. B. G. Cleland and C. Enroth-Cugell, "Quantitative aspects of sensitivity and summation in the cat retina," J. Physiol. (London) **198**, 17–38 (1968).
121. C. Enroth-Cugell and P. Lennie, "The control of retinal ganglion cell discharge by receptive field surrounds," J. Physiol. (London) **247**, 551–578 (1975).
122. K. Purpura, D. Tranchina, E. Kaplan, and R. M. Shapley, "Light adaptation in the primate retina: analysis of changes in gain and dynamics of monkey retinal ganglion cells," Visual Neurosci. **4**, 75–93 (1990).
123. K. I. Naka and W. A. H. Rushton, "*S*-potentials from colour units in the retina of the fish (Cyprinidae)," J. Physiol. (London) **185**, 536–555 (1966).
124. D. A. Baylor, A. L. Hodgkin, and T. D. Lamb, "Reconstruction of the electrical responses of turtle cones to flashes and steps of light," J. Physiol. (London) **242**, 759–791 (1974).
125. D. H. Kelly, "Visual responses to time-dependent stimuli. I. Amplitude sensitivity measurements," J. Opt. Soc. Am. **51**, 422–428 (1961).
126. B. B. Lee, D. M. Dacey, V. C. Smith, and J. Pokorny, "Time course and cone specificity of adaptation in primate outer retina," Invest. Ophthalmol. Visual Sci. Suppl. **38**, S1163 (1997).
127. M. M. Hayhoe, N. I. Benimoff, and D. C. Hood, "The time-course of multiplicative and subtractive adaptation process," Vision Res. **27**, 1981–1996 (1987).
128. W. S. Geisler, "Mechanisms of visual sensitivity: backgrounds and early dark adaptation," Vision Res. **23**, 1423–1432 (1983).
129. H. R. Wilson, V. P. Ferrara, and C. Yo, "Psychophysically motivated model for two-dimensional motion perception," Visual Neurosci. **9**, 79–97 (1992).
130. W. S. Geisler, "Effects of bleaching and backgrounds on the flash response of the cone system," J. Physiol. (London) **312**, 413–434 (1981).
131. P. T. Kortum and W. S. Geisler, "Adaptation mechanisms in spatial vision. II. Flash thresholds and background adaptation," Vision Res. **35**, 1595–1609 (1995).
132. M. M. Hayhoe, M. E. Levin, and R. J. Koshel, "Subtractive processes in light adaptation," Vision Res. **32**, 323–333 (1992).
133. M. A. Finkelstein, M. Harrison, and D. C. Hood, "Sites of sensitivity control within a long-wavelength cone pathway," Vision Res. **30**, 1145–1158 (1990).
134. G. Sclar, J. H. R. Maunsell, and P. Lennie, "Coding of image contrast in central visual pathways of the macaque monkey," Vision Res. **30**, 1–10 (1990).
135. K. Donner, D. Copenhagen, and T. Reuter, "Weber and noise adaptation in the retina of the toad *Bufo marinus*," J. Gen. Physiol. **95**, 733–753 (1990).
136. M. E. Rudd and L. G. Brown, "Stochastic retinal mechanisms of light adaptation and gain control," Spatial Vis. **10**, 125–148 (1996).
137. G. Sperling and M. M. Sondhi, "Model for visual luminance discrimination and flicker detection," J. Opt. Soc. Am. **58**, 1133–1145 (1968).
138. J. L. Schnapf, B. J. Nunn, M. Meister, and D. A. Baylor, "Visual transduction in cones of the monkey *Macaca fascicularis*," J. Physiol. (London) **437**, 681–713 (1990).
139. G. L. Fain, "Sensitivity of toad rods: dependence on wave-length and background illumination," J. Physiol. (London) **261**, 71–101 (1976).
140. G. L. Fain, H. R. Matthews, and M. C. Cornwall, "Dark adaptation in vertebrate photoreceptors," Trends Neurosci. **19**, 502–507 (1996).
141. M. Aguilar and W. S. Stiles, "Saturation of the rod mechanism of the retina at high levels of stimulation," Opt. Acta **1**, 59–65 (1954).
142. B. J. Nunn and D. A. Baylor, "Visual transduction in retinal rods of the monkey *Macaca fascicularis*," Nature **299**, 726–728 (1982).
143. K. Nakatani, T. Tamura, and K.-W. Yau, "Light adaptation in retinal rods of the rabbit and two other nonprimate mammals," J. Gen. Physiol. **97**, 413–435 (1991).
144. T. Tamura, K. Nakatani, and K.-W. Yau, "Light adaptation in cat retinal rods," Science **245**, 755–758 (1989).
145. R. D. Penn and W. A. Hagins, "Kinetics of the photocurrent of retinal rods," Biophys. J. **12**, 1073–1094 (1972).
146. W. A. H. Rushton, "Increment threshold and dark adaptation," J. Opt. Soc. Am. **53**, 104–109 (1963).
147. W. A. H. Rushton, "The sensitivity of rods under illumination," J. Physiol. (London) **178**, 141–160 (1965).
148. W. A. H. Rushton, "The Ferrier lecture: Visual adaptation," Proc. R. Soc. London Ser. B **162**, 20–46 (1965).
149. D. C. Burr, J. Ross, and M. C. Morrone, "Local regulation of luminance gain," Vision Res. **25**, 717–727 (1985).
150. C. M. Cicerone, M. M. Hayhoe, and D. I. A. MacLeod, "The spread of adaptation in human foveal and parafoveal cone vision," Vision Res. **30**, 1603–1615 (1990).
151. R. M. Boynton and D. N. Whitten, "Visual adaptation in monkey cones: recordings of late receptor potentials," Science **170**, 1423–1425 (1970).
152. K. T. Brown, K. Watanabe, and M. Murakami, "The early and late receptor potentials of monkey cones and rods," Cold Spring Harbor Symp. Quant. Biol. **30**, 457–482 (1965).
153. K. T. Brown, "The electroretinogram: its components and their origins," Vision Res. **8**, 633–677 (1968).
154. W. S. Baron and R. M. Boynton, "Temporal frequency dependent adaptation at the level of the outer retina in humans," Vision Res. **32**, 2043–2048 (1975).
155. J. M. Valeton and N. D. van Norren, "Light adaptation of primate cones: an analysis based on extracellular data," Vision Res. **23**, 1539–1547 (1983).
156. D. C. Hood and D. G. Birch, "Human cone receptor activity: the leading edge of the *a*-wave and models of receptor activity," Visual Neurosci. **10**, 857–871 (1993).
157. D. C. Hood, D. G. Birch, and D. R. Pepperberg, "The trailing edge of the photoresponse from human cones derived using a two-flash ERG paradigm," in *Vision Science and Its Applications*, Vol. 1 of 1996 OSA Technical Digest Series (Optical Society of America, Washington, D.C., 1996), pp. 64–67.
158. W. Makous, "Optics," in *Vision Research: a Practical Approach*, J. G. Robson and R. H. S. Carpenter, eds. (Oxford U. Press, Oxford, to be published).
159. H. B. Barlow and W. R. Levick, "The Purkinje shift in the cat retina," J. Physiol. (London) **196**, 2P–3P (1968).
160. B. B. Lee, J. Pokorny, V. C. Smith, and J. Kremers, "Responses to pulses and sinusoids in macaque ganglion cells," Vision Res. **23**, 3081–3096 (1994).
161. P. Lennie, "Roles of M and P pathways," in *Contrast Sensitivity*, R. Shapley and D. M.-K. Lam, eds. (MIT Press, Cambridge, Mass., 1993), pp. 201–213.
162. W. Makous, D. Teller, and R. Boothe, "Binocular interaction in the dark," Vision Res. **16**, 473–476 (1976).
163. J. J. Kulikowski, "What really limits vision? Conceptual limitations to the assessment of visual function and the role of interacting channels," in *Limits of Vision*, J. J. Kulikowski, V. Walsh, and I. J. Murray, eds. (CRC, Boca Raton, Fla., 1991), Vol. 5, pp. 286–329.
164. C. F. Stromeyer, S. Klein, B. M. Dawson, and L. Spillmann, "Low spatial frequency channels in human vision: adaptation and masking," Vision Res. **22**, 225–233 (1982).
165. R. M. Jones and U. Tulunay-Keesey, "Phase selectivity of

spatial frequency channels," J. Opt. Soc. Am. **70**, 66–70 (1980).
166. G. E. Legge and D. Kersten, "Light and dark bars: contrast discrimination," Vision Res. **23**, 473–483 (1983).
167. D. G. Albrecht, S. B. Farrar, and D. B. Hamilton, "Spatial contrast adaptation characteristics of neurones recorded in the cat's visual cortex," J. Physiol. (London) **347**, 713–739 (1984).
168. M. A. Finkelstein and D. C. Hood, "Cone system saturation: more than one stage of sensitivity loss," Vision Res. **21**, 319–328 (1981).
169. W. S. Stiles and B. H. Crawford, "Equivalent adaptation levels in localized retinal areas," in *Mechanisms of Colour Vision: Selected Papers of W. S. Stiles, F. R. S., with a New Introductory Essay*, W. S. Stiles, ed. (Academic, New York, 1978), pp. 36–53.
170. W. S. Stiles, "Color vision: the approach through increment-threshold sensitivity," Physics **45**, 100–113 (1959).
171. W. S. Stiles, "Separation of the 'blue' and 'green' mechanisms of foveal vision by measurements of increment thresholds," Proc. R. Soc. London Ser. B **133**, 418–434 (1946).
172. B. Chen and W. Makous, "Light capture by human cones," J. Physiol. (London) **414**, 89–109 (1989).
173. A. B. Saul and M. S. Cynader, "Adaptation in single units in visual cortex: the tuning of aftereffects in the spatial domain," Visual Neurosci. **2**, 593–607 (1989).
174. A. B. Bonds, "The encoding of cortical contrast gain control," in *Contrast Sensitivity*, R. Shapley and D. M.-K. Lam, eds. (MIT Press, Cambridge, Mass., 1993), pp. 215–230.
175. J. D. Allison, V. A. Casagrande, E. J. Debruyn, and A. B. Bonds, "Contrast adaptation in striate cortical neurons of the nocturnal primate bush baby," Visual Neurosci. **10**, 1129–1139 (1993).
176. D. Lehman, G. W. Beeler, and D. H. Fender, "Changes in patterns of the human electroencephalogram during fluctuations of perception of stabilized images," Electroencephalogr. Clin. Neurophysiol. **19**, 335–343 (1965).
177. D. J. Heeger, "Normalization of cell responses in cat striate cortex," Visual Neurosci. **9**, 181–197 (1992).
178. R. J. Snowden, "Adaptability of the visual system is inversely related to its sensitivity," J. Opt. Soc. Am. A **11**, 25–32 (1994).
179. A. B. Watson and J. A. Solomon, "A model of visual contrast gain control and pattern masking," J. Opt. Soc. Am. A **14**, 2379–2391 (1997).
180. M. P. Eckstein, A. J. Ahumada, Jr., and A. B. Watson, "Visual signal detection in structured backgrounds. II. Effects of contrast gain control, background variations, and white noise," J. Opt. Soc. Am. A **14**, 2406–2419 (1997).
181. G. T. Fechner, *Elemente der Psychophysik* (Breitkopf und Hertel, Leipzig, 1860).
182. M. Abramowitz and I. A. Stegun, eds., *Handbook of Mathematical Tables with Formulas, Graphs, and Mathematical Tables*, Vol. 55 of Applied Mathematics (National Bureau of Standards, Washington, D.C., 1964).